RECENT ADVANCES IN PULMONARY TUBERCULOSIS

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BY

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THIRD EDITION

WITH 48 PLATES AND 22 TEXT-FIGURES

LONDON

J. & A. CHURCHILL LTD.

104 GLOUCESTER PLACE

PORTMAN SQUARE

1937

To
M. L. B.

Printed in Great Britain

PREFACE TO THE THIRD EDITION

SINCE the last edition was published there has been considerable progress in the work on pulmonary tuberculosis, and in order to bring this book up to date a large part has been entirely re-written.

New chapters dealing with infectivity and immunity and with bovine and childhood tuberculosis have been added, and there is also a new chapter on classification and types of pulmonary tuberculosis, including the miliary form which was first found by radiography and may be followed by recovery. The chapters on radiology and surgical treatment have mostly been re-written.

The author has taken a broad view as to what should or should not be regarded as a "recent advance." The book is intended for general practitioners and students, and to be a guide to the modern methods of diagnosing and dealing with patients suffering from pulmonary tuberculosis. It is not meant to be a hash-up of new work done on the subject during the last few years. Artificial pneumothorax and sanatorium routine cannot truly be regarded as recent advances, and yet the conduct of pneumothorax treatment and the management of patients in a sanatorium are utterly different to what they were ten years ago.

Enormous advances have been made in surgical treatment, and the significance of cavities is now fully appreciated, and yet the modern tendency is to avoid drastic surgical intervention if possible and substitute partial for complete thoracoplastic operations.

References are given at the end of each chapter, and the author has tried to confine these as far as possible to works which may be found in any medical library.

Most of the radiographs are new, and in response to criticism they are inserted altogether at the end instead of being interspersed throughout the book.

Most of the radiographs were taken at the Brompton Hospital, and the thanks of the author are due to Dr. Rawlinson, not only for supplying these, but for his kindly help in their selection. The author is indebted to Dr. Todd and Dr. Hempson for other radiographs, and to Dr. Maxwell for the two showing miliary tuberculosis.

L. S. T. BURRELL.

LONDON.

PREFACE TO THE FIRST EDITION

TUBERCULOSIS has been one of the great problems of medicine since the earliest days of history. It is only natural in such a disease that many remedies should have been recommended, but their vogue has usually been of very short duration, and it has been wisely said that when a new "cure" for consumption appears those who want to use it should do so at once before it goes out of fashion and ceases to operate.

In writing this book, the author has tried to describe the recent increase in our knowledge of tuberculosis, and to separate the real advances in treatment from those methods which have no scientific basis and enjoy but brief, though occasionally recurrent, phases of popularity.

Most of the book deals with treatment, and no attempt has been made to describe physical signs or morbid anatomy, because the work is intended for practitioners and senior students, and not as an examination text-book. Chapters on diagnosis and prognosis are included, and an introductory chapter describes certain animal experiments, with modern views as to immunity and spread of disease, for treatment is largely based on these views.

The author wishes to thank Dr. Davies, of Cefn Mably Sanatorium, for permission to reproduce the skiagram of silicosis, and Dr. Stanley Melville for all the other skiagrams in the book.

L. S. T. BURRELL.

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RECENT ADVANCES IN PULMONARY TUBERCULOSIS

INTRODUCTION

TUBERCULOSIS has been described as a classic because its manifestations are so many and varied that a student who has been well grounded in the problems of tuberculosis must have a good framework of knowledge on which to base his study of other diseases. Not only may any part of the body be affected and produce symptoms resulting from injury to that particular organ, but the general effects of tuberculous affection, allergy, sensitivity, immunity and tolerance present endless opportunities for research.

For practical purposes there are two main problems, the first the prevention of tuberculosis amongst the community, and the second the treatment of the individual who has the disease.

This book is intended to deal with the problem as it affects the individual rather than the community, but it is proposed to show in this introduction that the epidemiology of tuberculosis is a subject of great complexity. Some contend that tuberculosis is merely a matter of hygiene and social conditions, but this view does not explain the variation in the tuberculosis death rate during the first quarter of this century in different countries; for example, it decreased by 50·5 per cent. in England and Wales, 22·6 per cent. in Italy, but increased by 12·7 per cent. in Japan. Social conditions do play an important part in the incidence of tuberculosis, which is, on the whole, a disease of poverty, and is more rife

in overcrowded districts; but there are other and probably more important factors, since several instances can be given of a steady decline in tuberculosis in districts which have not undergone any improvement in social or sanitary conditions.

Mild infection in infancy may produce some protection against the development of active tuberculosis in later life, and yet there is evidence that in certain cases the opposite is the fact. For example, Allen (1) in his study of raw recruit natives to the South African mines found that clinical tuberculosis developed in 738 amongst tuberculin reactors as against 347 amongst non-reactors per 100,000.

The war had a great effect on the tuberculosis death rate. In Germany in 1917 it was double that of 1914, but fell to pre-war level by 1920. In some countries it was treble, and almost every country showed some increase during the war. The influenza epidemic, which was world-wide just before and after the Armistice, had a slight effect on the death rate from pulmonary tuberculosis, but considerably less than is generally believed.

TABLE 1

Death Rate from Pulmonary Tuberculosis and Influenza in England and Wales per million Living

	Pulmonary Tuberculosis.	Influenza.
1914 . . .	988	148
1915 . . .	1,053	257
1916 . . .	1,046	210
1917 . . .	1,085	174
1918 . . .	1,165	3,024
1919 . . .	918	1,169

It is probable that, as with malaria, measles and other diseases, tuberculosis is most severe in natives who are not

accustomed to it, and this suggests some inherited or natural immunity. Thus in China the disease runs a very chronic course. It is common there, but the patients suffer from little, if any, toxæmia even with an advanced stage of the disease. On the other hand tuberculosis is very acute and caused 22 per cent. of all deaths amongst the North American Indians in Canada in 1930.

It has been suggested that amongst races where tuberculosis is common the majority of infants are infected and the survivors obtain a certain degree of immunity as a result of the childhood infection.

No doubt many causes contribute to this decline in the incidence and death rate of tuberculosis, for no one cause offers a complete explanation.

Some authorities regard tuberculosis as an endemic disease with epidemic waves. The last epidemic wave reached its peak some years ago, and is now steadily declining. Zimmerli (2) thinks this contention is supported by the conditions in Egypt where the same rapid abatement of consumption has occurred even in districts which he says are as unhealthy and as picturesque as they were a hundred years ago.

Improved social conditions, and especially cleanliness with its consequent diminution in the risk of getting a massive infection, and other modern methods of combating the disease no doubt play a part in the abatement of tuberculosis, but its almost world-wide decline cannot be due to these measures alone.

Nor can the credit go to acquired immunity, for the greatest decline has occurred amongst infants. The course of the disease is, however, modified by a previous infection, and it is important to consider the modern views on immunity both natural and acquired.

In most civilised countries there has been a steady decline in the death rate and incidence of tuberculosis during the last century.

TABLE 2

Annual Death Rates from Tuberculosis in England and Wales per million Living

	Tuberculosis all forms.	Pulmonary Tuberculosis.
1851–1860 . .	3,478	2,772
1861–1870 . .	3,263	2,590
1871–1880 . .	2,882	2,231
1881–1890 . .	2,444	1,810
1891–1900 . .	2,021	1,418
1901–1910 . .	1,646	1,143
1910–1920 . .	1,375	1,007
1921–1930 . .	993	768
1931–1935 . .	782	620
1935 . . .	687	552

Now it will be seen that this fall is steady and does not show any sudden changes to indicate the discovery of the tubercle bacilli and subsequent introduction of tuberculin in the early 'eighties, nor do artificial pneumothorax, sanocrysin, surgical or other modern methods of treatment appear to have any appreciable effect upon the death rates.

In the early part of last century there began a wave of industrialism in this country, and people flocked from the country into the towns and factories, so one would have expected a rise rather than a fall in tuberculosis.

TABLE 3

Deaths from Tuberculosis in England and Wales

Ages.	Tuberculosis in all forms.		Pulmonary Tuberculosis.	
	1920.	1934.	1920.	1934.
0–1 .	1,405	413	219	57
1–5 .	3,988	1,308	760	148
5–10 .	1,631	682	467	128
25–30 .	4,216	3,653	3,738	3,307

Analysis of these figures shows that the decrease in death rate chiefly affects infants and young children, and in recent years this has been very considerable.

Thus, between 1920 and 1934 the death rate has been reduced by 70 per cent. for all forms of tuberculosis and 78·5 per cent. for pulmonary tuberculosis in infants under one year of age, but only by 14 per cent. for all forms and 11 per cent. for the pulmonary form in adults of twenty-five to thirty.

Deaths from tuberculosis in early childhood are largely due to meningitis, and it will be seen that the death rate from tuberculous meningitis does not show the same abrupt rise at the age of puberty as occurs in the respiratory form.

TABLE 4

Deaths in England and Wales in 1935 *from Tuberculosis*

Ages.	All forms except that of the respiratory tract.		Central nervous system.		Intestines and peritoneum.		Bones and joints.		Respiratory system.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
0–1	167	120	101	81	32	13	2	1	25	23
1–5	526	411	373	294	65	33	13	6	58	75
5–10	243	238	157	157	26	19	16	10	55	49
10–15	176	177	90	100	18	20	20	12	85	215
15–20	235	203	88	81	35	57	25	14	624	1,110
20–25	207	184	58	49	31	47	44	26	1,459	1,919
25–30	183	138	37	35	33	35	45	14	1,541	1,676
30–35	135	114	33	21	19	32	35	17	1,424	1,279

Of the deaths from non-pulmonary tuberculosis in children under fifteen therefore, 65 per cent. are due to meningitis.

At the age of puberty there is an enormous increase in the death rate from pulmonary tuberculosis. This increase starts at an earlier age in girls than in boys, but it steadily declines in women after the age of thirty-five, whereas in men there is no appreciable fall in the death rate until the age of fifty-five.

Although there has been this steady decline in death rate from tuberculosis in general, there has been an actual increase in the case of the young adult female.

TABLE 5

Variation in Death Rate from Pulmonary Tuberculosis between 1901–1929 *in England and Wales*

Ages.	Male.	Female.
10–15 . .	42% decrease	42% decrease
15–20 . .	21% ,,	No change
20–25 . .	36% ,,	4% increase

TABLE 6

Death Rate from Tuberculosis of all Forms in England and Wales

Age.	1920.			1927.			1934.		
	Persons.	M.	F.	Persons.	M.	F.	Persons.	M.	F.
0–1	1,405	819	586	718	407	311	413	242	171
1–5	3,988	2,207	1,781	2,284	1,269	1,015	1,308	693	615
5–10	1,631	811	820	1,117	559	558	682	337	345
10–15	1,830	738	1,092	1,135	437	698	749	335	414
15–20	3,953	1,642	2,311	3,559	1,428	2,131	2,313	901	1,412
20–25	4,557	2,142	2,415	4,598	2,094	2,504	4,031	1,843	2,194
25–30	4,216	1,940	2,276	4,175	1,964	2,211	3,653	1,748	1,905
30–35	3,854	1,993	1,861	3,485	1,720	1,765	3,163	1,678	1 485
35–40	4,052	2,268	1,784	3,254	1,907	1,357	2,612	1,494	1,118
40–45	3,675	2,233	1,442	3,303	2,083	1 220	2,373	1,473	900
45–50	3,245	2,088	1,157	3,134	2,138	996	2,483	1,707	776
50–55	2,633	1,750	883	2,645	1,847	798	2,286	1,662	624
55–60	1,885	1,312	573	1,849	1,284	615	1,912	1,402	510
60–65	1,397	883	514	1,364	912	452	1,309	927	382
65–70	943	585	358	877	540	337	926	637	289
70–75	417	207	210	399	247	152	400	232	168
75–80	178	84	94	260	105	95	182	102	80
80–85	63	25	38	67	25	42	77	35	42
85	22	10	12	—	—	—	—	—	—

TABLE 7

Death Rate from Pulmonary Tuberculosis in England and Wales

Age.	1920.			1927.			1934.		
	Persons.	M.	F.	Persons.	M.	F.	Persons.	M.	F.
0–1	219	127	92	91	47	44	57	39	18
1–5	760	413	347	378	213	165	148	83	65
5–10	467	202	265	261	122	139	128	69	59
10–15	974	311	663	587	166	421	315	101	214
15–20	3,124	1,225	1,899	2,917	1,088	1,829	1,886	688	1,198
20–25	4,048	1,863	2,185	4,125	1,845	2,280	3,587	1,604	1,983
25–30	3,738	1,734	2,004	5,825	1,791	2,034	3,307	1,570	1,737
30–35	3,509	1,828	1,681	3,208	1,585	1,623	2,896	1,527	1,369
35–40	3,747	2,133	1,614	3,021	1,778	1,243	2,393	1,371	1,022
40–45	3,972	2,071	1,311	3,083	1,980	1,103	2,174	1,378	796
45–50	2,972	1,952	1,020	2,910	2,015	895	2,329	1,624	705
50–55	1,410	655	755	2,463	1,756	707	2,122	1,576	546
55–60	1,710	1,204	506	1,688	1,156	532	1,751	1,316	435
60–65	1,226	812	414	1,223	842	381	1,222	889	333
65–70	709	519	290	707	480	277	809	580	229
70–75	329	175	154	332	210	121	340	209	131
75–80	136	64	72	153	91	62	148	85	63
80–85	36	17	19	45	19	26	50	25	25

References

(1) ALLEN, F. T. *Tubercle*, 1932, XIII., 241.
(2) ZIMMERLI, E. *Brit. Journ. Tuber.*, 1936, XXX., 62.
(3) Registrar-General's Statistical Review of England and Wales, 1935.

CHAPTER I

IMMUNITY AND INFECTIVITY

WHEN tubercle bacilli enter the body they are attacked by polymorphonuclear leucocytes which may kill them or be killed. They are then carried into the lymph stream, where, together with the dead leucocytes, they are ingested by large mononuclear cells. Here again the bacilli may all be killed, but if not, the survivors reach a lymph node, where they multiply and form a tubercle. The tubercle is composed of a giant cell which probably is made from dead mononuclear cells with many nuclei, clear necrotic protoplasm, and around this layers of epithelioid cells flattened like the skins of an onion. Outside this is a layer of lymphocytes and here there may be fibrosis. The centre of the tubercle tends to liquefy partly because it is avascular and partly because of the toxic action of the bacilli. The tubercles often fuse one into another, break down and produce caseation, and when the lesion is in the lung the caseous material may be discharged through a bronchus and appear as sputum leaving a cavity in the lung.

The clinical manifestations of the infection depend upon the fate of the bacilli, which may :—

1. Be destroyed before they have produced any symptoms of ill-health.

2. Produce definite lesions which may heal completely or only partially. Those lesions which do not heal completely may remain localised and quiescent for years, but tend to spread at times either locally or by the blood or lymph streams, or by the bronchi. Thus the caseous, fibro-caseous or fibroid type of disease is produced.

3. Spread rapidly throughout the lungs, causing acute tuberculous broncho-pneumonia.

Which of these three results follows infection depends upon several factors, the chief of them being dosage in proportion to the size and resistance of the patient and to treatment after infection.

The animal body may be protected against tuberculosis by :

1. Natural immunity.
2. Acquired immunity.
3. Tolerance.

1. *Natural Immunity.* The degree of natural resistance to tuberculosis and other diseases varies in different races of mankind and in different individuals. It also varies from time to time in the same individual ; for example, infants are immune from mumps, but susceptibility develops later. It may be increased in the individual by good feeding, rest or other means ; decreased by cold, fatigue or insufficient food, especially fats. Animals show great variation in their natural resistance to tuberculosis and Hamerton (1) states that tuberculosis is uncommon in wild animals. In captivity all birds and most mammals are susceptible, and the disease is also found in cold-blooded animals such as crocodiles, snakes and frogs. Camels are very resistant, but llamas, to which they are closely allied, are susceptible. The great apes and old-world monkeys are susceptible to both human and bovine tuberculosis, but gibbons are highly resistant.

In human beings susceptibility and resistance vary enormously in different races. As with animals the susceptibility increases if the people live under unnatural or unusual conditions. Chinese have great resistance to the toxic effects of tuberculosis. Hall (2) suggests that this is due to the length of time the Chinese race has been exposed to the disease. Tuberculosis was known in China 2,600 years before Christ. It is still widespread, but extremely chronic, so that a patient with extensive disease in both

lungs may be working with few, if any, symptoms up to a week or so of his death, which usually results from an acute exacerbation and generalised tuberculosis. Natural resistance varies with age and sexual activity appears to increase the susceptibility of the individual to tuberculosis.

This variation in susceptibility is due largely to alteration in natural immunity. It does not appear to have any relationship to acquired immunity. It is a striking fact that young women are not included in the fall of death rate from pulmonary tuberculosis, which in the last half century has dropped by more than 60 per cent. in England and Wales (see Table 5, p. 6).

It may be that at the onset of and during sexual activity some change occurs in the body which impairs its natural resistance.

A negative tuberculin test in one who has been exposed to infection indicates a good natural immunity or complete recovery from an infection.

2. *Acquired Immunity.* The immediate results of injecting tubercle bacilli into the body depend upon whether or not there are antibodies as a result of previous infection. If tubercle bacilli are injected intracutaneously into an animal not previously infected nothing happens for the first few days. Then at the site of the inoculation appears a nodule which gradually increases in size. This nodule is essentially composed of epithelioid cells, the origin of which is uncertain, but which eventually become fibroblasts and lead to a fibrosis around the bacilli. The formation of the tubercle or nodule is not inflammatory, but due to proliferation of the cells, and may be compared to the growth of a neoplasm. Some two weeks after inoculation a zone of inflammation appears around the tubercle, which breaks down, forming an ulcer and, at the same time, certain general signs of ill-health develop. This marks the end of the incubation period or primary stage, and the body is now allergic.

If tubercle bacilli are injected intracutaneously into an animal previously infected the course of events is different. There is an inflammatory exudative reaction within a few hours at the site of the inoculation and no latent period as in the case of a first infection. In addition to the local inflammation, there may also be focal inflammation around already existing lesions in other parts of the body. If the dose of re-infection is sufficiently large local ulceration may occur, but it tends to heal and any new nodular formation which occurs appears sooner than in a first infection and tends to subside in the tuberculous animal.

The reaction peculiar to the previously infected animal is inflammatory. The tissues of such an animal are hypersensitive to tuberculin or tubercle bacilli, and this condition of hypersensitiveness is spoken of as "allergy." In the case of a first infection no symptoms of ill-health develop until the animal tissues become sensitive to the tuberculous infection, that is, until allergy appears. This difference in reaction to inoculation in the allergic or non-allergic subject is known as Koch's phenomenon. It may be said of tuberculosis, as has been said of other infections, that the beginning of symptoms marks the beginning of the immune period. The development of allergy is not peculiar to tuberculosis. The introduction of typhoid bacilli into the body produces no immediate reaction, but later symptoms and a positive Widal reaction appear. Large doses of tuberculin which would be fatal to the allergic animal cause no symptom at all in the non-infected one, and the same applies to the injection of tubercle bacilli, except that after a latent period the bacilli, being alive, will cause a general tuberculous infection. The latent or incubation period may therefore be regarded as the length of time taken by the body to establish resistance.

The allergic animal is made acutely ill by the injection of tubercle bacilli, but provided it recovers from the initial reaction it will live much longer than one infected by a

similar dose of bacilli for the first time, and in the non-immune or non-infected animal allergy does not exist. Allen K. Krause (3) describes a series of experiments on the non-immune and the immune guinea-pig designed to work out the paths of transit of the tubercle bacilli. He found that the transit of the bacilli was very rapid in the non-immune but delayed in the immune animals, and also that when the viscera are invaded the number of bacilli found in the immunes in the early stages is much smaller than in the case of the non-immunes.

It is known that the rate of spread of tubercle bacilli from the site of infection to internal organs depends on the quantity or dose of infection both in immunes and non-immunes. It is not unreasonable, therefore, to suggest that some substances form in the body of the immune animal and by killing a number of the bacilli reduce their number and retard their rate of transit. There are certain objections to this view. There is no bacteriolytic action on tubercle bacilli treated with serum of normal or immune animals, nor is there any definite experimental evidence of the existence of immuno-lysins. No success has followed the numerous attempts that have been made to treat tuberculous patients with immune serum. There are probably substances in the body tissues which are antagonistic to the growth of tubercle bacilli, but there is no satisfactory evidence of the presence of any such substance peculiar to the immune state. It is well known that cultures of the bacilli if kept in one tube for more than a few months cease to grow and cannot be subcultured, and it is possible that bacilli which have been fixed in one part of the body for a considerable time lose their power of development. Living tubercle bacilli may be found in an old lesion which has ceased to spread and has apparently been healed for many years. Yet in order to live they must have oxygen and nourishment from the body, and therefore be open to action by any immuno-lysin should it exist.

It would seem that some other factor is concerned in fixing the bacilli and retarding their rate of transit through the body, and the zone of inflammation at the site of re-infection in an allergic animal is probably of importance in this respect.

Eventually immune animals which have been re-infected die of generalised tuberculosis but they live much longer. The important fact is that re-infection can take place and lead to a fatal result. In the immune a larger dose will be required to cause disease than in the non-immune, but given a sufficient dose the barriers of defence will be broken down.

After staying dormant in the body for years a lesion may break out and cause a local spread, or the bacilli may be carried by the blood-stream and produce a separate lesion. The infected body is not immune from its own tubercle bacilli and it cannot seriously be maintained that it is immune from those introduced from outside. Previous infection produces some degree of resistance and tolerance, and these factors, added to the individual's natural immunity, enable him to live with comparative safety in a civilised community because he can overcome the dose of infection he is likely to meet. He is not, however, protected against an excessive dose and if he is unlucky enough to get one he will develop active disease.

Some authorities regard tuberculosis following an initial infection as primary and that which occurs in patients already infected as secondary. As an example of one they mention the acute tuberculosis of infants and of the other the chronic disease of adults. The adult type is regarded not as a re-infection but as a later manifestation of infection contracted in childhood. According to this theory, when once an individual is infected, as shown by his reaction to tuberculin, he cannot again be infected, but may develop some late form of tuberculosis, just as the syphilitic subject may develop tabes. This belief has lost most of its disciples, and the view now held by the majority of physicians is that

re-infection can and does occur provided the dosage is sufficient to overcome the natural resistance, and that which is conferred by a previous infection and tolerance.

Opie (4) investigated a number of families in the United States and makes an important distinction between the first infection or childhood type, and the re-infection or adult type. He does not, however, regard the adult type of tuberculosis as a continuation of childhood tuberculosis, but as an exogenous infection acquired in adult life. He divided the cases into three groups :—

A. Those who had been in contact with sputum positive cases.

B. Those who had been in contact with sputum negative cases.

C. Non-contacts.

Of children under five years of age in group A, 75 per cent. reacted to tuberculin, but of those in groups B and C, only 30 per cent. reacted, and X-ray lesions were found in 37 per cent. of children under five in group A, and 1·2 per cent. in group B.

After twelve to fourteen years clinical tuberculosis had been acquired by those who had been exposed to contact with a sputum positive case between birth and nine years of age in 10 per cent., by those exposed between ten to fourteen years of age in 20 per cent., and by those exposed after fifteen years of age in 10 per cent.

Since exposure to infection increases the incidence of tuberculosis in the adult as well as in the infant, it appears that the adult can and does contract the disease by contact even if he is a reactor to tuberculin, or in other words, has been previously infected.

It has been urged in support of the view that tuberculosis cannot be transmitted from one adult to another that a husband or wife rarely contracts the disease from a tuberculous partner. Dickinson (5) investigated 1,685 married people with tubercle bacilli in the sputum and found 141

instances (66 men and 75 women) of the husband or wife also being infected. Midgley Turner (6) found conjugal tuberculosis in nine out of 110 cases. Infectious diseases are not all equally infectious and tuberculosis has a low grade of infectivity, so that the consumptive is not so dangerous to the community as the scarlet fever patient. Protection given by an attack also varies in different diseases. Small-pox gives practically complete, and pneumonia practically no, immunity against a second attack; tuberculosis does give some protection, or at least the first infection modifies the effect of subsequent ones. But with tuberculosis there can be no question of getting from a first infection an immunity in any way comparable to that obtained by small-pox, mumps or scarlet fever.

Acquired resistance may be considerable or negligible in degree as is suggested by the varying responses to the tuberculin test, which does not give merely a negative or positive result, for one individual may respond violently to a very weak solution and another give only a slight reaction to a strong one. Sensitivity, however, is not a direct index of resistance or immunity. Sensitivity resulting from infection produces a state of allergy as a result of which the body reacts to its specific protein.

Infection certainly produces allergy, but the connection between allergy and immunity is uncertain. Rich (7) showed that active immunity can exist or be produced without allergy and that allergy can be produced without conferring immunity.

Allergy may itself be a danger, and Wingfield (8) believes that the sudden appearance of new lesions, often considerable in size, which sometimes develop in the lungs of tuberculous patients is due to allergic reaction, and that if patients are desensitised so that they are not strongly allergic the risk of the development of one of these lesions is diminished.

Infection of a non-reactor (or so-called non-immune subject) renders him a reactor in some ten days, so if one

speaks of a primary tuberculosis at all it should be just that phase of the disease between infection and the development of allergy, a phase usually referred to in other diseases as the incubation period. Infection of the non-reactor with tubercle bacilli may produce no noticeable effect at all, or effects increasing in severity up to death in a few weeks from miliary tuberculosis. The result depends largely upon dosage, and obviously it would require a smaller dose to harm an infant than an adult.

A chronic irritation or slow-spreading disease, produces fibrosis, in acute disease there is no time for fibrosis to occur. In acute pulmonary tuberculosis fibroid changes do take place if the rate of progress is slowed, and the most acute type may be compared to others each a little less acute until a chronic fibroid type is reached. One merges into the other as day into night.

In practice one finds that exposure to intensive infections leads to acute disease. For example, a young woman developed acute tuberculosis a few weeks after she had been nursing her fiancé in a small room until his death from advanced tuberculosis. She had been brought up in London so was almost certainly a tuberculin reactor.

As an example of the chronic type may be mentioned three sons who lived with a tuberculous father. One got a job abroad before the father developed a severe cough and sputum containing tubercle bacilli, the other two remained at home. After three years one, aged twenty-two, developed a cough, and on examination was found to have a considerable degree of fibrosis in both lungs. The other, aged twenty, had no symptoms except a slight morning cough, but examination revealed fibrosis and a cavity in one lung. In neither case was there any serious impairment of general health. The third son, who had been living abroad, was examined and found free from disease.

Some people prefer to speak of super-infection rather than re-infection because they think that the word re-infec-

tion suggests having the disease over again just as one might have a second attack of pneumonia, whereas super-infection implies that the second attack differs from the first. It seems to me that there is no general appreciation of the fact that a primary infection going on to an acute and general tuberculosis is extremely rare. In the overwhelming majority of cases a primary infection leads to no noticeable symptoms whatever, so that most adult reactors are quite unaware that they have ever been infected. A few show signs in bone, joint, gland, lung or wherever the bacilli happen to become fixed. Chronic pulmonary lesions are not common in infants because bacilli can pass freely through their lungs, being less likely to be held up by blocked lymphatics or lymphoid tissue.

I would suggest that the so-called childhood type of tuberculosis is essentially the same as the adult type. The real mystery to me has always been why childhood tuberculosis is so commonly non-pulmonary, whereas adult tuberculosis is almost entirely pulmonary.

Krause (9) was struck by the fact that in the case of the rabbit infected by tubercle bacilli the early anatomical changes were not the same as in the guinea-pig. In the rabbit the lungs are chiefly affected, whereas in the guinea-pig the lungs frequently show no obvious disease although the tracheo-bronchial glands are seriously affected. He found that in the guinea-pig there is very little lymphatic tissue in the lung and that what there is is supplied by the bronchial arteries, whereas in the rabbit's lung there is considerable lymphatic tissue supplied by the pulmonary arteries. Now in the infant there is very little lymphatic tissue but large lymphatic channels in the lungs, whereas in the adult lymphatic tissue has formed and there is not nearly such free lymphatic drainage. May not these anatomical differences account in some degree for the different types of tuberculosis characteristic of child and adult life? After all, the majority of children do "fix"

the invading bacilli, not in the lungs it is true, but in glands, bone, etc., and this can hardly be explained by assuming lack of immunity. The so-called primary complex consists of a primary focus where the bacilli become fixed in the lung tissue and produce a node sometimes known as Ghon's focus. It was, however, described by Parrot thirty-six years previously. Parrot's law is that where a tuberculous bronchial gland is found there is always a lesion of lung from which the gland has been infected. In addition to this node the primary focus consists of infection of the corresponding glands and the connecting lymphatics. The primary node usually heals or becomes calcified, in which case it may be found by radiography many years later. Inflammation may, however, occur around the node and produce the so-called epituberculosis. This inflammatory lesion often clears up completely, but may break down and produce cavities or a spreading fibrocaseous type of tuberculosis.

Although one must bear in mind that a primary infection tends to spread quickly along the lymphatics, and there may have been considerable spread (chiefly dependent upon the intensity of the infection) before allergy occurs, when once the body becomes allergic the disease proceeds as in a secondary infection and can no longer be called primary, although it may be a direct continuation of the primary infection. The result will depend largely on the amount of spread which has occurred during the incubation period. It is said that this is never more than six weeks, so true primary tuberculosis is a very short phase in the disease.

It would seem that one of the chief effects of acquired immunity is to slow the rate of spread and thus give time for nature to achieve healing. This probably accounts for the fact that tuberculosis is a local and not a general disease. It may, like cancer, become generalised or produce metastases, but broadly speaking it must be said that tuberculosis spreads locally from its original site; thus

there may be tuberculosis of the lung, kidney, bone, etc., just as there is cancer of the lung, stomach, breast or other organ.

3. *Tolerance.* This must be distinguished from natural or acquired immunity. In tuberculosis it is best seen in the chronic fibroid type of case where extensive disease may exist with very little impairment of general health. Patients who are exposed to small regular doses of infection over a long period of time often develop a considerable degree of tolerance, so that extensive fibrotic changes may take place in the lungs before any definite impairment of health is noticed. Tolerance consists in resistance to the toxic effects of tuberculous infection and may be compared to the tolerance for alcohol in those who take frequent small doses.

Conclusions (10)

1. The human body has some degree of natural resistance to infection by tubercle bacilli.
2. This resistance is less than that of some animals and more than that of others.
3. Some individuals are more resistant than others.
4. The degree of resistance varies from time to time in the same individual.
5. Small repeated doses or a chronic lesion lead to tolerance to the tuberculous toxins.
6. A primary infection leads to a state of allergy which may be harmful if too great, but in a moderate degree produces an acquired resistance.
7. As with natural resistance the degree of acquired resistance and tolerance vary with different people and wax and wane in the same individual.
8. Natural resistance is strong enough to overcome an ordinary dosage of infection, so that the overwhelming majority of individuals overcome an initial infection without any clinical disease or even any signs of ill-health.

9. Acquired resistance is not so important as natural resistance, so that when once the natural resistance breaks down the acquired resistance alone is rarely sufficient to check the progress of the disease. Tolerance merely prevents infection from producing toxic symptoms.

10. An important factor in acquired resistance is to fix the invasion and so to slow down its rate of spread. This tends to make tuberculosis a local disease spreading chiefly by local extension.

11. As a result of this check natural resistance has time to overcome those bacilli which wander from the local lesion and often to overcome the local lesion itself.

12. The change which takes place in the body some days after infection by tubercle bacilli may be compared to the change which takes place after infection by typhoid bacilli when the Widal reaction develops. If in either of these diseases one wants to speak of a primary stage this should be the incubation or pre-allergic period. After this brief latent period all tuberculosis is essentially the same.

13. It is wrong to apply the word primary to a tuberculous manifestation merely because it is the continuation of an original infection, and secondary because there has been a latent period between the original infection and the onset of symptoms.

14. It is not true that the individual acquires immunity to tubercle bacilli from another host, but remains susceptible to those harboured in his own body.

15. In common with all infections a slowly spreading lesion produces fibrosis and a rapidly spreading one does not.

16. Repeated small infections therefore are either overcome by the combined effects of natural and acquired resistance, or local areas of fibrosis appear and lead to the so-called chronic fibroid type.

17. In the rare event of an overwhelming infection, endogenous or exogenous, acute tuberculosis occurs.

18. In human beings as in animals the fatal dose has some relation to the weight of the individual. Hence a dose that would prove fatal in an infant might be resisted by an adult.

19. The site of the lesion is a matter of great importance in prognosis. If the bacilli happen to get fixed in the meninges the result is fatal, if they happen to lodge in the elbow recovery is the rule.

20. Pulmonary tuberculosis is common in adults and non-pulmonary in children because the bacilli having reached the blood-stream, must go through the lungs. In the child the passage through them is easy, whereas in the adult they meet obstruction and tend to become fixed. Children have as much resistance to tuberculosis as adults have.

21. Infantile and adult tuberculosis are fundamentally the same.

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CHAPTER II

PREVENTION

THE two main principles of prevention are destruction of infection and increase of resistance in the individual. In countries where there is no tuberculosis the people cannot, of course, become infected, but they fail to get the protection afforded by frequent small infections. It has already been shown that the degree of protection thus afforded is variable, but since tuberculosis abounds in all civilised communities, some authorities hold the view that it is wrong to protect children too much from infection, for the tubercle bacilli introduced by milk and other sources go to build up immunity, and so prevent the individuals from developing clinical tuberculosis in adult life. Heimbeck (1) found that nurses who have been infected by tuberculosis, as shown by the tuberculin reaction, are less liable to develop clinical tuberculosis than those who have not, or whose infection has so completely disappeared that the tuberculin test is negative. His work suggests that far from a positive tuberculin reaction being a danger signal it is actually a sign of protection, and that a new infection shows its tendency at once to conquer and produce disease or be conquered and establish immunity. If it is conquered, some tubercle bacilli may remain in the tissues and keep alive for years, but they do not tend to become virulent, multiply, and cause disease. Their presence maintains some degree of resistance, and tends to prevent a fresh infection from producing disease. This protection wanes unless it is kept alive by repeated small infections. Heimbeck found that at Oslo 85 per cent. of the children aged nine gave a positive Von Pirquet reaction,

but of 222 military recruits 55 per cent. gave negative results.

Following this school of thought one would like to see everyone infected, not sufficiently to develop clinical tuberculosis, but enough to render them immune to the maximum dose of tubercle bacilli they are likely to meet in ordinary life. This, however, is an impossible state to obtain without danger. Even if we admit that a few tubercle bacilli swallowed with milk or inhaled from time to time do protect, we must also admit that an overdose will cause disease. Moreover, a dose that at one time is harmless may prove fatal at another, when the individual's resistance may be lowered by cold, fatigue, or some other condition. It would seem wrong, therefore, deliberately to expose people, and especially children, to infection.

On the other hand, it is pointed out that without infection there can be no disease, and that the chief principle of prevention should be to remove all sources of infection, or at least to dilute the infection as far as possible. Bad ventilation, stagnant air, small rooms, dirt, etc., all tend to concentrate infection, but in a civilised community much can be done to prevent this concentration. The patient with advanced disease who expectorates a large number of virulent bacilli and is too ill to be careful is one of the greatest sources of danger, and such patients should be treated in institutions where proper nursing and precautions against infection are available. If it is impossible to remove the patient precautions must be taken to prevent other members of the household becoming infected. In this country when tuberculosis is diagnosed the case must be notified to the local medical officer of health. In each district is a dispensary with a tuberculosis officer, who acts as a consultant in doubtful cases and advises as to diagnosis and treatment. The patient is told how to live, or in most cases taught in a sanatorium, so as to minimise the risk of infecting others. The patient's home is visited, if necessary cleaned and disinfected, and the tuberculosis officer examines the members

of the family who have come into contact with the patient and keeps them under his observation.

Examination of Contacts

It cannot be too strongly emphasised that the principal object of contact examination should be to find the primary source of infection, which often is some elderly relation or other member of the household. It is only too often that the children alone are examined, and frequently only those children who have some symptoms suggesting infection. It is certainly wise to try to detect disease in its earliest stage when treatment is most likely to be successful, but it is clearly wrong to send healthy children back into an infected household to await infection. Open-air schools or preventoria are excellent to improve the health of children and also to get them away from a household infected with tuberculosis, but they should not be sent home until it is free from infection.

Dr. Lissant Cox, speaking at the Annual Conference of the National Association for the Prevention of Tuberculosis at Southport in 1935, said: "Find, isolate, educate and treat the adult positive case" and thus safeguard the child. In any case of tuberculosis the members of the household should be examined primarily to detect the source of infection. The discovery of other members recently infected is of secondary importance.

At the same conference Dr. N. Tattersall said that too much stress was laid on malnutrition as evidence of tuberculosis, since the child who develops the disease is often well-nourished, and looks healthy. He suggested that a positive tuberculin test is not an indication for treatment, and it is necessary to find the dividing line between simple infection and significant disease.

One great value of the tuberculin test is that a positive result in young children would suggest the presence of an adult carrier in the home.

Absence of physical signs does not exclude the possibility of pulmonary tuberculosis. An X-ray examination should be made in every case except possibly those that failed to react to tuberculin.

Although tuberculosis may become arrested, a relapse is always possible even after a number of years, and so the word " cure " is rarely used in connection with the disease. As time goes by after the arrest of the disease the chances of relapse become less and less likely, so that care is especially needed during the first few years after treatment has been completed. Many a patient leaves a sanatorium feeling well, having no signs or symptoms of disease, and apparently completely cured, and yet experience shows that he is much more liable than the ordinary individual to develop active disease during the next few years. The same applies to the patient who has apparently recovered from pleural effusion or hæmoptysis. It is not generally recognised that latent tuberculosis may exist without producing any signs or symptoms of disease, but a patient often presents himself for treatment with advanced disease in spite of only a recent history of symptoms.

A great deal of work has been done in recent years, notably at the Phipps Institute, in order to detect latent tuberculosis before symptoms appear so that early treatment may prevent development of clinical disease. Opie (2) found that amongst the schoolchildren of Philadelphia for the age group five to fourteen 0·6 per cent. required treatment for clinical tuberculosis of the lungs. Drolet (3) estimated that in New York, of the 123,000 children born in 1930, 5,000 were infected at the end of the first year, but the total number of infants whose death was certified as due to tuberculosis was seventy-five.

Stiehm (4) tested with tuberculin 2,412 Wisconsin students and 30 per cent. gave positive reactions. These included seventy students from rural Wisconsin, of whom only 5·71 per cent. reacted, but of 110 students from the Eastern

States 48·2 per cent. were positive. Of the positive reactors 5 per cent. were found on radiography to have adult type of tuberculous infiltration.

Hall and Chang (5) found 17·8 per cent. supposed healthy young Chinese adults to be suffering from latent tuberculosis and 6·3 per cent. having lesions needing immediate treatment in spite of complete absence of symptoms. An old infection as indicated by calcium deposits at the hilum was present in 71·5 per cent. They are of opinion, however, that active disease develops no more frequently in individuals who have well healed latent pulmonary tuberculosis than in those who show no X-ray evidence of infection.

In this country Wingfield and Macpherson (6) made an X-ray examination of a large number of young people between the ages of fourteen and eighteen. They accepted as positive only those films showing "unmistakably abnormal shadows indicating very definite pathological changes in the lung parenchyma, having the appearance which was compatible with, and usually associated with, tuberculous lesions of the adult type." All evidence of Parrot's focus and calcified tracheo-bronchial glands, though numerous, was ignored. Examination of 2,881 films showed fifteen positive under this standard. In addition, ten other films showed abnormal shadows probably due to tuberculosis. Thus they found a definite incidence in 0·65 per cent. and a possible incidence of 1·08 per cent. They suggest that these lesions do not always disappear or remain latent for ever. During adolescence there is a great psychological strain which is often associated with hard work and sometimes with under-nourishment, and it is at this time especially that a latent lesion is apt to flare up into activity. They point out that since the evidence of clinical tuberculosis in civilised countries is very low in relation to the high incidence of infection it is unnecessary and unprofitable to attack the problem of the prevention of tuberculosis for the whole community along the lines of prevention of infection.

They suggest therefore that the high incidence of pulmonary tuberculosis amongst young adults might be reduced by detecting those with latent lesions and giving them appropriate treatment before activity occurs.

This work opens up big possibilities, but as the authors agree, the task of dealing with the prevention of tuberculosis along these lines is tremendous. Moreover, there can be no doubt that a considerable proportion of adult tuberculosis (as Opie, quoted on p. 14, and others have shown) is due to a new exogenous infection or super-infection. None the less, it must be admitted that modern methods of prevention have failed to reduce the enormous incidence and death rate of tuberculosis in early adult life, and this work should receive the fullest encouragement.

The Grancher System. In 1903 Professor Grancher introduced a system in France whereby healthy children were removed from tuberculous infected surroundings. Dr. Armand Delille, of Paris, referred to this system in a paper read at the International Congress on Tuberculosis held in London in July, 1921. He said that the method had met with considerable success, and was not only the most economical, but the most successful method of dealing with the tuberculosis problem. The Grancher system is based on the belief that children do not inherit tuberculosis, but acquire it by repeated infections, and it removes the children whilst still healthy from the infected homes and sends them to live with healthy families. This system of boarding out is employed in England and other countries, but greater stress is laid on the delicate children, or those who are said to be mildly affected, the healthy ones often being left at home. When possible the patient is removed to an institution, thus removing the source of infection and allowing the children to live a normal life at home.

Immunisation. There are three methods described by Petroff (7).

1. Injecting living virulent tubercle bacilli. He found

ten living virulent bacilli could produce tuberculosis in a guinea-pig. The bacilli were counted by Barber's method, and he tried to produce immunity in guinea-pigs (8) by giving gradually increasing numbers of bacilli at weekly intervals, as follows : one, three, five, eight, twelve, and so on. He found that this method gave fairly good immunity, but it was necessary to keep up the injections, and there is a great risk of causing the disease if the resistance of the animal fails, so that the proceeding was considered too dangerous to employ on human beings.

2. By using avirulent tubercle bacilli. Many attempts have been made to protect by using attenuated bacilli, and the latest preparation is known as B.C.G. This consists of bovine tubercle bacilli, attenuated by 230 passages in thirteen years on potato-glycerin and bile. Petroff's conclusions are that this is a safer method than when virulent bacilli are used, but the end results are not known.

3. By using killed bacilli. He thinks that an increased state of resistance can be produced by injecting dead tubercle bacilli, and, although the immunity is more partial than when living bacilli are used, it is free from danger.

It is generally admitted that the use of virulent tubercle bacilli is too risky, and that tuberculin or dead bacilli when injected do not give an adequate protection. It is, therefore, the attempt to produce immunity by using attenuated bacilli that is receiving most attention at the present moment, and a great deal of work has been done with B.C.G. (Bacille-Calmette-Guerin). Irvine (9) has given a valuable summary of the work and modern views concerning B.C.G. He concludes that it has not been proved to have caused progressive tuberculosis in man and that a certain increase of immunity is produced in man by B.C.G. vaccination. He thinks it should be given to children in tuberculous families, but it is not possible to say as yet whether it gives enough protection to warrant the reorganisation of our present scheme for dealing with tuberculosis.

A large number of infants have now received B.C.G., and although a few cases of subsequent tuberculosis have been reported, they are very rare, and it has been suggested that they were due to failure of the B.C.G. to protect against an accidental infection, and not to direct infection by B.C.G.

The disaster at Lübeck in 1930 gave support to those who regarded B.C.G. as not altogether free from danger. In this case a number of newborn children were given B.C.G. orally, and seventy-three died of acute tuberculosis. Over 100 children were inoculated at Riga with the same strain without ill-effect. The evidence in the Lübeck disaster points definitely to contamination. Another series of deaths after B.C.G. occurred in Hungary, at Ujpest. Two infants were found with cavities in the lungs and as this type of disease is so rare in infants investigations were made, and it was found that both had been given B.C.G. by mouth. A third similar case was then discovered, and nine other deaths following the administration of B.C.G. Of these nine the certified cause of death was pneumonia in three cases, bronchitis in two, convulsions, marasmus, measles and meningitis in one each, but it seems probable that some of these deaths were really due to tuberculosis.

A third series occurred in Chile, where 10 out of 60 infants received B.C.G. at birth. The 50 unvaccinated remained well, but 4 of the 10 vaccinated developed tuberculosis.

Enquiry, however, threw considerable doubt on the view that in these outbreaks tuberculosis was really the result of the B.C.G. vaccine.

Irvine (9) points out that even if all the suspected cases really were infected by B.C.G., it would mean less than one disaster in 15,000 vaccinations.

So many cases of progressive tuberculosis following simple inoculation of B.C.G. into animals have been reported that they cannot be put down to coincidence or accident. At the same time Calmette admitted that tubercle formation occurs after inoculation, but regarded it as retrogressive and

harmless. Griffith (10) found it possible to kill a guinea-pig from shock with a large dose of B.C.G., but that a smaller dose produced a retrogressive harmless type of tuberculosis.

Petroff (11) plated out a culture of B.C.G. on gentian-violet egg and found two types of colony developed. Many with a rough surface which were non-pathogenic, and which he called R. A few with a smooth surface that caused progressive tuberculosis in guinea-pigs, and which he called S. These experiments were repeated by other investigators, some of whom confirmed Petroff's work and some did not. It has been suggested that bile-glycerin-potato is a good medium for R, but bad for S, so that by growing on this medium the culture becomes less and less virulent. It can, however, be made more virulent by changing to a medium favourable for the growth of the S part. The original culture was made avirulent on bile-glycerin-potato, and Calmette recommended that it should be subcultured on this medium after every ten passages on glycerin-potato.

Irvine points out that the controversy about the return of virulence under abnormal conditions might be avoided if it was agreed that B.C.G. was a "virus fixe" when cultured according to Calmette's instructions.

Of course, immunity is always relative, but there is no doubt that a considerable degree of protection is afforded by B.C.G. inoculations. Experiments on calves (12) showed that even a single inoculation of 50 to 100 mg. B.C.G. gave complete protection against 5 mg. virulent bovine tubercle bacilli injected a month later. The experiments on cattle by Lange and Lydtin (13) did not, however, give such favourable results. Calmette observed 982 babies whose inoculation with B.C.G. dated back more than a year and who lived with tuberculous persons, of them only 1 per cent. died of tuberculosis, whereas it was estimated that in Paris 82 per cent. of babies living with tuberculous parents died of tuberculosis. Greenwood (14) criticises these figures and suggests that the mortality from tuberculosis amongst

babies living with tuberculous parents is very much less than 80 per cent.

Baudouin (15) analysed the results of B.C.G. vaccination of 5,126 babies under the direction of the University of Montreal. It was found that definite protection was afforded especially to babies under twelve months, when the tuberculosis death rate for infants exposed to sputum positive cases was 8 per thousand for the vaccinated and 49 for the unvaccinated. Heimbeck (16) states that a considerable proportion of nurses joining the Municipal Hospital at Oslo develop tuberculosis. In 1927 44 nurses giving negative Von Pirquet test, but healthy, and most between the ages of twenty to twenty-five, were inoculated with B.C.G., and a year later not one of them had developed tuberculosis. In 1924, however, 13 out of 51 ; in 1925 17 out of 72, and in 1926 14 out of 62 nurses joining the hospital with negative Von Pirquet reactions developed tuberculosis. Wilbert's (17) experiments on apes went to show that a very satisfactory degree of immunity could be produced by B.C.G., although other workers have failed to obtain such promising results in apes. Stanley Griffith (18) at Cambridge conducted a series of experiments with monkeys, and found that B.C.G. did not prevent them contracting the disease from infected monkeys living in the same cage.

When B.C.G. is given by mouth as advocated by some for infants its value is doubtful, and it produces a positive Mantoux reaction in only 20 per cent. of cases. B.C.G. given subcutaneously, however, causes the animal or human being to acquire a positive reaction to tuberculin in some 90 per cent. of cases, although the sensitiveness may wear off and leave the subject once more negative to tuberculin.

In spite of varying experimental reports it would seem certain that B.C.G. injected subcutaneously does give relative immunity for a time, although not sufficient to do more than delay the spread of the disease and increase by a short time the life of the infected animal.

Fried (19) says that B.C.G. does not cause tuberculosis, but retains its antigenic properties. As long as living bacilli are in the body immunity persists, for there is a struggle between the body and the bacilli. He suggests that immunity in babies lasts about four years.

It has been shown that the most serious age for pulmonary tuberculosis to develop is about puberty. At this time the mortality is higher than at any age including infancy, and since tuberculin tests show that the majority of children at that age have been infected, it would appear that by the time puberty is reached either resistance has waned or some other change in the body has taken place and rendered it insufficient. It would seem that the most important time to increase immunity is just before and during the onset of puberty.

Desensitisation. Wingfield (20) points out that a definite proportion of patients relapse shortly after what appears to have been a successful course of treatment. By relapse he does not mean the gradual return of symptoms (though this may occur), but the appearance of definite fresh lesions in parts of the lungs which were previously believed to be healthy. He thinks these secondary lesions are due to a hæmatogenous deposit of tubercle bacilli in the lung with a surrounding area of acute inflammation as a result of allergic reaction. In the middle of this inflammatory area is a small core of tuberculosis which may persist and spread after the inflammation has subsided. He admits that the allergic reaction may to some extent be beneficial, but regards hypersensitivity as a definite danger. In order to prevent these violent allergic reactions he advocates giving a course of tuberculin to patients when they are beginning to take up life again after sanatorium or other treatment. It is easy and safe to desensitise patients to large doses of tuberculin and if they are hypersensitive it seems a wise procedure to do so.

Milk Supply. Milk is one of the cheapest and best of

foods, and its value for schoolchildren as well as for infants is becoming more and more recognised. No one would dispute the importance of a clean and pure milk supply, but at the same time tuberculosis of bovine origin is a minor problem compared with the human form, which claims twenty times as many victims.

The bovine tubercle bacillus is distinct from the human variety, and it has never been proved that one can change into the other.

Savage (21) says that 6·5 per cent. of raw market milk in this country contains tubercle bacilli.

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CHAPTER III

DIAGNOSIS

THE diagnosis of pulmonary tuberculosis is often a matter of great simplicity, and may, indeed, be quite obvious to the non-medical observer. In such cases, however, the patient has usually passed the stage when treatment can be of much value. In the early and doubtful cases the diagnosis is complicated by the fact that many patients who are infected and have evidence (by physical signs, X-ray, tuberculin, or other tests) of old disease are not actually tuberculous in the sense that they have active or spreading disease. Such a patient may be compared to a house in which there has been a fire, but which is no longer on fire. In other words, they do not require treatment for tuberculosis, though they may require treatment for bronchitis, overwork, Graves' disease, or some other condition to which their symptoms are really due. In certain cases a period of rest is required whether or not the symptoms are due to tuberculosis, and there is no better treatment than can be obtained at a sanatorium. Here the rest and routine have a rapidly beneficial effect. In many cases, however, a mistaken diagnosis not only leads to treatment which if not really harmful is useless, but prevents the patient from receiving the correct treatment for his actual disability.

It is possible to arrive at an accurate diagnosis if one remembers that no one sign, symptom or laboratory test can be relied on alone, but that the cumulative effect of several signs or symptoms all pointing one way may make a diagnosis certain. For example, loss of weight may be due to diabetes or a variety of conditions, but if other causes are eliminated and it is associated with changes such as night

sweats, cough and evening temperature, it becomes a very valuable piece of evidence in favour of active tuberculosis. The symptoms most often noticed in the early stages are :—

Loss of Energy. This is very frequently present, but is not often the complaint for which the patient seeks advice, as it is usually attributed to overwork or some other cause, and not taken seriously unless it becomes extreme. The patient feels tired after an ordinary day and is disinclined for any extra activity, such as going out to the theatre ; he is said to be growing lazy or suddenly to have become an old man. When such symptoms are given tuberculosis should be remembered as a possible cause.

Loss of weight is very common, but not constant, in the early stages of active tuberculosis. It is especially valuable as a guide to activity, and in chronic cases, when the disease is apparently arrested, a gradual loss of weight is often the first sign of renewed activity. Patients often put on superfluous fat during treatment and lose some of it on returning to a normal life. This loss must not be confused with wasting due to disease.

Cough is almost always present, but it has no special characteristics. As in bronchitis, it is most common on rising in the morning and after meals. It must be remembered that a chronic cough occurs in many other conditions, such as pharyngitis, nasal catarrh or excessive smoking.

Sputum is absent in the early stages, but appears as the disease progresses. At first it is mucoid, then purulent, and, in the advanced stages, it is mummular.

Tubercle bacilli in the sputum are definite evidence of infection, although there are patients who remain quite well without any evidence of active or spreading disease, but who have tubercle bacilli in the sputum either constantly or periodically. Tubercle bacilli are not often found in the earliest stages of disease, but when the sputum becomes purulent they are almost always present if the disease is due to tuberculosis.

Kingston Fowler (1) reported a series of 188 cases in which tubercle bacilli were found at Midhurst. In these the bacilli were found on the first examination in 167 and on the second in twelve. In one case they were not found until the seventh time. Repeated examinations are necessary, therefore, but at the same time failure to find them after one, or certainly two, examinations is strong evidence against tuberculosis provided the examination is thorough and the sputum is really purulent.

Cohen and Burton Wood (22) state that if a patient coughs during examination of his larynx he is likely to spray the mirror with tubercle bacilli, and that by this means the bacilli may be found before there is any expectoration. They advise making the test early in the morning and say that the mirror should be held with its surface horizontally above the larynx and the patient asked to give several short coughs which spray the mirror with secretion. The surface of the mirror is drawn along a microscope slide in order to leave a film which can be stained and examined in the ordinary way. The test frequently enables tubercle bacilli to be found before there is expectoration.

If the bacilli are not found a guinea-pig may be inoculated and the injection is best made into the groin. After a week or ten days enlarged inguinal glands will be felt in positive cases. A local swelling appearing after a day or two is due to other organisms and soon subsides, but the subsequent development of an inguinal lump indicates tuberculosis in the great majority of cases. Later, of course, the tuberculosis will spread in the guinea-pig and the diagnosis can be confirmed.

Recently the presence of tubercle bacilli in the sputum has been demonstrated by culture in cases where sputum has been repeatedly negative on direct microscopic examination. Some pathologists claim that the culture method gives more accurate results than does guinea-pig inoculation.

In cases where sputum is swallowed examination of

stomach washings microscopically, by guinea-pig inoculation or by culture, may show the presence of tubercle bacilli and is especially valuable in young children. The bacilli may also be discovered in the fæces in cases where there is no sputum.

The important point to remember regarding tubercle bacilli is that their absence in definitely purulent sputum, after repeated examination, is very strong evidence indeed against tuberculosis.

Cummins and Williams (23) have described a case which on clinical and radiological examination suggested acute pulmonary tuberculosis and numerous acid-fast bacilli were found in the sputum. On culture, however, they were found not to be tubercle bacilli. Details of their cultural characteristics and effect on animals were given by Griffith (24). In this case the only feature unusual in acute tuberculosis was clubbing of the fingers. The final diagnosis was pneumonitis around an abscess, and the patient made a complete recovery.

Branch (25) has studied acid-fast bacilli, other than mammalian tubercle bacilli, which may infect man. He states that some of these belong to the avian group, but others appear to be new strains of pathogenic acid-fast bacilli.

Fever. A certain amount of fever, usually in the evening, is common in cases of active tuberculosis. If the patient is non-febrile whilst in bed a rise of temperature may be obtained after he has taken some exercise. It is not sufficient, therefore, to take the temperature only whilst the patient is resting. A definite rise to 99·6° F. or higher after a brisk walk is a suggestive sign.

The range of temperature in pulmonary tuberculosis is an exaggeration of the normal. A rise begins about 2 p.m. and reaches its maximum about 7 p.m. After this it gradually falls until about 5 a.m., when it is subnormal until the afternoon rise. In other words, the characteristic temperature is

intermittent, being lowest in the early hours of the morning and highest in the evening. The greater the activity of the disease the more is the swing exaggerated, and in extreme cases the temperature is called "hectic," and this usually indicates great activity. A continuous typhoid type of temperature may be found and is of serious import, for it suggests miliary tuberculosis or a general spread of the disease. In some cases the temperature is higher in the morning than at night, this is known as the inverse type. The characteristic type, however, is the important guide to diagnosis in a doubtful case, and if it is not found in the resting patient it may be brought on by exercise.

Night sweats are present in the early acute stages of pulmonary tuberculosis or during an acute exacerbation in a chronic case. Some patients who sleep with too many bed-clothes feel hot during the night and speak of this as having sweats. True night sweats may be very severe, so that the patient has to change his night clothes two or three times in the night. Such cases are very suggestive of acute tuberculosis, and most of them have a characteristic smell. These symptoms are rarely present, however, except when the patient has many other signs which leave no doubt as to the true condition, so that they are rarely of great diagnostic value.

Digestive Symptoms. Loss of appetite and nausea are sometimes, though rarely, the first symptoms noticed. When a patient complains of a gradual impairment of digestive powers the possibility of pulmonary tuberculosis should be borne in mind.

Loss of Voice. Simple laryngitis with hoarseness or loss of voice frequently occurs in cases of pulmonary tuberculosis and may be an early symptom.

Pleural Effusion. Unless there is any obvious cause, such as neoplasm, cardiac failure, etc., the development of pleurisy or pleural effusion may be taken as evidence of tuberculosis. The infection comes from a pulmonary lesion which may not, however, develop into activity.

On the advent of any signs or symptoms suggesting pulmonary tuberculosis a past history of pleural effusion strongly supports that diagnosis.

Hæmoptysis. This may be due to mitral stenosis, bronchiectasis or other causes, but is usually due to tuberculosis and should be assumed to be of this origin unless there is evidence to the contrary. There may be veins at the back of the throat, but they do not bleed. It may be said that for all practical purposes in cases of hæmoptysis the blood does not come from the back of the throat. Many lives have been lost by the failure to appreciate the enormous importance of hæmoptysis as an early sign of tuberculosis of the lungs.

A **fistula-in-ano** is another condition which should cause serious suspicion.

Physical Examination. The study of physical signs is becoming more and more neglected as facilities for X-ray examination increase. Before entering for the final examination most students can interpret an X-ray film of the chest fairly well, but their ignorance of physical signs is often very apparent. It is true that a lesion deeply situated in the lung with normal tissue between it and the chest wall may exist without any physical signs whatever. A patient after hæmoptysis may be told there is nothing seriously amiss because no physical signs are found, and it is undoubtedly wise to impress upon students that disease, and even extensive disease, may be present in spite of the absence of signs. At the same time the modern tendency to regard the stethoscope as a museum specimen is a mistake. Physical examination will often enable the experienced physician to obtain information which laboratory and mechanical methods will not disclose. The patient is a human being and not a machine. Prognosis will be much more accurate, and treatment more satisfactory, if based on a knowledge of the patient from personal examination rather than on an X-ray film or report on the blood. It

sometimes happens that a pulmonary lesion is present, but cannot be detected by radiography.

The examination should be thorough but not unduly drawn out, and it is important to avoid seeking for unnecessary signs. For example, if a large cavity is discovered it is wrong to try and elicit the cracked-pot sound or test the difference in tone on percussion with the patient recumbent or erect, for these signs are superfluous.

Inspection should include an estimate of the patient's build, facial expression and temperament. One should note whether he sits still or fidgets, is calm or excitable, and his attitude to life. Prognosis is better in the case of a patient with a placid temperament.

Fingers should be examined for clubbing, which is not common in tuberculosis, but occurs early in abscess of lung and is almost always present in bronchiectasis.

The shape of the chest is not often important as regards tuberculosis, although, of course, there is flattening in chronic cases.

Slight differences in movement of the two sides of the chest may be noted, but this does not indicate early disease. A case (2) was recently described where the patient breathed almost entirely with her left lung, and, as no signs of disease could be found, it was regarded as hysterical.

Palpation will disclose the position of the cardiac impulse, a most important point, and the rate of the heart and pulse should be noted.

The value of percussion has been overrated, and slight differences in the note here and there are unimportant. Emphysema may mask the underlying condition of the lung. Scoliosis may produce an altered percussion note and so may muscular development. Absence of part of the pectoral muscle will produce flattening and increased vocal fremitus.

The accessory lobe of the azygos vein may produce signs suggesting tuberculosis (8). Normally the azygos major runs

up the mediastinum arches over the root of the right lung and enters the posterior aspect of the superior vena cava. Sometimes the azygos vein runs more laterally, indents the apex of the right lung, and enters the superior vena cava at a higher level. It carries with it both layers of pleura, which thus produce a septum known as the meso-azygos. Crepitations due to catarrh are sometimes heard over the azygos lobe, but they are not constant, and vary with cough. There is often some impairment of percussion note and altered breath sounds, but the diagnosis can only be made by X-ray, which shows a characteristic shadow, Plate IV.

Krönig's areas are the resonant bands at the apices, and naturally they are decreased or even absent if the apex is contracted by disease, but no patient should be condemned because of their absence or, alternatively, be regarded as fit. It is important to percuss the bases and axillæ and to note the expansion of the diaphragm by percussion during full inspiration and expiration.

Auscultation is the most important method of examination, but here again there are pitfalls. Constant crepitations which persist or become increased, and are heard over the upper zone of the chest, are almost pathognomonic of tuberculosis. Fine crepitations may, however, be heard at the edges of the lung, over the manubrium, or where the pleura is deflected over the heart or at the bases. These usually disappear if the patient coughs or takes a few deep breaths.

Cavernous breathing at an apex may be due, not to a cavity, but to a displaced trachea in a case of fibrosis.

Slight variations in the character of the breath sounds are more likely to be due to emphysema or temporary occlusion of a small bronchus than to early tuberculosis. Tuberculosis usually affects the upper portions of the lungs, so that disease at or near the apices is probably tuberculous, but a basal lesion is more likely to be non-tuberculous.

In the survey of school children carried out in Canada (4),

1,892 children were examined physically, by X-ray and with tuberculin, and the following conclusions were reached :—

1. Impairment of resonance over any part of the chest may be due to conditions other than tuberculosis.

2. Broncho-vesicular, or still higher pitched breathing, may be present apart from tuberculosis and is normal under conditions of forced breathing.

3. The presence of râles in the chest usually indicates a non-tuberculous infection.

(The authors are referring to school children, and this conclusion must not be taken to apply to the râles heard at the apex of adults. Such râles are usually due to tuberculous disease.)

4. Physical signs (including Eustace Smith and D'Espine signs) pointing to enlargement of the mediastinal glands are present in conditions other than tuberculosis.

I do not want to underrate the value of physical signs, but one frequently meets a patient who has been condemned as tuberculous on the strength of slight impairment of resonance on percussion at one apex, or some such sign, and no other evidence of tuberculosis whatever. There has been a most complete examination of the chest, with full notes of even the minutest detail, yet there are few if any notes on the history, and no examination of sputum, no tuberculin test and no X-ray examination have been made.

Tuberculin Test. The most satisfactory tuberculin test is the intracutaneous one, known as the Mantoux test.

For this purpose four dilutions are prepared. Dilution 1 is made by adding 0·1 c.cm. of Koch's old tuberculin to 0·9 c.cm. of normal saline containing 0·25 per cent. of phenol. Dilution 2 is made by adding 0·1 c.cm. of dilution 1 to 0·9 c.cm. of the diluting fluid. Dilution 3 is made by adding 0·1 c.cm. of dilution 2 to 0·9 c.cm. of the diluting fluid. Dilution 4 is made by adding 0·1 c.cm. of dilution 3 to 0·9 c.cm. of the diluting fluid.

Since 1 c.cm. of O.T. contains 1,000 mg., it follows that :—

0·1 c.cm. of dilution 1 contains 10 mg. (0·01 c.cm.) O.T.
0·1 c.cm. ,, 2 ,, 1 mg.
0·1 c.cm. ,, 3 ,, 0·1 mg.
0·1 c.cm. ,, 4 ,, 0·01 mg.

so that dilution 1 is 1 in 10, dilution 2 is 1 in 100 and so on; the number of noughts corresponding to the dilution, thus dilution 5 is 1 in 100,000.

To perform the test the flexor surface of the patient's forearm, just below the elbow, is cleaned with ether and 0·1 c.cm. of dilution 4 is injected intracutaneously with a very fine needle. The injection should not be made subcutaneously, but into the skin, leaving a slightly raised white weal. In the case of children 0·05 c.cm. may be used. If, as often happens, there is no reaction after three days, 0·1 c.cm. of dilution 3 is injected in the same way, and, failing a reaction, 0·1 c.cm. of dilution 2 is injected three days later. The reaction occurs twenty-four to seventy-two hours after the injection and consists of a raised area of redness at the site of the inoculation. In very sensitive patients this may go on to vesiculation, or even ulceration, and for this reason it is wise always to begin with the lower dilutions. A positive reaction indicates infection, but not necessarily active disease. Westwater (5) states that the Mantoux test is lessened or suppressed by measles or scarlet fever owing to the rash itself. It is also suppressed over a rash due to the irritation of a mustard poultice. In cases of chicken-pox or diphtheria the Mantoux reaction is not suppressed. It is somewhat curious that this test is employed so little in this country, and there are two chief criticisms of it. First, that since it is positive in cases where the disease is not active it is of little practical use. In other words, a patient with a positive intracutaneous tuberculin test does not of necessity require treatment. This is true, but there is no one symptom, sign or test which does indicate active disease for certain, and a correct diagnosis can be made only by the cumulative weight of evidence. The second objection

is that every one except in very early childhood gives a positive reaction. This is a common belief, but is not the case. Out of fifty adult patients sent into my wards at the Brompton Hospital for diagnosis the test was negative in nine, and they were all patients suspected of having tuberculosis. In Dundas (4) (Canada), and the surrounding country, 1,392 children of school age were tested, and of these only 32 per cent. reacted to tuberculin. Krause (6) writes that in 1927 a skin tuberculin test was made on people in Framingham, and the result was 38 per cent. lower incidence than it was when a similar test was made there in 1917–19, and he points out that this result is consistent with the fact that in New York City the mortality from tuberculosis between 1898 and 1925 declined three times for the general population and six times for infants under two years of age. Lloyd and Dow (7) found that the incidence of reaction to the Mantoux test increases gradually from infancy to adult life. 58·3 per cent. children between the ages of ten and fifteen gave a positive reaction. They found the incidence greatly increased in children who had been in contact with an open case of tuberculosis. Lloyd and Macpherson (8) re-tested 700 children (164 of whom were contact cases) after an interval of eighteen months to two years. Of 303 positive cases 291 (96 per cent.) remained positive, and 12 became negative. Of 397 negative cases 343 (86·4 per cent.) remained negative and 54 became positive. The children were all under fifteen years of age. Not one of them developed clinical tuberculosis, and although some of those who changed from negative to positive had some illness, such as measles, the majority had no indisposition whatsoever.

The tubercle bacillus contains several components and in the preparation of tuberculin for testing that producing a skin reaction is alone required. These components vary in different samples, partly owing to the use of different culture media and partly to the use of different strains of bacilli.

Standardisation is of the utmost importance, and a purified protein derivation has now been produced by Long and Seibert. The reader is referred to the Supplement to the *American Review of Tuberculosis*, December, 1934, where a full account of the preparation, properties and uses of purified protein derivative will be found. It is claimed to be non-antigenic, and so does not sensitise or lead to the formation of antibodies, but to be as highly potent and specific in the tuberculin skin tests as are the ordinary tuberculins which contain also antigenic substances. It is stated that patients repeatedly tested by the ordinary tuberculins become sensitive, and so soon give a reaction, whereas P.P.D. cannot cause a specific inflammatory reaction in subsequent doses as a result of sensitisation. It can be obtained in the form of soluble tablets.

A negative tuberculin test is by no means uncommon, and is of the greatest importance in diagnosis. A positive test is of less value in diagnosis as it indicates infection only. Some believe that a condition of activity can be distinguished from one of quiescence by the severity of the reaction, but it is an undoubted fact that some people give a strongly positive reaction although they have no apparent illness, nor do they subsequently develop any disease, whereas others with definitely active tuberculosis react mildly. At the same time, when a tuberculous lesion heals the sensitiveness to tuberculin does become less as time passes, and so a strong reaction suggests a recent infection or activity. There are several factors, such as intercurrent disease, which blunt a tuberculin reaction, and a course of tuberculin will blunt it or even eliminate it altogether. It would seem unwise, therefore, to set up a fixed standard and say that those who give a reaction of such and such a violence to such and such a dilution have active disease. Lobban (9) found that as tuberculosis advances the tuberculin test becomes less sensitive so that stronger solutions have to be used to get a positive result. It is the apparently fit or the patient with

good resistance who reacts to the weak dilutions. As a result of the South African enquiry it appears that natives with a strongly positive Mantoux reaction were more likely to develop tuberculosis than were the non-reactors. Hypersensitivity does not mean increased resistance. A negative reaction in a patient who has been exposed to infection suggests good natural immunity. In one who has not been exposed to infection it is no test for natural immunity.

Another method of employing the tuberculin test is by the cutaneous method of Von Pirquet. A solution is made consisting of Koch's old tuberculin, 2·5 c.cm., glycerin phenol, 5 per cent., 2·5 c.cm., saline, 5 c.cm.—that is O.T. in a strength of 1 in 4. A drop of this solution is placed on the forearm and a slight scratch made over it. The scratch must be just sufficient to draw blood. If positive an area of redness will appear round the scratch in three days. This test is equivalent to 1 in 10,000 dilution by the Mantoux method.

Heimbeck (10) applied Von Pirquet's test to girls aged from twenty to twenty-five who were becoming nurses at the Municipal Hospital, Oslo, and in 1924 he found that out of 109 there were 51 negative.

He also found that after two years' work at the hospital practically all gave a positive result to the Von Pirquet test. Other interesting facts found by Heimbeck are that not only were about half the nurses negative to the test, but of 79 medical students 47 per cent. and of 222 military recruits 55 per cent. were negative; but he found 85 per cent. Oslo children at the age of nine positive to the Von Pirquet test.

Out of twenty-one medical students in London in 1928 I found eight negative to Von Pirquet's test.

Gunter (11) uses preparations of Tuberculin Albumose Frei (T.A.F.), and makes dilutions of 1 in 10, 1 in 100 and 1 in 500; he places a drop of each solution and a drop of normal saline as control on the forearm and makes a slight scratch over them. He writes: "A reaction with 1 in 10

tuberculin or stronger denotes inactivity, a reaction to 1 in 100 activity and 1 in 500 great activity. The degree of activity does not, of course, signify the seriousness of the condition of the moment, but it implies that active processes are going on, and whilst this is the case there is every probability of the disease progressing, unless steps can be taken to bring about arrest."

Halliday Sutherland thinks that cutaneous tests can show infection only, but that activity can be detected by the subcutaneous tuberculin method. In my opinion a tuberculin reaction by any method indicates infection, but cannot be depended on to distinguish between active and arrested disease. For the subcutaneous test Sutherland (12) suggests the following doses of T.A.F. injected into the subcutaneous tissue below the scapula. Each dose is made up to 1 c.cm. by adding diluting fluid which consists of 0·8 grm. sodium chloride, 0·5 c.cm. carbolic acid and 99·5 c.cm. water.

1st	dose	0·25	of	Dilution	4	and if there is no reaction three days later.		
2nd	,,	0·5	,,	4	,,	,,	,,	,,
3rd	,,	0·1	,,	3	,,	,,	,,	,,
4th	,,	0·2	,,	3	,,	,,	,,	,,
5th	,,	0·5	,,	3	,,	,,	,,	,,
6th	,,	0·1	,,	2	,,	,,	,,	,,
7th	,,	0·2	,,	2	,,	,,	,,	,,
8th	,,	0·5	,,	2	,,	,,	,,	,,
9th	,,	0·1	,,	1	,,	,,	,,	,,

If the Mantoux test has not previously been made or proved positive to 0·1 c.cm. of D_5, he starts the subcutaneous test at 0·25 D_4. If the Mantoux test was negative to weaker dilutions than 0·1 c.cm. D_4 he starts the subcutaneous test with 0·2 D_3 and if negative to dilutions weaker than 0·1 D_3 he starts the subcutaneous test with 0·1 D_2, and in any case increases subsequent doses according to the table. He regards a febrile reaction to any dose up to 0·2 c.cm. D_2 as proof of active disease. A negative reaction of doses between 0·5 D_2 and 0·1 D_1 excludes active tuberculosis. A positive reaction to dilutions as strong as 0·1 D_1 may occur apart from activity. A positive result produces :—

1. A local reaction consisting of redness and swelling at the site of the injection.

2. A general reaction consisting of malaise and a temperature which may rise to 102° F. or more.

3. A focal reaction which consists of inflammation round the various tuberculous lesions.

In the case of lupus this focal reaction can be seen; in pulmonary tuberculosis it may be indicated by X-ray evidence, increased crepitations, more sputum and, possibly, some hæmoptysis. A focal reaction is rare, and even when it does occur it is not often serious; indeed, when it subsides it may be followed by actual improvement. In my opinion, the danger of this test, and in fact of tuberculin treatment, has been much exaggerated.

Lastly, Moro's method of applying the tuberculin test may be mentioned. This consists of rubbing into the skin an ointment containing equal parts of lanolin and O.T. If positive, redness appears over the surface. Hamburger's modification consists in rubbing tuberculin ointment over the sternum and in positive cases papules appear in from two to seven days. These methods may be used for infants when objection is made to an injection or even to scratching the skin, but failing the intracutaneous method I should always advocate Von Pirquet's test.

Complement Fixation Test

The Wassermann test for syphilis proved so satisfactory that it was hoped to apply similar methods in the diagnosis of tuberculosis. In practice, however, the usual difficulty arose, namely, how to distinguish active from non-active infection. Moreover, a positive result not infrequently is obtained in those who are apparently well and remain so, whereas a negative result is sometimes obtained in patients with definite and active tuberculosis. Alcock, Douglas and Lucey (13) obtained a positive result in 90·6 per cent. T.B. positive cases, but it was also positive in 36·5 per cent.

T.B. negative cases. In arrested cases who had at some time been T.B. positive the test was positive in 47 per cent. only.

A positive complement fixation test may be taken as evidence in favour of the patient having or having had tuberculosis and a negative one as evidence to the contrary. Opinion as to the value of this test varies enormously amongst different workers, but the majority do not regard it as of any special value except as giving some additional evidence one way or the other in a doubtful case.

Sedimentation Test

I shall refer to this test when dealing with prognosis, and mention it here only to say that it has but little value in diagnosis. It is true that in the majority of cases of pulmonary tuberculosis the sedimentation rate is increased, but so it is in many other conditions, and the doubtful case of pulmonary tuberculosis where there were no toxic symptoms and a fair condition of general health would quite likely show a normal sedimentation rate. Heaf (14), after testing 150 patients at King Edward VII. Sanatorium, Warwickshire, wrote : " We wish to emphasise that the test is useless for diagnostic purposes."

Arneth Count

Arneth attached importance to the indentations or number of individual nuclei in the polymorphonuclear neutrophile leucocytes. He found in healthy individuals :—

Mononuclear forms with round or indented nucleus, 5 per cent.
Forms with two nuclei, 35 per cent.
Forms with three nuclei, 41 per cent.
Forms with four nuclei, 17 per cent.
Forms with five nuclei, 2 per cent.

In tuberculosis he found a predominance in the forms with one or with two nuclei. In one fatal case of pulmonary tuberculosis he found 46 per cent. with one and 49 per cent. with two nuclei. Professor Cummins (15) discusses the value of this test and the sedimentation test, and regards them of value as additional evidence.

Von Bonsdorff's modification of this method is to count the nuclei of 100 polymorphonuclear leucocytes. He takes the normal as 275 nuclei per 100 cells. When there are less than this it is called a shift to the left and the prognosis is bad, whereas a shift to the right indicates a good prognosis.

Blood Picture

Increase of monocytes and decrease of lymphocytes indicate activity.

Increase of eosinophils indicates a good prognosis.

Lymphocytes increase with healing, monocytes increase with tubercle formation, and polymorphonuclear leucocytes when there is breaking down tissue and suppuration. Houghton (16) has worked out an index the formula for which is V.B.$-$(P + M + S $-$ 2L) where V.B. is the Von Bonsdorff count, P the polymorphonuclear cells, M the monocytes, L the lymphocytes and S the sedimentation count. The index may vary from 0 to 800, and the higher it is the better the prognosis. Percentage of cells to total white count is used.

The following example illustrates a case with a good outlook : V.B. 280, P 50, L 41, M 6, S 4. The index would thus be $50 + 6 + 4 = 60 - 82 = -22, 280 - (-22) = +252$.

Bacillæmia

Löwenstein (17) claims to have obtained cultures of tubercle bacilli from the blood not only of tuberculous patients but of patients suffering from rheumatism, disseminated sclerosis and other conditions not usually regarded as tuberculous, by the use of a special technique. His

results have been confirmed by some workers, but others have failed to obtain tubercle bacilli from the blood even of definitely tuberculous patients although his technique has been strictly observed. Weatherall (18) conducted a series of experiments which are of special interest in view of conflicting evidence. Following Löwenstein's technique she made cultures from the heart blood of 40 tuberculous guinea-pigs, but tubercle bacilli failed to grow. From 30 sanatorium cases of pulmonary tuberculosis she also failed to grow the bacilli from the blood. Blood from 15 tuberculous patients was injected into guinea-pigs and only one developed tuberculosis. Pearce (19) made 53 cultures, and inoculation tests from 51 tuberculous patients. A definitely positive result was not obtained, but in 4 cases an acid-fast bacillus was found, but one which failed to infect guinea-pigs. Stanley Griffith (20) investigated 24 strains of recent blood cultures sent to him by Professor Löwenstein in 1932. The clinical diagnosis of the cases from which the cultures were made were 11 cases of skin tuberculosis, 8 of rheumatism, 2 of disseminated sclerosis and one each of dementia præcox, renal and pulmonary tuberculosis. He found that of these 24 strains, 17 were culturally and in virulence for the guinea-pig typical human bacilli, and 7 were typical avian tubercle bacilli. No evidence of any acid-fast bacillus other than tubercle was found in any of the cultures.

Wilson (21) conducted an investigation of tuberculous bacillæmia for the Medical Research Council and reached the definite conclusion that tubercle bacilli are not to be found in a variety of conditions such as nervous disease or rheumatism, but may occasionally be cultivated from the blood in the case of miliary or advanced pulmonary tuberculosis.

Vital Capacity

As with the sedimentation rate, vital capacity is altered by many conditions besides tuberculosis and is of more

value in estimating prognosis and the result of treatment than in aiding diagnosis. At the same time a normal vital capacity does undoubtedly render a diagnosis of active pulmonary tuberculosis very unlikely.

System of Diagnosis

At the Trudeau Sanatorium, Saranac Lake, New York State, stress is put on certain signs and symptoms which are considered of special importance, these are :—

Tubercle bacilli in the sputum.
Persistent râles in the upper part of the chest.
Parenchymatous lesion as shown by X-ray.
History of unexplained hæmoptysis.
History of unexplained pleural effusion.

In addition to this, symptoms of toxæmia, such as fatigue, loss of weight or strength, fever over 99° F. in men or 99·6° F. in women, pulse over 90 in men or 96 in women, are regarded as indicating activity if not explained on other grounds.

At Trudeau, patients sent for diagnosis of pulmonary disease are classified under four headings :—

Non-tuberculous. No direct evidence.

Suspected tuberculosis. A history of unexplained pleural effusion or unexplained hæmoptysis, but not both. No other evidence of a lung lesion.

Non-clinical pulmonary tuberculosis. Here X-ray shows a parenchymatous lesion not explained by other causes, but there are no symptoms and no tubercle bacilli have ever been found in the sputum.

Clinical tuberculosis. In this group there are, or have been, definite symptoms, together with one of the first three cardinal signs already mentioned or both unexplained hæmoptysis and unexplained pleural effusion.

This is a good system of diagnosis, but should be taken only as a guide and not as a rule. The physician must form

his opinion on the cumulative evidence just as a jury forms theirs in the law courts.

It is important to remember that there are two problems to be decided in diagnosis.

1. Is the patient infected with tuberculosis ? and if so,

2. Are the symptoms (or some of them) due to the tuberculosis or to some other cause ?

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CHAPTER IV

RADIOLOGY IN PULMONARY TUBERCULOSIS

THE great value of radiology in pulmonary tuberculosis is now generally admitted; indeed, the modern tendency is to overrate rather than underrate its importance. In order to obtain full value from X-rays the work should be done by a competent radiologist, and in chest cases especially considerable experience in interpreting the shadows is required.

Before studying the X-ray shadows of an abnormal chest it is necessary to understand those of a normal one, or rather of a healthy one (Plates I. and II.), for certain abnormalities which produce shadows are consistent with perfect health. Riviere (1) discussed the X-ray appearances of the healthy chest and pointed out that in infancy there is little to see in the normal lung field outside the root shadow, but as time passes trunks and twigs of the lung network become visible, and at school ages may be very striking, especially as regards strands passing out fanshape from the root. These appearances, which are certainly compatible with health, often become less obvious when adult life is reached and may be due to catarrhal attacks, infective fevers or fleeting tuberculous changes, but they certainly do not indicate active or clinical tuberculosis.

In the adult just outside the central shadow and partly covered by the heart on the left side can be seen shadows of the lung roots, and these are due to the main bronchi, blood vessels and lymphatics. Running out from these central root shadows are striæ passing into the lung tissue and composed of bronchi and their accompanying vessels. The bronchi appear as double shadows and look like railway lines

and the blood vessels as streaks resembling the branches of a tree. If seen in optical section, the bronchi may appear as vague rings or small cavities and the blood vessels as dense nodules which may be mistaken for calcareous glands if in the section there happen to be much crossing and recrossing of the vessels.

It is important to recognise the round breast shadow in the healthy chest; it may be very prominent, and I have known it to be mistaken for a tumour.

The nipple shadow (Plate III.) is sometimes very clear and has been mistaken for a tuberculous focus.

The shadow of the accessory lobe of the azygos vein (Plate IV.) should be recognised. It appears as a thin line running from the upper border of the right lung downwards and inwards to the second costal cartilage. It is called the comma-shaped shadow, the upper thin line being formed by the deflected pleura, and the thicker terminal portion by the azygos vein seen in section.

One must also remember that the lung forms a cupola fitting over the dome of the diaphragm, so that the dense shadow of the diaphragm does not indicate the lowest level of the lung, for there is lung tissue below this around the dome of the diaphragm. If there is gas in the stomach X-ray will show the lowest level of the lung below the dome of the diaphragm. Plate V. shows lipiodol at the extreme base of the left lung after the patient had taken a Seidlitz powder in order to produce gas in the stomach.

Lynham (2) finds it more frequent for the radiological report to indicate disease when the physician finds no signs than for the opposite to occur, and in my experience X-ray findings usually indicate more disease than would be expected from physical examination. Post-mortem examination shows that there is almost invariably more disease present than the physical signs indicated. It sometimes happens that physical signs are present and tubercle bacilli exist in the sputum, and yet no definite X-ray abnormalities are

noted. Such cases are rare, but they go to show that X-rays should be combined with other methods in the diagnosis of a difficult case and should not be relied on alone. Melville (3) said :—

1. Definite infiltration can be demonstrated upon an X-ray film at quite an early stage of the disease, and frequently before definite physical signs are evident.

2. X-ray evidence extending over a period of some months and consistently negative may be taken as conclusive evidence of the non-existence of pulmonary tuberculosis.

3. In cases where physical signs are present it may be assumed that the initial stage has passed, and in such cases the X-ray picture will show, as a rule, much more extensive disease than can be demonstrated by physical examination.

One common source of failure to interpret X-ray findings is the attempt to draw conclusions from a film alone. In chest work it is essential to screen the patient, as this will show the movements of the chest and diaphragm, hypertranslucency or failure of a certain area to light up normally during breathing, movements of heart and mediastinum during inspiration and expiration, etc., and one can, moreover, move the patient so that his chest can be examined from several angles. A film will often show detail which was overlooked on the screen, but no X-ray examination of the chest is complete without careful observation under the screen.

Another error is to centre the tube incorrectly and to have the patient in a faulty position, so that the shadows are distorted and misleading. There may be so much error that the heart appears on the right side of the chest in a normal case.

The tube should be at least 3 feet from the patient. Some like it to be 6 feet, as this gives a beam that is almost parallel and a clearer picture, but requires much more power or a long exposure.

There are two common heresies referred to by Melville (3).

One, that of so-called peribronchial tuberculosis. In certain cases, and especially those who have lived long in a city or suffered from chronic bronchitis, dense striæ are seen radiating into the lung tissue from the hilum, and when these were very dense they were supposed to indicate tuberculous spread along the lymphatics accompanying bronchi, and hence the term peribronchial phthisis arose. They are but an exaggeration of the normal linear markings, and the term peribronchial phthisis is not now used by radiologists, nor is such a condition known in the post-mortem room.

The other heresy is hilum tuberculosis. That enlarged bronchial glands may exist and be seen by X-ray, and that they may be of tuberculous origin is not denied. But that X-ray examination can detect tuberculosis of the mediastinal glands before the disease has spread into the lung tissue is another matter. The infection passes from the lung to the glands, and, although a tuberculous mediastinal gland may suppurate into lung tissue and set up acute disease, in the ordinary way the infection does not pass against the lymphatic stream from the gland back into the lungs.

On this subject Lynham (2) wrote: "I have seen many cases with root areas involved, but they all show lung tissues involved as well."

One must also remember that considerable exaggeration of the hilum shadows may exist without any enlargement of the glands. Lynham says that when screening, the patient should be made to inspire and then expire to his fullest capacity. It will then be found that "solid bodies, such as calcified glands, retain their shape; fibrotic areas show up at full inspiration like the fine branches of a moving tree; caseating areas resemble crows' nests in tree branches; whilst fluid areas, such as the large veins, undergo a change of shape which one can only describe as amœbic."

No mere heaviness of the hilum shadow or increase of the linear striation can be taken as evidence of tuberculosis (4). A simple inflammatory lesion (Plate VI.) may resemble

tuberculosis very closely. A fine mottling of the lung parenchyma usually just below and external to the midline of the clavicle is a common early radiological sign.

Kerley (5) states that Assmann's focus (Plate VII.) may be the first sign. This is a rounded shadow usually about the size of a sixpenny piece and most commonly in the infra-clavicular region. Streaky shadows showing lymphatic spread are seen leading from it to the hilum.

Ghon's primary focus (Plate VIII.) is a small rounded shadow seen in children, and usually in the lower lobe. It may be obscured by the shadow of the dome of the diaphragm. A triangular shadow (Plate IX.), with the narrow end at the hilum, extending to the axilla or apex, is sometimes an early X-ray manifestation and is probably produced by an inflammatory reaction around a tuberculous focus. It may lead to chronic tuberculosis of the lung, spreading from the local lesion, or largely clear up, leaving a central core of tuberculosis, but not infrequently it clears up altogether. The condition is known as epituberculosis or by the French as *spleno-pneumonie*. It produces a shadow not unlike that produced by collapse of a portion of the lung as a result of a bronchus blocked by carcinoma. Morlock and Scott Pinchin (6) suggested that the shadow might be due to collapsed lung resulting from enlarged glands pressing on a bronchus, and that when it follows injection of tuberculin it might be due to swelling of a tuberculous gland rather than to an allergic focal reaction in the lung.

Collapse of portion of a lung is occasionally seen in association with pulmonary tuberculosis, and is probably due to the blocking of a bronchus or bronchiole and absorption of air from the alveoli. It may at times be due to obstruction of a bronchus by contracting fibrous tissue.

Possibly the most important function of lung radiography is the detection of cavities. A cavity with a thick wall is unmistakable. The presence of a fluid level which is due

to secretion in the cavity and which shifts with change of position of the patient is often noted. In some cases (Plates X. and XI.) lipiodol will show up a cavity not previously seen, but it often happens that lipiodol fails to enter a cavity. In my experience no harm results from injecting lipiodol into a tuberculous cavity. Sometimes a cavity not seen in an antero-posterior film will show in a lateral or oblique one (Plates XII. and XIII.).

Recently tomography has been used to eliminate parts of the body lying in front of or behind the lesion which it is desired to radiograph and to avoid superimposition of shadows. Chaoul and Grossmann (7) of Berlin have designed a tomograph (Fig. 1) which consists of a pen-

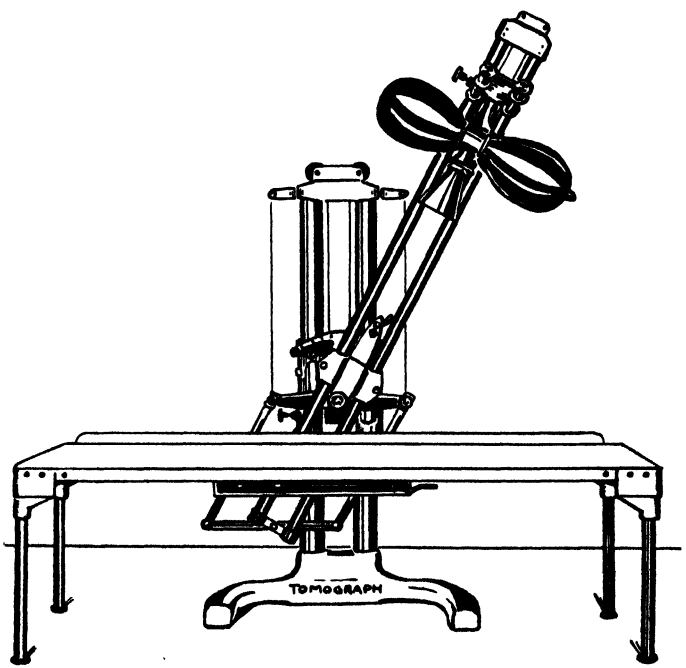

FIG. 1.

dulum revolving round a horizontal axis. At the top of the pendulum is the X-ray tube and at the bottom the film. The object to be radiographed, of course, rests between the tube and the film, and if the pendulum is moved during exposure, that part of the object at the centre of the axis will remain clear whereas the parts above or below will be blurred. Plates XIV., XV. and XVI. are examples of films taken by the tomograph.

Rounded shadows are often seen in the lungs of tuberculous subjects. These are known as annular shadows and are almost always due to cavities.

In a series of cases at the Brompton Hospital where an annular shadow was present during life a corresponding cavity was found in every case that came to autopsy.

The reverse, however, is not true, for at autopsy it was not uncommon to find cavities which had not been revealed by radiography during life, and it is in the detection of such cavities that tomography may prove valuable.

These annular shadows may on rare occasions be due to air bubbles or emphysematous bullæ, for otherwise it is difficult to account for the fact that in certain cases they appear and disappear in a series of radiographs.

The localisation of cavities and adhesions is best done by taking a series of films at different angles. In the case of a lung partially collapsed by artificial pneumothorax, but with an adhesion, screening in the antero-posterior position will show the general position of the adhesion. Now, if the patient is slowly revolved so that the shoulder on the healthy side becomes nearer to the screen and the other shoulder further away, the adhesion will appear longer if it runs anteriorly, and shorter if it runs posteriorly. If the shoulder on the pneumothorax side is turned towards, and the other shoulder away from, the screen an adhesion which runs anteriorly will appear shorter and one running posteriorly longer.

Stereoscopic radiography may be used to determine the

direction and position of adhesions and to localise foreign bodies, cavities or other lesions in the lung. Stott (8) discussed the optical principles involved in stereoscopy. He pointed out that whereas in solid vision many factors take part, in the stereoscopy of shadows only convergence is used. He concludes that it is probably safer for the clinician to depend on the antero-posterior and lateral flat films rather than risk an incorrect deduction from stereoscopic illusion.

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CHAPTER V

PROGNOSIS

In no disease is the prognosis so uncertain as in pulmonary tuberculosis, and it is quite impossible to foretell the future of any given case. At the same time if a large number of cases are taken it is possible to give a correct prognosis in a big percentage.

Physique. Patients who are physically weak, and especially those who have badly developed or deformed chests, tend to do badly if they become tuberculous. Insurance statistics show an increasing mortality from pulmonary tuberculosis as the weight of the individual falls below normal.

Temperament. The temperament of the patient is also of the greatest importance. Kingston Fowler (1) wrote: "No fool ever gets rid of tuberculosis of the lungs. He may be no fool in relation to literature, science or art, but if he is so in relation to his own well-being he is doomed for certain." The patient with a placid temperament who takes things as they come and does not look too far ahead has the best chance of recovery. During the active stage he will follow out the treatment for his own sake, and, having reached the highest level of improvement he will adjust his life so as to get the most out of it, in spite of any limitation of activity which may be necessary. Many people in all walks of life have done much useful work and left great names behind them in spite of having suffered for years from chronic pulmonary tuberculosis, and probably in many cases their lives were prolonged by the fact that they did work. It is play, far more than work, that kills in tuberculosis. If, therefore, one finds a patient steadily

working under suitable conditions and not worrying about his health the prognosis is always hopeful.

It often happens that a patient with a temperament that is bad for a tuberculous subject is able to control himself, and eventually becomes reconciled to his condition, but many patients are never able to change, and for them the prognosis is grave. It is often possible to help a patient. If, for example, he is getting restless and unsettled after some months in a sanatorium, a few weeks' leave at home or with friends may make all the difference, and enable him to return and continue the treatment. It is the monotony of life which tells on a certain type of patient after a time.

There are three types which are especially bad :—

1. The patient who finds the necessary restrictions so extremely irksome that he has to disregard them in order to prevent a mental breakdown. The same applies to the patient who has not sufficient self-control to regulate his life to suit his state of health.

2. The patient who is over-nervous and unable to interest himself in anything except his own health. This class of patient is continually taking his temperature and is in a constant state of anxiety over the slightest ache or symptom that may arise. He will not take up any employment even when well enough to do so, and very rarely lives long, in spite of the great amount of trouble and thought he takes of his own health.

3. The opposite type of patient, who cannot realise that there is any need to take precautions unless he has some definite symptom, such as hæmoptysis, is also unlikely to do well. When he is febrile and feeling ill he will consent to go to a sanatorium and do his best to get well, but on leaving the sanatorium, if he has no symptoms and feels well, he will play games and return to his ordinary life, not from lack of self-control, but because he thinks he is cured. An attack of hæmoptysis or pleurisy will again lead him to seek advice, but as soon as he feels well he once more returns to

a life of full activity. He is quite willing to receive treatment when he has a breakdown, but does not see the necessity of taking any steps to prevent one.

As an example of this type of patient the following case may be mentioned. A patient, aged thirty-two, who had always been healthy, lived an active and athletic life and weighed 11 st. 5 lb. In April, 1928, he caught a cold, which was followed by a cough, sputum developed and tubercle bacilli were found. On examination occasional crepitations after cough were heard at the right apex, but there were no other signs. X-ray showed a slight shadow at the right apex. He was non-febrile and felt well in himself. He was strongly advised to have a period of rest, followed by sanatorium training, and the prognosis was considered good. However, he refused to rest or go into an institution, saying that he was not ill enough for sanatorium treatment, and felt sure that he would get well after a short open-air holiday with plenty of milk and good food. He took a holiday, playing golf and living what he thought an ideal life, but in a month he had lost a considerable amount of weight and began to feel ill. In June he had extensive signs in the upper part of the right lung, crepitations being heard as low as the angle of the scapula behind. At the root of the left lung crepitations were heard also, and X-ray showed great extension in the right lung and shadows in the upper part of the left. He rapidly got worse and died in December. If he had had a large hæmoptysis at the beginning of the illness it would probably have saved his life, as he would have treated the condition seriously from the onset and had treatment in the early stages of the disease. Some patients who have arrived at a chronic stage of the disease cannot realise that they must adjust their lives to meet their disability. There may be dyspnœa owing to extensive fibrosis, though no active disease. The patient will argue that he cannot be well because he is short of breath when he runs upstairs, and therefore he needs treatment. He will

not meet the difficulty by abstaining from running upstairs, but tries treatment after treatment. This is the type of patient who tries the various quack "cures." Most of these cures are harmless, and often produce a temporary benefit if the patient has faith. Some, however, are very expensive, and indeed it is occasionally the high cost which attracts the patient. "There are some fools whom none but knaves can serve."

Steady work or an interesting hobby is the best treatment for this type of case.

Environment. It is necessary, of course, to distinguish between a bad temperament and inability to have the necessary treatment, or to lead the correct life owing to financial or other reasons. The patient whose home conditions are good, and who can afford a reasonable amount of comfort, has a far better chance than one who has to return from a sanatorium to poverty, unsuitable work and bad home conditions. In judging the prognosis the greatest weight must be given to the sort of life the patient will have to lead after his treatment is finished.

Extent of Disease. The extent of the disease is also important, and it has been found that patients in Group II. of the Turban-Gerhardt classification (which is based on the anatomical extent of the disease) have a greater expectation of life than those in Group III., but less than those in Group I. Other things being equal, therefore, the greater the amount of disease the worse the prognosis. The type of disease is, however, of more importance, for if there is evidence of resistance, as indicated by fibrosis, the outlook is much better than in the case of a caseating lesion which spreads rapidly without any sign of arrest. Extensive disease does heal sometimes under simple sanatorium treatment and this is illustrated in Plates XVII. and XVIII. It is unwise to give a bad prognosis until one sees how a patient responds to treatment.

Cavities. It has been said that the treatment of pulmonary

tuberculosis is the treatment of cavities. Now this is a very dangerous doctrine because it implies that treatment is not required and prognosis is good in the absence of cavities. I contend that treatment should be instituted at the earliest moment to prevent the disease reaching the stage when cavities develop. Of course, it often happens that when the patient is first seen a cavity is present, but to refrain from treatment, such as artificial pneumothorax, until the disease has progressed to cavity formation is, in my opinion, absolutely wrong. When a cavity forms the prognosis becomes worse, but many patients with cavities make a complete recovery. Plates XIX. and XX. illustrate the disappearance of cavities without any artificial collapse or surgical intervention.

In the case shown in Plate XXI. there is an apical cavity held up by adhesions and the patient was strongly advised to have them cauterised. She refused and also refused phrenic evulsion, and yet the cavity did eventually heal as shown by Plate XXII., taken three years later.

A cavity usually contains tubercle bacilli in very large numbers and even a dry cavity is apt to become infected; the bacilli are scattered to other parts of the lung by cough and at autopsy it is often possible to trace a bronchogenic spread from a cavity. A cavity, therefore, is a potential danger and a very real one, especially if it is not dry and fails to close; but as regards prognosis one should bear in mind :—

1. That cavities often heal spontaneously.

2. That if they do not close they may remain dry and the patient may live for many years in comparatively good health.

3. That if there are cavities associated with purulent sputum containing tubercle bacilli the patient is not likely to live more than one or two years unless the cavities can be closed, and the activity of the disease arrested.

4. That in a chronic fibroid case prognosis should chiefly

be based on symptoms and the general condition of the patient. Signs of a cavity do not of necessity indicate a bad outlook, but if the patient with fibroid disease is toxic and has sputum containing tubercle bacilli, failure to find a cavity on clinical or X-ray examination does not render the prognosis less grave.

Type of Disease. Miliary tuberculosis was until quite recently regarded as part of a general tuberculosis and always fatal. Radiography may show miliary tuberculosis in the lungs in a patient with or without symptoms of toxæmia or pyrexia and yet recovery occurs. Maxwell (2) described a remarkable case of recovery in such a condition which is illustrated in Plates XXIII. and XXIV. Healing may occur leaving scattered calcareous deposits as shown in Plates XXV. and XXVI.

Tuberculous broncho-pneumonia (Plate XXVII.) is an extremely serious condition. It may be the terminal picture in a case of chronic tuberculosis or follow hæmoptysis.

Tuberculous lobar pneumonia is also serious, but it may be largely an allergic reaction around a small tuberculous focus. The inflammatory area may clear up leaving a tuberculous centre or core of varying size or activity, or possibly of no activity at all.

Response to Treatment. The response of the patient to treatment is a valuable guide to prognosis. In the great majority of cases a patient improves when he first comes under treatment, and this is probably due to the rest, which is the basis of almost every form of treatment in the initial stages, but after a few weeks some patients fail to improve or begin to lose ground, and for these the outlook is much less hopeful than for the patient who steadily improves, and is able to go from stage to stage of the treatment without a setback. Temporary relapses do occur often during convalescence, and the patient must not be disheartened by one. At the same time each setback makes the outlook less hopeful. For example, in the case

of a patient who has reached a quiescent stage, and is doing well, but has had three relapses, the prognosis is worse than in one who has reached the same stage of improvement without any relapse at all.

Age. The type of disease varies to a considerable extent according to age. In young children tuberculosis tends to be arrested or fixed in the glands; if it occurs in the lungs it is frequently part of a general infection and the outlook is serious. The prognosis in the case of glandular tuberculosis in children is good. Delicate children who are undergrown, and perhaps have a slight dry cough with some enlarged hilum shadows seen by X-ray, are often diagnosed as having tuberculous mediastinal glands, and no doubt some of them have, but they are no more likely to develop tuberculosis of the lungs than other children. It is, of course, important to improve their general state of health, and a prolonged stay at the seaside with healthy exercise and outdoor games is beneficial whether the debility is really due to tuberculous glands or not, but it is wrong to regard them as being especially liable to become consumptive. Young children with definite pulmonary tuberculosis may recover and the view that the disease is always fatal in the very young is no longer held.

About the age of puberty pulmonary tuberculosis usually runs a very acute course, and this is the age of greatest mortality. The prognosis, therefore, is very grave, although recovery may take place even in cases which appear to have very extensive and rapidly-spreading disease. The prognosis here depends to a great extent on the response of the patient to treatment. In adult life the disease is generally chronic, and arrest takes place under proper treatment. In old age pulmonary tuberculosis is not common, but when it does occur the prognosis is serious.

Family History. This is usually of no great importance in prognosis. The fact that an aunt or cousin, or even brother or sister, died of pulmonary tuberculosis does not

make the outlook more serious. Indeed, I have found that patients who develop an acute form of tuberculosis of the lungs some few months after nursing or being in close contact with an infected relative, often respond readily to treatment and make good recoveries. In some families, however, there seems to be a special susceptibility or lack of resistance, and one must take a grave view when a member develops the disease. In one family the patient's mother died of pulmonary tuberculosis when thirty-three, two of her sisters died of it between twenty and thirty. Her son died of acute tuberculosis when nineteen, and her daughter contracted it at the age of twenty-three. Although her general condition was good and the signs very few when she was first seen, one had to take the family history into account in giving a prognosis. The disease did run an acute course and she lived less than a year.

I once saw a woman of sixty-five with signs of early pulmonary tuberculosis, but the disease ran a rapid course and she died in six months. A few weeks before she became ill she had been nursing her sister, who died of the same complaint. Although there was no history of tuberculosis in her father's or mother's family, she had lost two brothers and four sisters from pulmonary tuberculosis. Their ages at death were thirty, forty-four, seventy-six, seventy-six, seventy-eight and eighty-one.

Drolet (8) records an investigation into the effect of heredity on tuberculosis. He came to the opinion that tuberculous parents confer some degree of immunity on their offspring which renders them actually less liable to contract the disease than are the children of healthy parents. His results seem to show that children do not inherit any susceptibility to tuberculosis. The matter is affected by many factors, such as contact with infection, home conditions, other illnesses, etc. Moreover, the difficulty of following up all members of the family throughout the whole of their lives and then getting reliable evidence of death is

so great that figures are apt to be misleading. Family histories where several members develop tuberculosis at about the same age are not uncommon, and frequently the same type of disease attacks each member, and it runs a similar course in each case.

It is difficult to explain such cases without supposing that there is some inherited quality which renders them susceptible. Of course, children brought up in a home with tuberculous parents are sure to be infected, and apart from any inherited predisposition, repeated mild infections might confer some immunity, but large ones would produce actual disease.

Ford (4) studied the histories of over 1,000 cases of pulmonary tuberculosis and found that 79·5 per cent. gave no family history of tuberculosis. His results also suggested that a family history of tuberculosis did not increase the gravity of the prognosis. On the contrary, he found that those cases whose mothers had been tuberculous did much better than the others.

Temperature. Before discussing temperature it may be as well to say a few words about the best method of taking it.

The chief difference of opinion concerns the oral and rectal methods. Probably the rectal method is slightly less liable to error than the oral, but it has certain disadvantages. Not only is it unpleasant for the patient or nurse, but it is undoubtedly apt to aggravate any nervous or hypochondriacal tendencies on the part of the patient. A patient who has been accustomed to having the temperature taken by the mouth finds himself in another institution where it is taken by the rectum. If neurotic he will immediately think that the slightest variation of temperature is of extreme importance. his spirits will rise and fall with his temperature, and long after he should have discarded his thermometer he will secretly use it, and a mysterious fit of depression may be accounted for by the fact that his rectal temperature was 99·4 after tea. I have frequently noticed,

especially in foreign institutions, how common a topic for conversation is the temperature chart, and what a bad effect it has on the patients as a whole. It is quite true that a patient may worry about a temperature taken by the mouth, but it has not the same significance to most of them. The rectal method suggests that the slightest rise of temperature is so vital that the most accurate method of obtaining it must be employed and the natural result is that a slight elevation will cause the patient serious anxiety. I do not wish to underrate the value of the temperature chart in

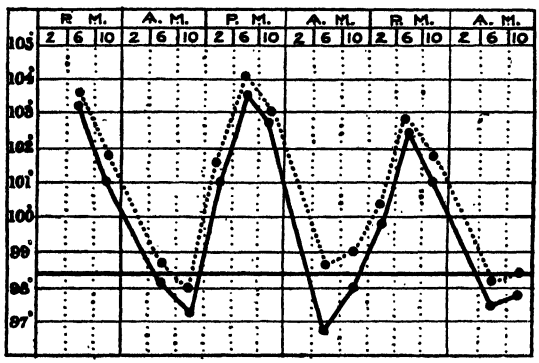

FIG. 2. Dotted line, rectal temperature ; solid line, mouth temperature.

pulmonary tuberculosis. On the contrary, I entirely agree with Kingston Fowler that "the temperature is the guide to the activity of the disease," but I maintain that consideration for the mentality and temperament of the patient is of far greater importance than any slight difference between rectal and oral temperatures. For special purposes, such as after tuberculin or exercise, in diagnosis a rectal temperature may be useful, but in my opinion it is wrong to use it as a routine. I have often had the temperature taken by the mouth, axilla and rectum simultaneously in order to demonstrate to my class how similar are the curves, and for the purpose of this book I had rectal

and oral temperatures taken in three patients, and the results are shown in Figs. 2, 3 and 4. The higher curve is the rectal temperature, but it will be found to rise and fall exactly as the oral one, and these charts support my contention that the objections to the rectal method outweigh its advantages.

With regard to the axillary temperature, this also corresponds with the oral and rectal methods, but the axilla must

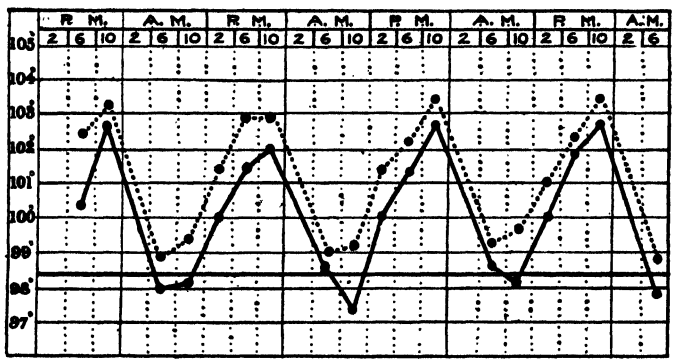

FIG. 3. Dotted line, rectal temperature ; solid line, mouth temperature.

be carefully dried first. It is unreliable to take the temperature in a stream of urine.

Milne (5) wrote: "By taking the oral and rectal temperatures simultaneously, twice daily for three weeks in one series, I found the tracings on the charts to be almost parallel, the rectal being the higher by about half a degree." He thinks the oral method of taking the temperature is the most suitable.

The temperature in pulmonary tuberculosis may be taken as an exaggeration of the normal curve, being lowest about 4 a.m. and highest between 6 to 9 p.m. In the morning from about four to seven it remains low, and then gradually rises, reaching normal about ten. It remains there until two in the afternoon, when it again rises until 6 p.m., and

then reaches and remains at its maximum until 9 or 10 p.m., at which time the drop begins. Fig. 5 illustrates the usual variation in the temperature, and it will be seen that by taking an early morning and evening temperature the minimum and maximum can be obtained.

When there is a big difference between the maximum and

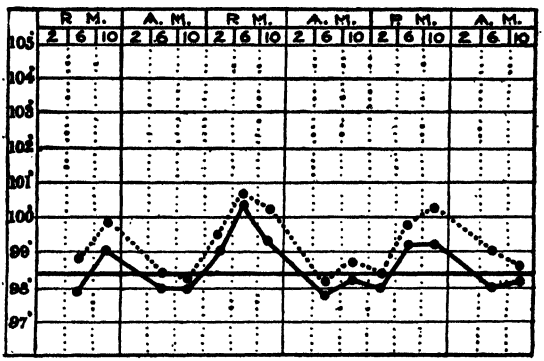

FIG. 4. Dotted line, rectal temperature ; solid line, mouth temperature.

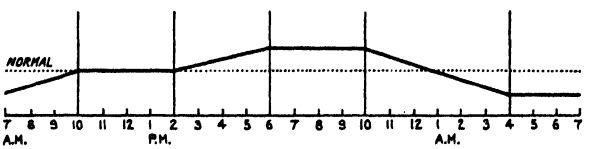

FIG. 5. Diagram showing the daily variation of temperature in the normal subject.

minimum the temperature is called hectic, and some have thought this is due to a superadded infection by pyogenic organisms. Koch thought a temperature of over 100·4 was due to the presence of septic organisms, and consequently should be taken as a contra-indication for tuberculin. It has been stated that various forms of treatment, climatic and other, should depend on whether the case is a pure tuberculosis, or complicated by mixed infection, and it has been claimed that the advent of mixed infection can be

diagnosed by the temperature chart. Since it affects treatment and prognosis, as well as our views of the course of the disease, it is of great importance to consider how far, if at all, the temperature in tuberculosis is affected by secondary organisms. Of course, in long-standing cases of pulmonary tuberculosis, other organisms will collect, and tuberculous patients are just as liable as any to fever from other causes; but there is no doubt that typical hectic fever is seen in cases of tuberculous peritonitis, pleurisy, etc., where post-mortem examination shows simply tuberculous disease. Kingston Fowler (1) said: "The parrot cry of mixed infection does not come from the pathologists." And again: "the term 'mixed infection' has no place in relation to pulmonary tuberculosis, but of course other organisms are present in the lungs when breaking down of the lesions and the formation of cavities is in progress."

Thus we may consider the hectic type of temperature as an extreme exaggeration of the normal, and as indicating active breaking down of the lung tissue. The temperature is the guide to the activity of the disease, and all degrees may be seen from the hectic in rapidly-spreading tuberculosis to normal in the chronic fibrotic disease when there is little or no spread. It must not be forgotten, however, that there may be gradual spread of disease in a fibroid case without pyrexia, and in advanced cases where resistance fails the terminal stages are non-febrile.

Apart from this usual type of fever, varying according to the acuteness of the infection, there are other types met with in tuberculosis. In the inverse type (Fig. 6) the morning temperature is high and the evening low, and it is of serious import. Kingston Fowler (1) found that of 30 cases of tuberculosis of the lungs, mostly of the chronic variety, under his care in the Brompton Hospital, in which the inverse type of pyrexia was observed, 14 died in hospital, and 1 immediately after returning home; 8 left the hospital worse; 5 were relieved or about the same; 2 improved,

but in these the inverse type of temperature was of only temporary duration, being present twice in one and eight times in the other.

The inverse type is usually seen in cases of remittent fever. The morning rise persists, but in the evening the patient

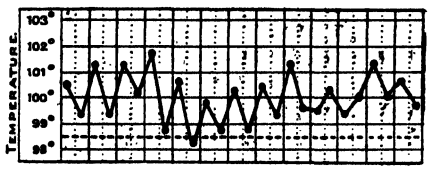

FIG. 6. Showing inverse type of temperature.

fails to react, and consequently there is no rise of temperature. A high temperature in tuberculosis can usually be reduced by giving cryogenin grs. x. twice a day, but it rises again as soon as the drug is stopped. In some cases of high remittent fever the drug reduces the evening rise of temperature, but has little or no effect on the morning one,

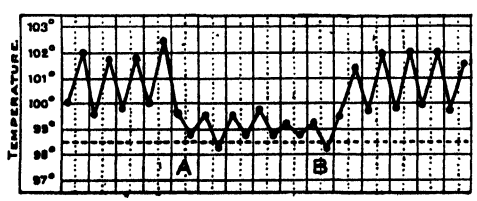

FIG. 7. Remittent type of temperature. At A the patient was given cryogenin gr. v. twice daily and at B this was stopped.

so that an inverse type of temperature results, which takes on the usual form again as soon as the cryogenin is discontinued. This is shown in Fig. 7.

The continuous type of temperature is often due to the occurrence of miliary tuberculosis, and is therefore a serious sign. If in a case of chronic pulmonary tuberculosis, or pleural effusion, the swinging temperature rises and the

morning intermissions disappear, as shown in Fig. 8, miliary tuberculosis and a fatal termination must be expected.

The most serious sign is when, in spite of much active disease, the temperature falls to normal but at the same time

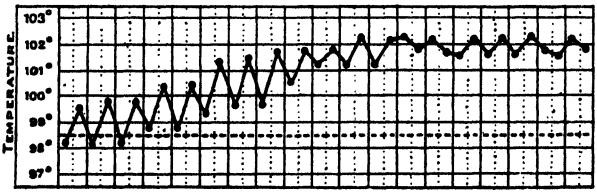

FIG. 8. Intermittent type of temperature becoming continuous and high owing to the onset of miliary tuberculosis.

the patient shows signs of collapse. This indicates failure to react and complete loss of resistance, and, as seen in Fig. 9, is usually followed by a fatal issue in a few days, but a similar fall of temperature associated with improved condition of

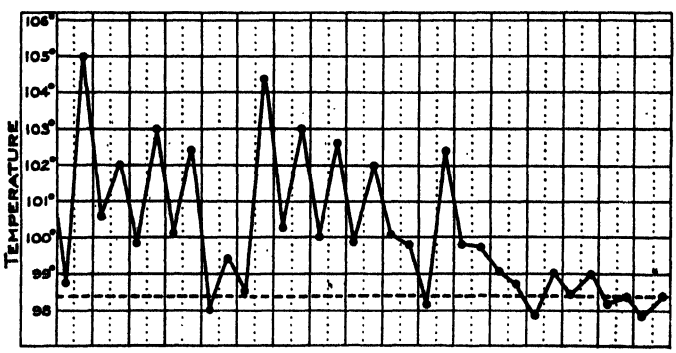

FIG. 9. Fall of temperature owing to exhaustion of the patient.

the patient would be a very good sign. Fig. 10 shows an intermittent temperature, becoming remittent as the disease progressed. At the onset the temperature was quite consistent with a favourable course, but as, in spite of rest and other treatment, it not only failed to subside but grew worse,

the prognosis became increasingly grave, and the patient died later of tuberculous broncho-pneumonia.

A persistent but slight rise of evening temperature from 99° to 99·4° F. is usually not due to pulmonary tuberculosis, but to some complication, such as tuberculous peritonitis or pleurisy. It may persist for many months, as in the following case: the patient, a man of twenty-three, developed pulmonary tuberculosis, and after a few months in a sanatorium he improved, the signs in the chest showed that satisfactory fibrosis was taking place, tubercle bacilli disappeared from the sputum, and eventually the sputum dried up altogether.

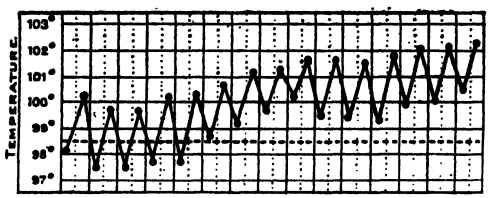

FIG. 10. Intermittent type of temperature becoming remittent owing to increased activity of disease.

In spite of these favourable signs he continued to feel slack, and he always had an evening temperature of between 99° and 100° F., whether he was resting or up and about. He left the sanatorium and was living a quiet but fairly normal life at home, when suddenly, about eighteen months after the onset of his symptoms, he had an acute attack of vomiting and abdominal pain, which was thought to be due to food poisoning as it occurred immediately after a picnic. Five months later he had another attack, and laparotomy was performed. Extensive tuberculous peritonitis was found, and there were many adhesions, some of which were divided as they were obstructing the intestine. A period of rest following the operation restored him to health, and he no longer has the evening rise of temperature.

Lastly, there are two types of periodic fever in tuberculosis. One is seen in women and occurs as a slight pre-

menstrual rise, beginning a week or ten days before each period and rapidly falling after the first or second day of menstruation. Such a slight variation in temperature is not peculiar to tuberculous patients, and is of no significance in prognosis, but occasionally there is a definite rise of temperature to 101° F. or 102° F. for a day or two before each period. This is very similar to a tuberculin reaction, and passes off when the period begins. These menstrual reactions are frequent, but not of serious import.

A second type of periodic fever may occur every few weeks or months, and is usually associated with pocketing of sputum in a cavity or dilated bronchi. The patient coughs up a large quantity of sputum for a day or two, and then the temperature falls and the health improves for a time, when the same occurrence is repeated. Prognosis in these cases depends on the severity of the symptoms and the response of the patient to pneumothorax, postural drainage, or other treatment.

The following conclusions may be drawn concerning the value of the temperature in prognosis :—

1. Pyrexia must be considered in conjunction with other factors in regard to prognosis. For example, although a normal temperature is usually of good import, progressive fibrotic disease may occur without causing a rise of temperature and absence of temperature may be due to failure of resistance in the terminal stages of the disease.

2. The temperature is, however, the best guide to the activity of the disease. Other things being equal, the best prognosis should be given in cases who are not febrile in spite of exercise, and the worst in those who remain febrile in spite of rest in bed. An intermediate position is occupied by those who are not febrile whilst at rest, but febrile after exertion. The less exercise it takes to send up the temperature the more serious becomes the outlook.

3. The inverse type of temperature is of serious import.

4. When the type of temperature changes to a high con-

tinuous pyrexia the prognosis is very bad, for it indicates the occurrence of miliary or generalised tuberculosis.

5. A prolonged slight elevation of the temperature is usually due to some cause, such as tuberculous pleurisy or peritonitis.

6. Prognosis in cases of periodic rise of temperature depends on the cause of the rise and the response of the condition to treatment. Slight premenstrual rise of temperature is of no significance.

Sedimentation Test. Although this test is of little value in diagnosis, it is of some value in prognosis and in determining the patient's response to treatment. It consists in observing the rate at which the erythrocytes fall, leaving clear serum above in the column of blood, and depends chiefly on the fibrinogen in the plasma, on the globulin to a smaller degree and on the red-cell volume to some extent.

There are several methods of performing the test and, since its main object is to follow the progress of the disease by noting whether there is an increase or decrease in the rate of fall of the corpuscles, any method will serve the purpose provided the same one is used throughout the series. It would however be convenient if one standard method were adopted instead of the present practice of using different methods at different institutions which make comparison impossible. I prefer the Westergren method.

The technique is simple, but it is important to observe every detail. Into a 2 c.cm. syringe 0·4 c.cm. of a 3·8 per cent. solution of sodium citrate in distilled water is drawn up. The patient's vein is then punctured and 1·6 c.cm. of blood withdrawn to fill the syringe. The citrated blood is thoroughly mixed and then put into a Westergren tube up to the 200 division, care being taken to avoid any air bubbles in the column of blood. In time the column of red blood will sink, leaving clear serum above it and the rate of this fall is noted by taking readings every hour for six or more hours.

Now there are many causes of error. In the first place

the length of the column of blood should always be the same, because calculations based on the fall of the red cells as a percentage of the height of the column are not accurate during the first few hours. The following examples show the drop of the red cells each hour in columns of 200 mm., 150 mm., 100 mm. and 50 mm.

In case 1 it will be noticed that when the height of the blood column was 200 mm. the drop in the first hour was 56, or 28 per cent., in the 150 mm. column 49, or 32·6 per cent., in the 100 mm. column 35, or 35 per cent. and in the 50 mm. column 27, or 54 per cent. At the end of the eighth hour the corresponding readings are 62·5, 66, 63 and 68 per cent. (Table 8).

Female, aged 24. Red blood cells 4,800,000. Hæmoglobin 77 per cent. White blood cells 10,700.

Diagnosis. Pulmonary tuberculosis. (Two years standing. Progressive. Bilateral. Pyrexial.)

TABLE 8.

Height of column in mm.	200		150		100		50	
		%		%		%		%
Fall after 1 hour .	56	28	49	32·6	35	35	27	54
,, 2 hours .	92	46	76	50·6	54	54	32	64
,, 3 ,, .	108	54	88	58·6	33	58	58	66
,, 4 ,, .	118	59	95	63·3	60	60	34	68
,, 5 ,, .	122	61	96	64	62	62	34	68
,, 6 ,, .	123	61·5	97	65·6	62	62	34	68
,, 7 ,, .	124	62	98	65·3	62	62	34	68
,, 8 ,, .	125	62·5	99	66	63	63	34	68
,, 9 ,, .	126	63	100	66·6	63	63	35	70
,, 10 ,, .	126	63	100	66·5	63	63	35	70
,, 11 ,, .	126	63	100	66·5	63	63	35	70
,, 12 ,, .	127	63·5	100	66·5	65	65	36	72
,, 18 ,, .	128	64	101	67·3	65	65	36	72
,, 24 ,, .	129	64·5	101	67·3	65	65	36	72

Case 2 also shows how the red cells fall quickly in the first few hours and then reach a level, after which there is but little more drop. Reading in percentages the figures for the four lengths of blood column, 200, 150, 100 and 50 mm. are in this case for the

1st hour	18	20·6	28	40
10th ,,	60·5	61·3	64	64

It appears that readings taken after eight or more hours are roughly comparable if expressed in percentages of the blood column, but cannot be compared in this way if taken in the first hour or two (Table 9).

Male, aged 21. Red cell count 4,600,000. Hæmoglobin 70 per cent. White cell count 11,400.

Diagnosis. Pyopneumothorax, swinging pyrexia.

TABLE 9.

Height of column in mm.	200		150		100		50	
		%		%		%		%
Fall after 1 hour .	36	18	31	20·6	28	28	20	40
,, 2 hours .	62	31	53	35·3	42	42	28	56
,, 3 ,, .	81	40·5	69	46	54	54	30	60
,, 4 ,, .	96	48	81	54	59	59	30	60
,, 5 ,, .	107	53·5	86	57·3	61	61	31	62
,, 6 ,, .	114	57	88	58·6	62	62	31	62
,, 7 ,, .	118	59	90	60	63	63	32	64
,, 8 ,, .	119	59·5	91	60·6	63	63	32	64
,, 9 ,, .	120	60	91	60·6	63	63	32	64
,, 10 ,, .	121	60·5	92	61·3	64	64	32	64
,, 11 ,, .	122	61	92	61·3	64	64	33	66
,, 12 ,, .	122	61	93	62·6	66	66	34	68
,, 24 ,, .	124	62	95	63·3	66	66	34	68

Case 8. Female, aged 35 (Table 10).

Diagnosis. Lung abscess. (Acute one year ago, now appears healed.)

TABLE 10.

Height of column in mm.	200		150		100		50	
		%		%		%		%
Fall in 1 hour . .	24	12	20	13·3	22	22	16	32
,, 2 hours . .	57	28·5	48	32	40	40	24	48
,, 3 ,, . .	78	39	62	41·3	46	46	27	54
,, 4 ,, . .	92	46	70	46·6	52	52	28	56
,, 5 ,, . .	98	49	74	49·3	54	54	29	58
,, 6 ,, . .	100	50	77	51·3	58	58	30	60
,, 7 ,, . .	107	53·5	80	53·3	58	58	30	60
,, 8 ,, . .	108	54	82	54·6	60	60	31	62

Case 4 shows the readings in the first two hours and demonstrates the danger of taking the first hour's reading only (Table 11).

Male, aged 17.

Diagnosis. Recent pleural effusion (afebrile).

TABLE 11.

Height of column in mm.	200		150		100		50	
		%		%		%		%
Fall in 1 hour . .	7	3·5	6	4	5	5	3	6
,, 2 hours . .	20	10	19	12·6	14	14	13	26

Case 5 is a normal control (Table 12). Male aged 28.

TABLE 12.

Height of column in mm.	200		150		100		50	
		%		%		%		%
Difference 1 hour .	0	—	1	0·6	0	0	0	—
,, 2 hours .	2	1	2	1·3	1	1	1	2
,, 3 ,, .	3	1·5	3	2	2	2	1	2
,, 4 ,, .	3	1·5	4	2·6	3	3	2	4
,, 5 ,, .	3	1·5	4	2·6	3	3	2	4
,, 6 ,, .	4	2	4	2·6	3	3	3	6
,, 7 ,, .	4	2	4	2·6	3	3	3	6
,, 8 ,, .	5	2·5	4	2·6	4	4	3	6
,, 9 ,, .	5	2·5	5	3·3	5	5	4	8
,, 10 ,, .	5	2·5	5	3·3	5	5	4	8
,, 11 ,, .	6	3	5	3·3	5	5	5	10
,, 12 ,, .	6	3	6	4	5	5	5	10
,, 18 ,, .	11	5·5	8	5·3	7	7	7	14
,, 24 ,, .	14	7	12	6	9	9	11	22

Miller (6) states: (1) That the fall is not equal in tubes of different lengths in the first few hours. (2) That the fall is only proportional to the length of the tube when a proportionately long time has been allowed. (3) That there is an optimum time at which each tube should be read, and after this the increase in the fall occurs not because of the factors affecting the sedimentation rate, but owing to packing of the red cells from their own weight.

Reference to the examples given will show that in a certain number of hours the red cells reach a position and do not fall much afterwards. The time taken to reach this position is called by Miller the optimum time for reading, and if this time is taken it is possible to express the sedimentation rate as a percentage of the length of tube and time taken.

Thus, if there is a fall of 10 mm. in two hours in a column of 50 mm. it would be one of 20 mm. in a tube of 100 mm. in four hours, of 30 mm. in a tube of 150 mm. in six hours, of 40 mm. in a tube of 200 mm. in eight hours.

It is not uncommon for a patient to be sent from an institution with full notes as to his condition on discharge, and a statement that the sedimentation rate was 3, 10, 20 or some other figure, but no information is given as to the height of the column of blood nor as to the time at which the reading was taken. Reference to case 4 shows the futility of such a statement.

If the tube is not absolutely vertical the reading is misleading, for a slant in the tube of 5 degrees may make a difference of over 20 per cent. Again, the sedimentation rate is affected by the red cell count and so an increase or decrease in the rate of fall may be due to variation in the number of red corpuscles. After hæmorrhage there is an increased rate and fall in direct proportion to decrease in red corpuscles. Temperature has an effect on the sedimentation rate, so that the turning on or off of a radiator in the room may make a great difference. A fall of 40 mm. in a tube kept at 37° C. was found to be only 23 in a column of the same height and of the same sample of blood kept at 12° C.

Lockett (17) has described the variation of the erythrocyte fall under various conditions, and certain sources of error. For example, digestion affects the test considerably. She found that blood taken from one patient half an hour before a meal showed a drop of 24 mm. in the first hour, but blood from the same patient half an hour after the meal showed a drop of 86 mm.

Exercise must be taken into account. A lady medical student showed a fall of red cells of 35 mm. in two hours in a column of 200 mm., but of 52 an hour later after half an hour's tennis. The drop of 35 mm. in two hours is high, but not uncommon in women who are apparently healthy and who remain in good health.

In another young woman in apparent health the sedimentation rate was taken every day for a month to show the effect of the menstrual cycle. At the onset of menstruation the fall after two hours was 60 mm., at the finish 55, and at mid-period 50, in a column of blood of 200 mm.

Roche (7) states that altitude renders the fall slower. He regards the test as a valuable guide to treatment and prognosis, and considers a fall of 4 to 15 mm. from a 200 mm. column in one hour in men, or 6 to 15 in women, as a slight increase, a drop of 15 to 30 as medium and 30 to 50 a great increase of the normal.

Heaf (8) noted that the rate of sedimentation was increased in cases in which the disease was obviously progressing and decreased in cases in which the lesion was healing. Of 150 patients whom he watched for from six to ten months it was found that prognosis based on a series of sedimentation tests was correct in 82 per cent.

Cummins and Acland (9) have published some investigations into the value of the sedimentation rate and Arneth count. They state that " while neither the sedimentation test nor the nuclear index provides new information unobtainable by ordinary clinical methods they afford two additional methods of measuring the constitutional balance of a case before, during and after treatment."

The sedimentation test is a useful guide in treatment because if conducted carefully it gives a rough indication of the patient's progress forwards or backwards, but from what has been said it will be clear that it is not a delicate test and may easily be misleading and consequently harmful.

Lee (18) has described a sedimentometer to record the

rate of fall of the corpuscles and it can be used for any of the recognised methods of performing the test.

Vernes' Flocculation Test. This was first used in cases of syphilis and later applied to tuberculosis. It depends on the degree of turbidity of the serum, which is measured by a complicated apparatus known as the Vernes-Brieg-Yvon photometer (10). The blood should not be taken until fifteen hours after a meal, for otherwise the serum may be cloudy. A few cubic centimetres of blood are taken from a vein and allowed to clot. The serum is decanted, centrifuged, and again decanted. This clear serum may be kept on ice for a time, but should be used within twenty-four hours. 0·6 c.cm. of this serum is added to 0·6 c.cm. of 1·25 per cent. solution of resorcin in doubly distilled water, and is immediately examined in the photometer to determine the optic density. Four hours later the optic density is again taken. If the difference between the two readings is under 15, active tuberculosis can be excluded, whereas a reading of over 30 is said to indicate an active lesion. Readings between 15 and 30 may be regarded as suspicious. Vernes claims that the test will enable one to eliminate the larval forms of tuberculosis, and determine the nature of a pleurisy or pulmonary focus of doubtful origin. It can, moreover, be used to follow and control the results of treatment.

Jordan (11) describes the results of the test, and Ralph and Davies (12) applied the test to 200 patients with proved tuberculosis, 150 suffering from some disease other than tuberculosis, and to 25 normal individuals.

The average optic density for the cases of acute tuberculosis was 95, but in convalescent cases it was between 40 and 50. The average figure for inactive tuberculosis was 28, but in 6 it was below 15, although tubercle bacilli were present in the sputum.

In typhoid fever the average during the second week was 55. Advanced malignant disease was usually above 100, and one case of carbuncle was 269.

All of the twenty-five normal cases gave a reading below 15, but one of them subsequently developed a common cold, and the test was given again and found to be 77. A month later it was taken again, and had fallen to 2. It is clear, therefore, that the test, like the sedimentation test, is affected by other conditions as well as tuberculosis. Ralph and Davies came to the conclusion that the Vernes test cannot be considered specific for tuberculosis, and that its chief value in diagnosis is that a figure below 30 is evidence against active tuberculosis although it is found in some 6 or 7 per cent. of such cases; the converse does not obtain.

James (13) applied the Vernes flocculation and the sedimentation tests to a series of 407 patients having or suspected of having pulmonary tuberculosis. He concluded that both tests are valuable for controlling treatment but are affected by other conditions besides tuberculosis and so are useless for diagnosis except that normal findings render active tuberculosis unlikely. He found the results of both tests comparable except that in very advanced cases the sedimentation test was more accurate. The Vernes test lags behind the sedimentation test in returning to normal as the patient improves. It is, moreover, more difficult and elaborate than the sedimentation test, which can be done at the bedside.

Vital Capacity. The vital capacity is definitely lowered in pulmonary tuberculosis, so that a normal finding may be taken as useful evidence against this disease, although a lower one is no evidence in its favour. The vital capacity increases if the patient improves and decreases if he becomes worse, and is therefore a good method of observing the effects of treatment. It is also of value in prognosis if repeated readings are taken. It is very necessary to take great care in obtaining the vital capacity or the readings will be inaccurate. I use the spirometer of C. Verdin, made by Boulitte in Paris.

The patient should be seated comfortably in such a position that he cannot see the dial of the spirometer. He should inspire to his fullest capacity and then expire com-

pletely but slowly into the mouthpiece of the spirometer. At first many patients fail to take a full inspiration, or to empty their lungs completely, but with a little practice they understand what they are required to do. The first two or three readings should be ignored and the patient encouraged to try again, until it is found that each time he gives the same vital capacity. It has been stated that patients can increase their vital capacity with practice, but this is not really the case, and it will be found that the capacity for a given patient is remarkably constant when once he has been taught what to do.

Dreyer showed that definite relationships exist between vital capacity and certain body measurements, and he published (14) a book containing tables showing what the normal capacity should be under various conditions. With Dreyer (15) I studied vital capacity in relation to pulmonary tuberculosis and found that of 267 cases with tubercle bacilli in the sputum the vital capacity was above normal in one only. This patient had tubercle bacilli discovered in the sputum once when he had a cough following a cold ; they were not found at any subsequent examination. X-ray and physical examinations failed to reveal any sign of disease, and he returned to work and kept well without developing any signs of clinical tuberculosis.

In our series it was found that vital capacity does diminish as the extent of the disease increases, but still more does it diminish with increasing toxæmia.

The following case shows the gradual decline in vital capacity as the patient grows worse :—

1920.	March.	Vital capacity .	— 33·1 per cent.
	May.	,, .	— 36·0 ,,
	September.	,, .	— 37·2 ,,
	December.	,, .	— 41·4 ,,
1921.	February.	,, .	— 52·0 ,,
	May.	,, .	— 52·0 ,,

The patient died in June, 1921.

The following two readings were taken from an acute case. The patient became ill in February and died in July. In March the vital capacity was — 16·9 per cent., in May it was — 62 per cent.

The following readings were taken from a chronic fibroid case, and it will be noticed how constant they remain whilst the condition of the patient kept practically the same :—

1919.	April.	Vital capacity .	— 24·5 per cent.
1921.	May.	,, .	— 21·0 ,,
1922.	March.	,, .	— 20·2 ,,

Even in advanced cases of fibrosis the vital capacity remains constant provided the condition of the patient remains unchanged, as the following readings indicate :—

1919.	April.	Vital capacity .	— 50·0 per cent.
1920.	February.	,, .	— 50·6 ,,
1921.	April.	,, .	— 50·7 ,,
1922.	March.	,, .	— 51·9 ,,

A good prognosis is suggested when the vital capacity is nearly normal, improves under treatment, or remains constant whilst the patient is leading an ordinary life.

A bad prognosis is probable if the vital capacity falls as soon as the patient begins to take exercise, and the outlook is still more serious if the capacity gradually becomes worse during treatment. A low vital capacity is produced by many other conditions besides tuberculosis, and provided it remains constant it does not necessarily indicate a bad prognosis.

Jacobæus (19) has described a method of separating the air from one lung from that of the other. He uses two bronchoscopes, one of which is introduced into the main left bronchus and the second one ends in the trachea. Both ends of the bronchoscope are surrounded by dilatable rubber cuffs, so that the air in either lung can be cut off and recorded

on separate spirometers. Analysis of the air from either lung can also be made. The information obtained may be of some help for prognosis but is chiefly useful to enable one to judge whether the better lung is sufficiently healthy to justify thoracoplasty. In testing 150 healthy students by this method he found that the function of the right lung is slightly greater than that of the left in about the proportion of 55 to 45. He also found that with the patient lying on his side the uppermost lung does less work than the other. When a patient has a lung collapsed by pneumothorax or thoracoplasty the healthy lung only is used for coughing, so that secretions tend to be sealed up in the collapsed lung. This may prevent the spread of infection to the better lung, but it means that the very lung most in need of getting rid of its secretion has the greater difficulty.

The following is an example of the information which may be obtained by broncho-spirometry :—

	R. Lung.	L. Lung.	Both.	R. % of total.	L. % of total.
Oxygen elimination .	266·6	4·1	270·7	98·5	1·5
CO_2 elimination .	180·8	10·2	191	94·7	5·3
Ventilation . .	5565	200	5765	96·5	3·4
Vital Capacity . .	1145	40	1185	96·6	3·4

The procedure is complicated and as yet in its infancy, but may well become a valuable method of testing the efficiency of either lung.

Complications. The development of certain complications, such as meningitis or enteritis, make the prognosis hopeless. Toxic diarrhœa must be distinguished from ulceration of the intestine. Tuberculous peritonitis occurring during tuberculosis of the lungs usually proves fatal if accompanied by continuous pyrexia. Patients often live for many months, but it is a far more serious condition than tuberculous peritonitis occurring in a patient whose lungs are healthy.

Tuberculous laryngitis was formerly thought to be a fatal complication, but, although it undoubtedly adds to the gravity of the disease under modern treatment many patients recover. St. Clair Thomson (16) stated that 25 per cent. made a complete recovery. The lupoid type of laryngeal tuberculosis has the best prognosis. He also said that although the larynx might heal in spite of spread of disease in the lungs the reverse is not the case, so that a spreading laryngeal lesion indicates a bad prognosis.

Hæmoptysis rarely proves fatal, and it has been said that patients do not die of hæmoptysis, but after it. The after-effects, and especially tuberculous broncho-pneumonia, are certainly more dangerous than the hæmorrhage itself. Some patients are liable to repeated attacks of hæmoptysis, and many of them live for years, and, except that they have to rest for a few days during each attack, they maintain a good standard of health. The prognosis is always uncertain in such cases, owing to the possibility of an acute exacerbation following hæmoptysis. A sudden large hæmoptysis is not common, and more often occurs in the chronic fibrotic case than in other types of the disease. It may be due to the rupture of a vessel crossing a cavity and so unsupported by lung tissue. In this case it often proves fatal in a minute or even less.

Hæmoptysis may actually improve the prognosis by drawing attention to disease which might not otherwise have been detected, and more especially by alarming the patient, and thus making him willing to undergo thorough treatment at the beginning of his illness instead of waiting until the disease has passed into the incurable stage.

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CHAPTER VI

CERTAIN COMPLICATIONS

Hæmoptysis

MUCH has been written about the treatment of hæmoptysis and the number of remedies advocated bears testimony to their failure.

The first thing to remember when confronted with a case is that it is a most alarming occurrence for the patient and his friends, and it is necessary to reassure them and give them confidence.

The second is that, although something definite must be done to calm the patient, some methods of treatment are actually harmful, and even if one cannot stop the hæmorrhage one can at least avoid making it worse.

The bleeding may vary from a mere staining of the sputum to a large and fatal hæmoptysis, although such a serious one is rare. Patients do not die of hæmoptysis, they die after it as a result of its complications. A large hæmoptysis usually comes from a ruptured vessel or an aneurysm of the pulmonary artery in a cavity, a small one from capillary congestion. The blood may come from the systemic (bronchial vessels) and not from the pulmonary circulation, and in any case pulmonary pressure is not of the first importance, since whether raised or lowered the same quantity of blood has to pass through the pulmonary system, as all blood reaching the left auricle has to go through the lungs and right side of the heart.

Drugs given for the purpose of affecting the pressure are of doubtful value therefore, and they would probably be actually harmful but for the fact that if they do have any

effect on the blood pressure at all it is very transient. Drugs such as amyl nitrite, adrenalin, pituitrin and ergot are best avoided. Adrenalin is said to constrict the peripheral arteries except the pulmonary, which it does not affect, and the cerebral and coronary arteries, which it causes to dilate; pituitrin is said to lower the pulmonary but raise the general blood pressure. In practice, however, no effect is produced on hæmoptysis. Turpentine is harmless in small doses but poisonous in large ones, and even large doses taken by the mouth have no effect whatever on hæmoptysis. Cold drinks or sucking ice have been advocated, but they dilate the pulmonary vessels and tend to increase the hæmorrhage. Hot drinks constrict the vessels, but have a stimulant effect and should be avoided. An ice-bag applied to the chest is said to constrict the vessels beneath it. I have never seen it have any effect on the hæmorrhage, but it may soothe the patient and so have a good mental effect.

Sodium chloride given by mouth (℥i. in water ℥v.) or intravenously (5 c.cm. of a 10 per cent. solution) has also been advocated and is said to increase the coagulability of the blood by drawing into the blood stream tissue fluid and so thrombokinase. If given by the mouth it may cause vomiting, which I think should be avoided, although some advocate emetics partly because they tend to clear the bronchial tubes and partly because the effort of vomiting may rupture the bleeding vessel and so, by allowing the ends to contract, stop the hæmorrhage.

Horse serum is quite useless in my experience, and I have not seen any good results from hæmostatic serum or coagulenciba, given subcutaneously. In one case I injected coagulenciba through the crico-thyroid membrane into the trachea, but it had no effect.

Calcium chloride increases the coagulation rate of the blood considerably. Elving (1) found the rate altered from five and half minutes to half minute in one case after the intravenous injection of 20 c.cm. of a 15 per cent. solution of

calcium chloride. Many cases have been described where hæmoptysis has stopped rapidly after such an injection, but it tends to stop automatically, and I have never convinced myself that this result was due to the calcium. Osler pointed out that treatment which might do good in a case of congested mucosa would be as much out of place in hæmorrhage from a ruptured pulmonary aneurysm as in a cut radial artery. However, any effect the calcium did have would be for the good and it is quite rational to use it. In certain cases of continuous bloodstained sputum calcium may stop the bleeding which is most alarming for the patient. Ten c.cm. of Sandoz calcium intramuscularly or intravenously, or 5 c.cm. of a 10 per cent. solution of calcium chloride intravenously every eight hours are the best ways of using calcium in these cases.

Morphia is advocated by one school and denounced by another in the treatment of hæmoptysis. Riviere (2) discussed the advantages and disadvantages of opiates and gave a very full account of the various methods of treating hæmoptysis. He said that the modern tendency is :—

1. To delay the use of narcotics in large hæmorrhages so that cough may be efficient in clearing the tubes.
2. To avoid their routine use.
3. To replace morphine by codein, heroin or dionin.

At the conclusion of his paper he stated that, after all, the outcome of hæmoptysis in any individual case is "on the knees of the Gods," and commonsense methods to assist Nature are all that can really be done apart from collapsing the lung. To this I would repeat my warning of the possibility of doing harm by over-treatment. Riviere said: "Nature's method will here be plugging of the area around with blood clot—until, soon, her concern for clear air tubes leads her to defeat these efforts by the induction of cough. How clear a passage can be steered under such circumstances between the Scylla of infarcted lung with its threat of pneumonia or subsequent tuberculosis, and the Charybdis of

continuing hæmorrhage is for the physician to ponder, for he holds the fatal rudder, opium and its derivatives, in his hands."

One view is that hæmoptysis is not often fatal, and in the rare cases when it did prove fatal morphia would not have saved the patient. On the other hand, tuberculous broncho-pneumonia or bronchitic spread of the infection does develop not infrequently after a hæmoptysis, and this is due to blood getting into the bronchi and remaining there. Treatment should be to clear the bronchi, whereas morphia tends to prevent cough. All this is perfectly true, but a small amount of morphia does not prevent cough and certainly does not lead to accumulation of blood in the tubes any more than natural sleep does, and it undoubtedly pacifies the patient. Most physicians will agree that it is bad practice to keep a patient deeply under the influence of morphia or to give an injection of morphia every time a fresh hæmoptysis occurs, but an initial injection at the onset does no harm whatever. On the contrary, it does a great deal of good by calming the patient and preventing restlessness. It must be admitted that blood should not be allowed to remain in the bronchial tubes, but too much coughing undoubtedly breaks clots which have formed over the bleeding surface and the violent expiratory and inspiratory efforts of cough prevent clotting and increase the hæmorrhage. In order to clear the tubes of blood some advocate the ambulatory treatment of hæmoptysis. The patient is allowed out of bed, given stimulants and even emetics. Whilst admitting the danger of broncho-pneumonia or spread of disease after hæmoptysis, I think it is far more dangerous to allow a patient to walk about and to encourage too much coughing.

Morlock and Scott Pinchin (8) stated that broncho-pneumonia following hæmoptysis may be caused by lobular collapse as a result of aspirated blood. They have found the intravenous injection of 10 c.cm. of a 1 per cent. solution

of Congo Red checks hæmoptysis. It may lead to a slight rigor and larger doses to severe shock, but smaller doses are not efficacious. It is said to reduce the clotting time and increase the blood platelets, monocytes and fibrin content of the blood. Courcoux (4) treated twenty cases of hæmoptysis by the subcutaneous injection of oxygen. In twelve the hæmorrhage was checked immediately and in four the injections had to be repeated on three or four consecutive days. He advocates injecting it under the skin of the thorax on the affected side and regards 500 c.cm. as a minimum dose, but considers it unnecessary to give as much as a litre. Good results have, however, been reported from small doses, such as 200 c.cm. given under the skin of the thigh.

In order to decrease the quantity of blood passing through the lungs the thighs have been bandaged sufficiently tightly to stop the venous circulation. After an hour and a half one arm is bandaged and one thigh unbandaged, and then the other arm is bandaged and the second thigh unbandaged. Half an hour later first one arm and then the other is unbandaged. This method is also said to increase the thrombokinase in the blood, since the bandaging produces œdema, and on unbinding the limbs the tissue fluid flows into the blood stream. A similar result is said to follow dry cupping the back or abdomen.

In a severe case of hæmoptysis artificial pneumothorax should be induced if one can determine from which lung the blood is coming : 300 or 400 c.cm. of air introduced into the pleural cavity are usually sufficient to stop the bleeding. Formerly I used to give a large initial quantity of air, but now I do not think it necessary. Should the first injection fail to check the hæmorrhage it is easy to give another one, and if too much air is given at once a reaction is apt to occur and add to the patient's discomfort.

One patient, a girl of eighteen, had slight hæmoptysis in August, 1920. She quickly recovered, but was found to have a lesion of the apex of the right lung and T.B. were found in

the sputum. On December 18th hæmoptysis returned and she coughed up 5 oz. of blood, on the 19th 8 oz., and on the morning of the 20th another 8 oz. In the afternoon of the 20th she was coughing up a quantity of blood and a right artificial pneumothorax was induced : 1,400 c.cm. of air were given and the intrapleural pressure was raised from — 11 —8 to — 1 + 5. She had no further hæmoptysis. The first refill was given two days later, the first few injections being as follows :—

December 20th, 1920.	— 11	— 8	1,400	— 1	+ 5
,, 22nd ,,	— 11	— 8	1,200	0	+ 3
,, 26th ,,	— 3	— 1	900	+ 4	+ 6
,, 30th ,,	— 4	— 1	700	+ 5	+ 7

After the initial treatment she had a reaction with a temperature of 103°F, but it fell to normal in thirty-six hours. A slight reaction followed the first and second refills, but after that there were no reactions and the temperature remained normal. She recovered and kept at work until 1934 when symptoms recurred, and she died of tuberculous bronchopneumonia in 1936.

In cases of repeated hæmoptysis artificial pneumothorax may be successful. One of my patients, a man of twenty-three, had hæmoptysis in 1906. He had recurrent attacks at intervals of about a year until 1918, when they became more frequent. In 1921 he was having hæmoptysis up to half a pint every few weeks. In one month he had three attacks and he had been unable to work for over a year. In March, 1921, during an actual attack of hæmoptysis artificial pneumothorax was induced on the right side. This stopped the bleeding and he is now (November, 1936) well and has had no more hæmoptysis. The lung was kept collapsed until July, 1922, but he returned to work in May, 1921, and attended every three or four weeks for refills.

In some chronic cases it is impossible to induce a pneumo-

thorax owing to adherent pleura, and under these circumstances thoracoplasty should be considered.

Thoracoplasty is also indicated in cases of recurrent hæmoptysis associated with chronic fibroid disease.

Phrenic evulsion has been advocated in some cases when pneumothorax could not be induced owing to adhesions, and good results have been claimed. It often fails to have any effect on the bleeding however, and Morriston Davies says a fatal hæmoptysis may occur some twenty-four to forty-eight hours after the operation owing to venous stasis of the lung following the rise of the diaphragm.

The treatment of hæmoptysis must, of course, depend on its severity, but the following may be suggested as a basis for treatment.

Put the patient to bed and keep him propped up with pillows in the semi-upright position. A day and night nurse should be employed and no visitors should be allowed, although it may be necessary to let certain members of the family see him. The patient must be reassured, for however well he may control his feelings he is certain to be very alarmed and anxiety leads to restlessness and a quickened heart-beat. In some cases the physician is able to pacify the patient by his personality and assurance that all will be well, but it is often best to give a small injection of morphia. This will tide the patient over the initial period of terror, will relieve restlessness and assist clotting to take place. It is not necessary to give enough morphia to stop cough and it is very rarely necessary to repeat the injection. I agree that morphia should not be given as a routine, but, in my opinion, it has a very real value in the treatment of certain cases of hæmoptysis. The nurse should be instructed to keep the patient as quiet as possible. Too much fussing should be avoided. Cough should be encouraged if there is anything to be expectorated and it should be explained to the patient that there is a certain amount of blood in the tubes which has to be coughed up and that there is no reason to feel

alarmed about it. On the other hand, the continuous dry, useless cough, which so many patients develop, should be checked—it is largely a matter of habit and the patient can usually control it. A good cheerful nurse who has the patient's confidence is of the utmost value.

The bowels should be kept open and for this purpose senna pods, cascara, agarol or some simple aperient are sufficient. The diet should be light and not stimulating, but it need not be cold. Ice to suck or iced drinks are harmful. Fluid should be restricted and should not exceed a pint and a half daily during the actual hæmorrhage ; 8 oz. of pounded fish or chicken, or 5 oz. of rabbit, raw meat sandwiches, bread and butter, milk and eggs are suitable foods, and it is not necessary or even wise to keep a patient strictly on a milk diet.

No medicinal treatment is required in mild cases, but as a placebo to comfort a nervous patient it may be wise to give a mixture such as :—

Pot. Brom.	gr. xv.
Tinct. Hyoscyami.	℥xv.
Syr. Aurantii.	℥i.
Aq. ad.	℥ss.

three times a day.

An icebag on the chest is harmless and may be ordered if necessary for its effect on the mental state of the patient.

By these methods the great majority of mild cases will soon improve. The patient should be kept in bed for at least three days after the sputum is free from all traces of blood, and in the event of bronchitis or fever following the hæmoptysis he should be kept propped up in bed and given an expectorant mixture. After-treatment must depend on the condition of the patient and whether any activity of the tuberculosis has been set up by the hæmoptysis.

In cases of moderate severity, where the hæmoptysis continues for six or more hours, an intravenous injection of

10 c.cm. of a 10 per cent. solution of calcium chloride may be given. It is, of course, quite useless in a large hæmorrhage due to a ruptured pulmonary aneurysm, but may help in a case of congestion or oozing.

A better method of treatment for moderate or continued cases is to bind the limbs as already described.

In more severe cases artificial pneumothorax is indicated, and in any case where the bleeding continues for more than three days and there is no contra-indication a pneumothorax should be induced not only to stop the hæmoptysis but to lessen the chances of a subsequent tuberculous broncho-pneumonia.

Pleurisy

A. Dry Pleurisy. Pleurisy is a very common occurrence during the course of pulmonary tuberculosis. Sometimes it is painless and it may be unsuspected until revealed by post-mortem examination. There may, however, be considerable pain in a case of acute dry pleurisy, and to relieve this the affected side should be rested as much as possible. In mild cases rest in bed with antiphlogistine or some other warm application over the painful part will be sufficient. Strapping the side with rolls of adhesive plaster passing from the vertebral column completely round the chest to beyond the sternum, and applied when the chest is in a state of deep expiration, will rest the lung and alleviate the pain. It is essential to make certain that the strapping is put on evenly as if it becomes crumpled it is very uncomfortable. Sodium salicylate is frequently given, and a draught consisting of Phenazone, gr. x., Sp. Amm. Arom., ʒss., Aq. Chlorf. ad ʒi., will usually relieve the pain. In severe cases, however, an injection of morphia is required to give relief. Cupping over the painful area or the application of leeches is often very comforting to the patient. In really bad cases, however, the induction of a partial pneumothorax is the best treatment and should always be considered in any case bad enough to

justify giving morphia. It is not necessary to give more than 200 or 300 c.cm. of oxygen, which are quite enough to separate the visceral and parietal layers of pleura. When absorption of the oxygen occurs and the two pleural surfaces again come into contact the pain does not, as a rule, return, because a thin layer of lymph has formed over the inflamed surfaces. Oxygen is preferable to air in these cases as it is more quickly absorbed, and if the pleural surfaces are kept apart for long an effusion is very likely to result. In cases where the underlying lung is affected and it is desired to maintain an artificial pneumothorax it is better to use air. In certain cases, of course, it is not possible to produce any collapse owing to adhesions, but in the majority where a pleural rub can be heard a sufficient collapse can be obtained to relieve the pain and the relief is often dramatic. Even in pleurisy from malignant disease the pain may be stopped by pneumothorax. One of my patients with pulmonary neoplasm developed pleurisy which caused great pain, prevented her from sleeping and would not yield even to morphia. A very partial pneumothorax was induced and the pain immediately ceased and did not return throughout her illness although the lung quickly re-expanded and a coarse pleural rub could be both heard and felt.

In cases of continuous dragging pain, especially that resulting from diaphragmatic pleurisy or adhesions, phrenic evulsion may be successful, and its possibilities should always be borne in mind.

In chronic fibroid cases there is occasionally very severe pleural pain and this may be relieved by the injection of alcohol into the intercostal nerves near the angles of the ribs.

B. Pleural Effusion. Clear serous fluid is usually straw-coloured, but may be tinged with red from admixture with blood. It may form in the pleural cavity as a transudate, the result of circulatory stasis, or as an exudate which develops from inflammation. Lord (5), in a description of

pleural fluid, says that a transudate and exudate differ, as shown in table 18.

TABLE 18

	Transudate.	Exudate.
Specific Gravity.	1010—1015 Hydræmic fluids having lower specific gravity than transudates from venous stasis.	1018 or more.
Albumin.	1—3 per cent.	4 per cent. or more.
Fibrinogen.	Trace.	Larger amount.
With acetic acid.	Very slight precipitate.	Bigger precipitate.
Coagulates.	Slowly or not at all.	Quickly.

It has been stated that when the predominant cells are small lymphocytes the effusion is due to tuberculosis, if polymorphonuclear leucocytes predominate the cause is some pyogenic infection (such as Pneumococcus, Streptococcus, Staphylococcus), whereas an excess of endothelial cells suggests an effusion of mechanical origin. Lord points out that the intensity of the pleurisy as well as the cause affects the cells in the effusion. Thus in long-standing transudates the endothelial cells may diminish and the lymphocytes increase. A transient excess of polymorphonuclear leucocytes may occur in the early acute stage of a tuberculous effusion and a secondary infection in a chronic effusion will affect the cells. Gloyne (10) states that the predominance of polymorphonuclear cells in the cell count of a serous effusion, together with the presence of numerous tubercle bacilli, is strong presumptive evidence of spontaneous pneumothorax. In malignant disease the cells of the effusion are similar to those found in transudates, endothelial cells being in excess.

An excess of eosinophil cells in the effusion is regarded by some as evidence against tuberculosis.

Tubercle bacilli are not found in the early stages, but in the long-standing tuberculous effusions they are usually to be found and often in large numbers. Inoculation of a guinea-pig may reveal the presence of tuberculosis, although the bacilli were not found microscopically, but even this test is usually negative in the early stages of the effusion. I have known patients with tubercle bacilli in the sputum and yet the liquid from the pleural cavity was negative to guinea-pig inoculation.

Treatment. As soon as an effusion develops it is wise to remove some 5 c.cm. for examination in order to confirm the diagnosis and exclude empyema. If the fluid is clear and is of tuberculous origin it is best to allow a natural absorption, which usually occurs in the course of a week or two. No medicinal treatment is required except to relieve the symptoms, such as constipation, sleeplessness, etc., but the patient should be kept in bed until the temperature is normal and the fluid sufficiently absorbed for the heart and mediastinum to be in the correct position.

Auto-serotherapy has been practised by some physicians. It consists in the subcutaneous injection of 1 c.cm. of the exudate directly it has been aspirated, but in my experience this method has not proved of any value.

Some patients with clear tuberculous effusions run a high hectic temperature for several weeks, but it eventually settles and does not indicate the onset of empyema. An intermittent or hectic temperature, which alters and becomes a high continuous one, is of serious omen, as it usually indicates the development of miliary tuberculosis.

Most authors agree that aspiration should be avoided during the acute stage but there is considerable difference of opinion as to whether or when it should be undertaken. On the one hand it is argued that to leave an effusion is to risk extensive adhesion of the visceral and parietal pleura

and possibly extreme fibrosis of the lung. On the other hand, it is held that an effusion is Nature's method of resting the lung and that aspiration by relieving the tension is likely to lead to an acute exacerbation of tuberculosis in the lung tissue. It must be borne in mind that a tuberculous pleural effusion is secondary to a lesion in the lung; activation of disease in the lung after the effusion is therefore due to a flare-up of an existing pulmonary lesion and not to a fresh one spreading from the pleura. In some countries the practice is to aspirate the effusion, replace with air and maintain a pneumothorax. It is claimed that by this treatment the chance of subsequent active tuberculosis of the lung is lessened and that if pneumothorax is not done, adherent pleura will almost certainly occur, so that should collapse of the lung become necessary later it will mean thoracoplasty. In my opinion it is best to leave a tuberculous effusion to absorb naturally, and apart from taking a sample, not to aspirate unless pressure symptoms become severe, the effusion shows no sign of absorption, pyrexia persists after two months, or activity exists in the underlying lung so that pneumothorax is indicated.

In a large effusion causing pressure symptoms absorption may take place after some 8 or 10 oz. have been aspirated, but if it is decided to remove more than 15 oz. some air or oxygen should always be introduced to replace the liquid withdrawn. If this is not done the sudden expansion of the lung is apt to set up an acute tuberculosis by activating a quiescent lesion. Moreover, the rapid lowering of the intrapleural pressure may cause œdema of lung, faintness, or even sudden death. Oxygen should be used to replace the liquid if one wants the lung to expand in a day or so, but air is better if it is desired to maintain a pneumothorax (6).

In order to aspirate or replace the pleural effusion with gas I use the apparatus shown in Fig. 11. It has three nozzles, one of which can be used for the purpose of drawing the liquid from the pleural cavity into the syringe. By

turning the handle the syringe is then connected with the second nozzle, through which the liquid can be expelled into a receiver, and a further turn of the handle connects the syringe with the third nozzle, through which any solution can be drawn up into the syringe if it is needed to wash out the pleural cavity. If pleural lavage is not required the

FIG. 11.

third nozzle will not be needed, and in such cases Dieulafoy's syringe, which has a two-way cock, is equally convenient. Some prefer Potain's vacuum aspirator for simple aspiration. It is certainly quicker but the effusion is sucked into the exhausted bottle with a series of spurts, and although the rate of flow can be controlled by the tap, it is not so easily done as with a syringe.

Technique of Simple Aspiration. The patient should lie on the healthy side so that the opposite axilla is uppermost. The best site for aspiration is the fifth or sixth intercostal space in the mid-axillary line and the skin over this area should be cleansed and painted with tincture of iodine; 0·2 c.cm. of a 2 per cent. solution of novocain is injected into the skin through a No. 20 Record needle. This produces a small bleb and renders the skin anæsthetic. Then through this bleb 2 c.cm. of the novocain solution are injected into the subcutaneous tissue and right down to the parietal pleura, against which the last 0·5 c.cm. of the solution should be

injected. The needle should then be pushed through the parietal pleura and a few drops of the effusion withdrawn. This is important in order to make certain that an effusion actually is present and to ascertain its nature, for on this the subsequent procedure largely depends. All this should, if possible, be done with the same small needle, but it may be necessary to use a slightly larger one in order to reach the pleura. The patient should be left for five minutes, by which time the pleura and skin will be quite insensitive to pain. The needle connected with the aspirating apparatus is then introduced into the pleural cavity and the required quantity of liquid aspirated. In an ordinary simple effusion it is usually sufficient to remove 15 oz. in order to relieve pressure symptoms or to start absorption, but if more has to be aspirated it is best to introduce a little air or oxygen into the pleural cavity.

Technique of Gas Replacement

1. *With one Needle.* The patient should be in the same position and the skin and pleura anæsthetised in the same way as for a simple aspiration. Instead of the ordinary aspiration needle a pneumothorax needle (Fig. 12) is

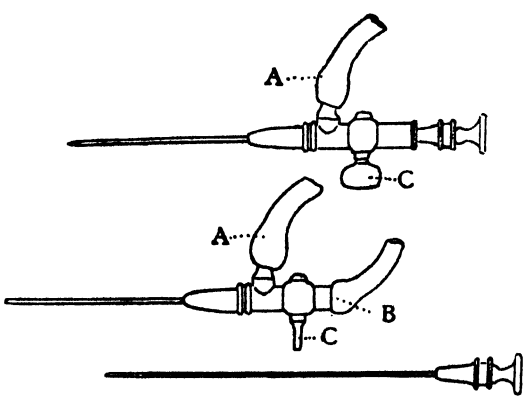

FIG. 12. Needle with tubing as used for fluid replacement.

used. A rubber tube A connects the needle with a pneumothorax apparatus and tube B connects it with the aspirating syringe. Tube A is clipped and the needle is then inserted into the pleural cavity and the trocar withdrawn. Tube B is then fitted on to the end of the syringe and 800 c.cm. of the effusion are aspirated. Tap C is then turned off and after unclipping tube A, some 200 c.cm. of air or oxygen are introduced. This produces a gas bubble, so that in order to aspirate more liquid it may be necessary to make the patient sit up or turn more over to the affected side. Removal of the effusion and introduction of the gas should then be continued alternately in small stages. If too much liquid is withdrawn at a time the patient may feel dizzy or may actually faint, but the introduction of a little gas immediately relieves the feeling of faintness. The end pressure should be left slightly below atmospheric, such as -5 -2, and it is usually necessary to introduce about 800 c.cm. of gas for every 500 c.cm. of liquid aspirated. If quick re-expansion of lung is required a lower pressure should be left and oxygen used instead of air.

2. *With two Needles.* The skin and pleura over the fifth intercostal space are anæsthetised as already described, and the same is done to the skin and pleura over the seventh intercostal space just below the angle of the scapula. In both areas the needle is pushed through the parietal pleura and a few drops of liquid removed in order to make certain that the needle is actually in the effusion. With the patient lying on the healthy side a needle connected with a pneumothorax apparatus is introduced through the prepared site in the axilla and gas allowed slowly to flow into the pleural cavity. A second needle connected with an aspirating apparatus is then introduced through the site prepared in the back and aspiration is performed at the same time as gas is entering the pleural cavity through the axilla. By regulating the rate of aspiration and of the introduction of

the gas it is possible to keep the intrapleural pressure constant throughout the procedure.

After gas replacement the case is similar to an ordinary one of artificial pneumothorax complicated by effusion and should be managed in the same way if it is intended to maintain the pneumothorax. If, however, it is desired to let the lung re-expand, oxygen should have been used for the replacement and any re-accumulation of the effusion should be treated by further aspiration and replacement with small quantities of oxygen.

Sometimes, and especially as a complication of artificial pneumothorax, the effusion develops into tuberculous pus. Two types of tuberculous empyema have been described (7).

The first a cold pleural abscess or benign pyothorax. This condition may persist for years like an ordinary clear effusion without producing any toxæmia or impairment of the general health of the patient. In such a case some authorities advocate leaving the empyema and not aspirating unless it increases in size or begins to produce any symptoms. In my opinion, however, it is safest to aspirate and repeat if the pus re-accumulates and to try to get re-expansion of the lung. Although I agree that many patients tolerate a benign pyothorax well, yet there are definite dangers such as the development of very thick pleura or rupture of the empyema into the lung leading to a bronchial fistula and a serious risk of secondary infection.

The second type is known as malignant pyothorax, and in this case toxæmia is usually severe and the outlook extremely grave. It is often associated with spontaneous pneumothorax occurring in a tuberculous patient or complicating artificial pneumothorax as a result of a torn adhesion.

Aspiration is here indicated and it is usually best to wash out the pleural cavity. A solution of 1 in 4,000 methylene blue may be used and is useful as an indication of the presence of bronchial fistula, for if one is present the blue will appear in the sputum. Alepol, which is made from sodium

hydnocarpate, or a solution of iodine 0·5, potassium iodide 1 and sterilised water 4,000 (Jessen's fluid) may be used, but in my opinion the effect of the irrigation is mechanical and the choice of solution unimportant. Ordinary saline is quite satisfactory. Dakin's solution or Eusol are the best if there is much fibrinous deposit on the pleura, as they definitely help to clear it and so prevent organisation and the formation of thick fibrous pleura which will permanently prevent re-expansion of the lung. These solutions consist of chlorinated lime and are irritating to the bronchial tubes, so that they cannot be used if there is a bronchial fistula. Oleothorax has also been advocated in cases of tuberculous empyema and the oil commonly used is 5 per cent. gomenol in olive oil. In my experience this has not proved successful. But I have found Crocket's (8) solution helpful in cleansing the pleural cavity and rendering the condition of the patient more suitable for thoracoplasty. Crocket states that an oily solution floats on the top of the effusion and advocates a compound of acriflavine and gelatin which mixes in even suspension with the effusion. A 2 per cent. solution of gelatin in normal saline is made and acriflavine added just before use to make a dilution of 1 in 2,500.

If, however, the lung fails to re-expand as a result of simple aspiration and irrigation with saline it is extremely unlikely that any other irrigating fluids will succeed although they may help to cleanse the pleura, relieve toxic symptoms and improve the general condition of the patient.

Technique of Gas Replacement and Pleural Irrigation

The patient is prepared and put in the same position as already described for gas replacement, and oxygen is introduced from the pneumothorax apparatus through the axilla into the pleura in exactly the same way as in ordinary gas replacement. Through the anæsthetised skin at the back is inserted an aspirating needle connected with the three-

nozzled syringe shown in Fig. 11. As much as possible of the pus is aspirated whilst the intrapleural pressure is kept negative, but not sufficiently low to produce faintness, by the slow flow of oxygen entering through the axilla. The irrigating solution is then drawn into the syringe and injected into the pleural cavity, the quantity introduced being roughly the same as the amount of pus removed. The liquid is then again aspirated and more of the solution introduced and this procedure is repeated two or three times until the liquid in the pleural cavity is clear. A negative intrapleural pressure of about — 15 — 8 should be left if the intention is to encourage re-expansion of the lung. The object of introducing oxygen is to prevent the pressure from falling too low and to control the final pressure. Very little oxygen is required except at the end and it is not wise to let any flow in at the same time as the irrigating solution is being injected into the pleural cavity. Should the pressure rise too high, gas should be removed from the pleural cavity. Some prefer to introduce the irrigating solution through the axilla, and in this case aspiration is performed in the ordinary way through the needle in the back. The pneumothorax needle (Fig. 12) is connected through tube A, with the pneumothorax apparatus and through a long rubber tube B with a funnel. After aspirating some of the pus and allowing a little oxygen to enter and form a bubble in the axilla tube A is clamped, tap C is turned on and the irrigating solution allowed to flow into the pleural cavity by pouring it into the funnel which is held above the patient.

Spontaneous Pneumothorax

This may occur suddenly or gradually. In some cases it produces violent pain and severe dyspnœa, which may rapidly prove fatal. This type has been called *pneumothorax suffoquant.* I saw one case mistaken at first for a perforated gastric ulcer. In other cases a patient who complains of dyspnœa for several days, but not enough to

prevent him from working, is found to have a complete pneumothorax. Sometimes the condition is found accidentally by routine clinical or X-ray examination in a patient who has no symptom suggesting it.

In many cases the lung quickly re-expands and no treatment beyond a few days' rest is required. If the symptoms persist, however, and there is no sign of re-expansion after a week, it is best to remove some of the gas from the pleural cavity and to assist the lung to begin to expand. The gas should be removed gradually and no more than 700 c.cm. taken off at the first aspiration, otherwise a paroxysm of cough, severe dyspnœa and frothy expectoration are likely to occur.

In the severe acute cases where there is urgent dyspnœa gas should always be aspirated. It is often under great pressure and can be heard rushing out when the needle is put into the pleural cavity. The perforation in the visceral pleura is sometimes valvular, so that the air, which is forced into the pleural cavity when the patient coughs, cannot escape, and the intrapleural pressure consequently becomes very high and may lead to great displacement of the heart and mediastinum. In such a case the aspirating needle should be left in the pleural cavity with a long piece of rubber tubing leading from the needle to a bottle of water on the floor. This forms a water valve which allows the gas to escape, but prevents any air from entering the pleural cavity. If there is much dyspnœa, even when the pressure has been reduced to atmospheric by leaving the aspirating needle in the pleural cavity, it may be necessary to make the pressure negative. This may be done as suggested by Boland (9) by leaving in the pleural cavity an aspirating needle connected by a rubber tubing with a glass tube leading through the cork of a bottle placed on the floor. Another glass tube passing through the cork is connected with a Sprengel's pump fixed on a water tap so that when the tap is turned on a negative pressure is produced.

In some cases there is considerable dyspnœa, but only a low intrapleural pressure, and in these there is usually a nervous element so that the symptoms quickly subside after the patient has been given a small dose of morphia.

Tuberculosis accounts for most of these cases, and now that X-ray examinations are so frequently made it is known that a partial pneumothorax is quite a common occurrence in cases of chronic pulmonary tuberculosis. In the great majority of tuberculous cases an effusion develops and contains a large proportion of polymorphonuclear cells. When an excess of these cells is found in a tuberculous effusion perforation of the visceral pleura has probably occurred (10). The effusion, though clear at first, is apt to develop into tuberculous pus and mixed infection is not uncommon.

Spontaneous pneumothorax is more common in non-tuberculous cases than was formerly believed. Kjaergaard (11) described fifty-one cases all of whom recovered and only one subsequently developed tuberculosis. These cases are probably due to rupture of an emphysematous bulla. It occurs in men much more commonly than in women and they are usually under forty-five years of age. Recurrence is common, but if the patient is put to bed the lung usually re-expands in a few days. An effusion is very rare in non-tuberculous cases and this fact agrees with the findings of artificial pneumothorax, where over half of the tuberculous cases develop some liquid during treatment, but the non-tuberculous cases practically never get an effusion.

When a spontaneous pneumothorax is followed by the development of tuberculous pus in the pleural cavity the patient almost always dies of the disease sooner or later. Tubercle bacilli are often absent at first in the effusion, but later appear in large numbers, and a secondary infection is liable to occur. Repeated aspiration is apt to cause sinuses and make the prognosis still more grave. Even if the original perforation in the visceral pleura does heal the pleura tends to become very thick and to prevent re-

expansion of the lung so that the pleural cavity is nothing more than a bag of tuberculous pus. Every effort should be made therefore to re-expand the lung.

In order to stimulate re-expansion of the lung the pus should be aspirated completely and the intrapleural pressure left negative. If the pus is thick 15 or 20 c.cm. of Gauvain's modifying fluid (guaiacol, 2 grm., creosote, 2 grm., iodoform, 5 grm., ether, 10 grm., sterile olive oil, 100 c.cm.) or 30–50 c.cm. of a 0·5 per cent. solution of chloramine T. may be injected into the pleural cavity a day before aspirating to render the pus more liquid. Of course, if the perforation in the visceral pleura is open the patient will taste this modifying fluid, and failure to taste it may be taken as evidence that the perforation has healed and therefore that there is more likelihood of the lung re-expanding. The technique of pleural irrigation and gas replacement have already been described. The pleura should be washed out and if the lung fails to re-expand thoracoplasty should be performed. The condition of the patient should, however, be made as good as possible by medical treatment and pleural lavage before the operation is undertaken.

The operation is not free from danger, especially in cases where there are sinuses, but I believe the patient is in far greater danger if left alone. If the perforation in the visceral pleura remains patent the danger is considerable, not only of the pleural cavity being infected but of dissemination of disease in the lungs. In one case of spontaneous pneumothorax, when some lipiodol was introduced into the pleural cavity, X-ray showed traces of it in both lungs on the following day, suggesting that the lipiodol, and consequently the tuberculous pus also, is aspirated into both lungs by the inspiratory efforts which accompany cough. Under modern conditions, and in the hands of a surgeon accustomed to this type of work, the operation has not the same terrors it possessed a few years ago, and I feel convinced that it is more often advised too late than too early

in these cases. The reason for this is that so many cases do well for a time and appear to be recovering when an accident such as infection of the cavity, as shown by the beginning of pyrexia, return of cough or sputum, leads to a steady or rapid decline in health and ultimately renders thoracoplasty essential. West (12) gives the general mortality from spontaneous pneumothorax as 70 per cent. In my opinion, the following conclusions may be drawn :—

1. Spontaneous pneumothorax of simple or non-tuberculous origin usually heals quickly and is not complicated by the formation of effusion. If the lung does not re-expand after a few days some gas can be removed from the pleural cavity and this usually leads to re-expansion of the lung.

2. Spontaneous pneumothorax, followed by the development of effusion, is almost always of tuberculous origin. In some cases the liquid is absorbed and the lung re-expands with or without aspirating. If, however, the liquid turns into tuberculous pus the prognosis becomes much more serious and steps should be taken to cause re-expansion of the lung. If there is no sign of re-expansion thoracoplasty should be performed whilst the condition of the patient remains good. The operation is also indicated if the perforation in the visceral pleura shows no sign of healing after three months.

3. In some cases of pulmonary tuberculosis spontaneous pneumothorax is beneficial, and it is then best to keep up an artificial pneumothorax by giving refills and so keeping the lung collapsed.

Cavities

It must be borne in mind that cavities occur at some stage in the disease in the great majority of patients with clinical pulmonary tuberculosis. They may be associated with disease that heals or with disease that proves fatal. It is only when they become chronic that they in themselves are the danger. In the acute and subacute stages it is the

tuberculous lesion in the lung and not the actual cavity that is dangerous and needs treatment.

They may be considered under five groups.

1. Recent cavity with a ragged wall of necrotic tissue as seen in acute tuberculosis of lung. Many of these cases settle down and the patient may make a complete recovery, but a rapid spread through the lungs is frequent, so that artificial pneumothorax is urgently indicated, not because of the cavity but because of the disease producing it. It is wrong to postpone pneumothorax until a cavity appears.

2. The subacute cavity which is a stage later than the above. It is very commonly found in patients when they first present themselves for treatment. The disease either started insidiously or the acute stage has subsided and the patient is often non-febrile, at any rate whilst resting, but has cough, sputum and physical or radiological signs of cavitation in one or both lungs. This is the type of case that improves so frequently as soon as the patient is made to rest. Jaquerod (18) describes a number of patients with advanced disease who have made a complete recovery by simple rest at Leysin. Climatic conditions are not of the first importance, for similar cures occur in all countries, and numerous cases have been reported where an attempt at pneumothorax having failed the patient is treated by simple rest and sanatorium treatment and has made a complete recovery. In my opinion it is safest to induce an artificial pneumothorax in these subacute cases if the disease is unilateral whether cavities can be demonstrated or not. In any case there is probably some cavitation. In bilateral cases the presence of cavities should be taken as an additional indication for inducing pneumothorax. Although I think artificial pneumothorax should almost always be tried in these subacute cases, if it fails owing to adherent pleura, the patient should not immediately be subjected to thoracoplasty in an attempt to close the cavities since they frequently heal by rest alone.

3. The cavity lined with a fibrotic wall but containing pus. This is the so-called open cavity and the patient expectorates sputum often loaded with tubercle bacilli. Here the cavity should be collapsed and preferably by pneumothorax, but failing this by surgical means. The cavity forms a breeding ground for the bacilli and bronchogenic spread and a fatal issue is only a matter of time.

4. The dry cavity with a firm fibrotic wall. It is often best to leave this alone unless the disease is progressing or symptoms such as hæmorrhage necessitate treatment, when thoracoplasty is usually indicated.

5. The cavity which has no signs or symptoms and is discovered only by X-ray by an annular shadow which was formerly called a pleural ring. The mere finding of such a shadow need not affect treatment, which should depend on the associated symptoms.

McMahon and Kerper (14) investigated 296 patients with cavities at Loomis sanatorium; 234 of these had a preliminary period of observation and 62 an early collapse of the lung. Of the 234 the cavity closed in 65 (27·75 per cent.). In 83 of the remaining 169 collapse therapy was given and closure obtained in 23 (27·75 per cent.). Thus closure was obtained ultimately in 88 (37·6 per cent.) of the 234 cases who had preliminary observation. Of the 62 who had immediate collapse (54 artificial pneumothorax and 8 phrenicectomy) the cavity closed in 27 (43·5 per cent.). In 52 per cent. of the 234 observation group, however, but in only 7 per cent. of the immediate collapse group the cavities were bilateral. Spread of disease occurred in 25 per cent. of the early collapse group and 24 per cent. of the whole series. Spread is especially liable in febrile cases. Prognosis varies with the size of the cavity. Spontaneous closure occurred in 40 per cent. of cavities measuring 2 × 2 cm., in 20 per cent. of those of 3 × 3 cm. and in 6 per cent. if over 4 × 4 cm. Right-sided cavities had twice the percentage of spontaneous closure of left and three times that of bilateral cavities.

Healing is less likely to occur in peripheral cavities and thick-walled ones and the bad age group is 15–25 and over 45.

Poisonous Gases

The effects of gassing on pulmonary tuberculosis were brought into prominence during the war. It was thought at first that the acute symptoms produced by the gas would frequently be followed by tuberculosis, either by activation of an old lesion, or by the development of tuberculosis in the damaged lung. Such, however, is not the case, and experience has shown that although chronic bronchitis may follow gassing, the patients are no more liable to develop tuberculosis than are ordinary bronchitics. Moreover, it appears that old lesions are not likely to be activated by gassing. Mustard gas injures the mucous membranes and trachea, and although causing very severe damage, does not affect the smaller bronchi as the asphyxiating gases do. Recently 200 cases have been analysed by the Ministry of Pensions to show the late effects of gas (15).

Of these, 9 were accepted later as pulmonary tuberculosis due to gassing, 141 chronic bronchitis, 16 cardiac conditions, 8 neurasthenia, 26 other conditions.

Of the 9 tuberculous cases, T.B. were present in 6. In the other 3 there was chronic bronchitis, but tuberculosis was never proved, and did not develop. In only 2 cases did the tuberculosis appear to be directly activated by gassing.

Laryngitis

Tuberculous laryngitis is a common complication of pulmonary tuberculosis and is occasionally the first symptom. Persistent or recurrent huskiness should always make one bear in mind the possibility of early tuberculosis of the lung.

St. Clair Thomson (16) said that the interarytenoid region is the one most commonly affected. A simple laryngitis affects the whole larynx. Any one-sided congestion or

irregularity is suspicious of tuberculosis, which affects most commonly the posterior part of the larynx, whereas carcinoma affects the anterior part and spreads only from one centre so that a second separate focus is not found. Morell Mackenzie said that he was doubtful if he had ever seen a case recover; nowadays 25 per cent. recover, but tuberculous laryngitis is a very serious condition, for nearly three out of every four patients are still doomed to death. Failure to respond to treatment is a bad sign, but even extensive ulceration may eventually heal. Treatment consists in resting the voice by silence. If the patient must talk it should be in whispers. Lactic acid application, tuberculin or artificial light has not proved satisfactory in St. Clair Thomson's experience and sometimes makes matters worse. Treatment with the galvano-cautery is sometimes helpful and artificial pneumothorax often leads to prompt improvement in the laryngeal condition. Smoking is definitely harmful and should be stopped altogether. Raine and Banyai (17) advocated an operation to expose the superior laryngeal nerve, inject it with 95 per cent. alcohol and crush and sever it in order to relieve pain. Injection of alcohol into the superior laryngeal nerve is not always successful. Except in severe cases pain is relieved by inhaling through a Le Duc's tube orthoform or anæsthesin before meals.

Bronchitis and Mixed Infections

When a patient with chronic bronchitis develops pulmonary tuberculosis the prognosis is grave. In any case a liability to bronchitis in a consumptive is a serious complication and necessitates special treatment. Such patients must be kept warm and it is harmful to try to harden them by getting them acclimatised to cold fresh air. Climatic treatment is indicated and the winters should be spent in a warm, sunny district, where the temperature is as constant as possible. If the patient cannot afford to go away for the

winter he should be kept in well-ventilated but warm rooms and allowed out only when the weather is suitable.

It has been suggested that the tubercle bacillus itself is not very harmful and that the serious symptoms in pulmonary tuberculosis are due to superimposed infection by pyogenic organisms. Attempts have been made to treat this mixed infection by anti-streptococcal, anti-pneumococcal and other sera and vaccines, but with no striking success.

Pregnancy

Pregnancy appears to have no harmful effects on the course of pulmonary tuberculosis, but an acute exacerbation may follow parturition so that the question of terminating pregnancy in the early stages often arises. There is difference of opinion on this matter, but most agree that if seen within the first four months, pregnancy should be terminated: (1) In cases of acute tuberculosis; (2) in cases where the woman is just holding her own. This is the usual type, as it includes the patient who becomes pregnant soon after leaving a sanatorium, and who still needs the very greatest care to avoid a relapse.

In chronic fibroid cases the patient usually gets through the confinement and remains well. If seen after the fourth month of gestation it is often as harmful to produce abortion as to leave the pregnancy to run to term, but it is most important to give the patient prolonged treatment afterwards. If she has to leave a sanatorium for the actual confinement, she should be readmitted as soon as possible afterwards.

Some authorities hold the view that abortion is as dangerous as labour and think it best to let pregnancy go to term. Others feel the danger of an exacerbation after labour is so small that provided the patient is well looked after the termination of pregnancy is not necessary.

It certainly is hard to deprive patients of marriage or of having children because they have had an arrested tuberculous lesion. During activity, however, most authorities will agree that pregnancy should be avoided and it is in the acute rather than in the arrested stage that one can apply the dictum, "If a girl no marriage; if a wife no pregnancy; if a mother no suckling."

Crocket (18) thinks that pregnancy is beneficial to patients with pulmonary tuberculosis because the increased pressure causes the diaphragm to rise into the thorax and rest the lung. The elastic tissue of the healthy lung is more resistant than the diseased areas to pressure, which is therefore selective and the circulation through the tuberculous portions is lessened. After labour the pressure falls, the lung opens out and dissemination of infection in the lung or throughout the body may occur. He therefore advocates artificial pneumothorax or, failing this, phrenic evulsion and bandaging the thorax after labour from the lower ribs upwards. He also thinks that Cæsarean section is safer than a natural delivery, but if the latter it should be induced a fortnight early. In any case prolonged sanatorium treatment is needed afterwards.

Mayer (19) advises treatment with crisalbine during pregnancy. Of thirty-six cases two died from acute exacerbation of disease after parturition but the other thirty-four had no increase of their disease. All the babies of these patients were normal.

Floyd (20) says that all unnecessary burdens should be avoided by patients with pulmonary tuberculosis and that in the case of pregnancy there is an extra demand on the calcium, iron and iodine. Hypertrophy of the parathyroids causes a further drain on the calcium, the basal metabolism is raised 30 per cent., the renal threshold for sugar is lowered and there is a glycogen deficiency in the liver. Labour throws a great strain upon the patient, so that in his opinion pregnancy and especially labour are dangerous but both

may be rendered safer by artificial pneumothorax or phrenic evulsion.

Lloyd and Richard (21) have studied intrapleural pressure during pregnancy and labour. They state that violent expiratory effort with closed glottis causes high intrapleural pressure, squeezes blood out of the alveoli into the left side of the heart, compresses the heart itself and the big veins, and the lymph flow may be reduced by 50 per cent. They obtained intrapleural pressures of from + 50 to + 65 in women, and from + 101 to + 249 in men, by getting them to make big expiratory efforts during pneumothorax refills. In one case they record the following pressure before, during and after labour :—

Anæsthesia.	Inspiration.	Expiration.	Remarks.
None.	− 20	− 8	—
,,	− 5	− 2	After refill of 600 c.cm.
,,	− 13	− 3	Between pains.
,,	+ 20	+ 35	During pains.
Gas and O_2 deep.	+ 24	+ 40	Head delivered.
None.	− 8	+ 1	After delivery.
,,	− 11	+ 7	After refill of 500 c.cm.

In one of my patients the following pressures were obtained :—

Refill May 2nd, − 6 − 1, 500 c.cm. − 3 + 1.

Delivery on May 4th and immediately afterwards a refill was given and the following pressures found − 4 −2, 500 c.cm. − 2 + 1.

On May 6th another refill was given, − 5 − 1, 400 − 3 + 1.

Delivery does not appear to cause any appreciable lowering of the intrathoracic pressure and the acute exacerbation of disease in the lung which occasionally follows labour is probably due to some other cause.

Trauma

An old lesion may become activated as a result of injury not only to the chest, but to any part of the body. Thus after a motor smash or other accident active tuberculosis may occur. Tuberculosis following wounds in the war was remarkably rare. Chest wounds and especially retained foreign bodies did appear to have some tendency to activate tuberculosis. Price (15) analysed 50 cases claiming pulmonary tuberculosis as due to chest wounds received during the war. Of these 37 (74 per cent.) had proved tuberculosis, in 29 the injuries appearing to have been big factors in the development of the disease, but in 8 the tuberculosis being independent of the injuries. In the 29 cases there was not only surface injury, but actual penetration of the lung. The tuberculosis was either confined to the injured side or, if bilateral, there were grounds for believing it started on the injured side. Price found that surface injuries, unless involving the thoracic cage and its contents, were relatively unimportant in their relationship to the incidence of tuberculosis. He states that in the cases analysed, the average length of time between the date of injury and recognition of tuberculosis was eleven years.

Collapse of Lung

This may be associated with pulmonary tuberculosis (22), and especially the chronic fibroid form. It is probably due to blocking of a bronchus or bronchiole by secretion. Lobular collapse may occur or the lobules may coalesce and produce massive collapse. The intrapleural pressure is low, the heart and mediastinum are displaced to the affected side and dyspnœa is a constant symptom. There may also be persistent tachycardia and some pyrexia.

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CHAPTER VII

TUBERCULOSIS IN CHILDREN AND BOVINE TUBERCULOSIS

TUBERCULOSIS in children is essentially the same as in the adult, but largely for anatomical reasons and because a dose of infection which would be small for an adult might be enormous for a child, the course of the disease is usually more acute in the child.

The so-called primary complex, Plate VIII., consists of:—

1. A primary focus at the site where the tubercle bacilli become fixed.
2. Tuberculous lymphangitis from this focus to the glands draining the area.
3. Inflammation and sometimes caseation of these glands.

A primary focus in the lung is usually known as Ghon's focus although it had been described many years previously by Parrot. It may heal without leaving any scar, or leaving a calcified node. Healing may not be complete, so that the focus can form a centre from which a tuberculous lesion spreads into the surrounding lung tissue or around which an allergic inflammatory zone appears. This condition was described by Grancher in 1890 as *spleno-pneumonie*, but is now usually called epituberculosis. Armand Delille (1) regards it as the development of an intense congestive reaction in a previously healthy portion of the lung around a tuberculous focus. The reaction may occur around any tuberculous lesion, and not necessarily a Ghon's focus.

It is benign clinically and usually clears up altogether but may lead to renewed activity in the tuberculous focus, from

which the disease may spread into the surrounding lung tissue, producing fibroid or fibro-caseous tuberculosis.

There may be few symptoms, but there is usually some pyrexia at night associated with fatigue and loss of general health. The diagnosis is made by X-ray. After a few weeks there is gradual improvement followed by a rapid convalescence and recovery.

Parsons (2) thinks epituberculosis is a benign form of tuberculosis and not a pure allergic reaction. In the case of one child he states that tubercle bacilli were found by needling the solid area and again when it was needled in another place, and he points out that this indicates the tuberculous nature of the whole area, as the needle could not twice have chanced to enter a Ghon's focus. It is not entirely an area of collapsed lung, although the X-ray appearances are very similar and there is probably some collapse.

As explained in Chapter I., primary tuberculosis might be regarded as the incubation stage or that stage between infection and the onset of allergy. One effect of allergy is to localise the lesions so that tuberculosis, like cancer, tends to spread by a local extension. Before the body becomes allergic there is rapid spread through the lymphatics and in the case of a large dose of infection a generalised tuberculosis may occur and prove fatal before any resistance develops so that allergy never occurs. When once the body is allergic, resistance to the spread of disease is offered and its subsequent progress should be regarded as secondary, whether it is a direct continuation of the original infection or whether there is a latent period between the formation of the original focus and subsequent activity. Such a latent period may be one of several years.

Tuberculous broncho-pneumonia is not uncommon in the child, probably because owing to its small size a dose of infection which would not be considered large for the adult is sufficient to produce a general bronchogenic spread. This acute and fatal condition is not due to absence of acquired

resistance, for it occurs as a terminal condition in the adult when a large infection is scattered in such cases as hæmoptysis or from a cavity

In the child the passage through the lymphatics of the lung is clearer than in the adult, so that bacilli have more chance of getting through them and into the general circulation, where they may become fixed in some distant organ. Hence non-pulmonary tuberculosis is more common in the child than in the adult.

There is also the so-called adult type of tuberculosis which modern methods of diagnosis show to be less rare than was formerly supposed.

One often sees a child who is pale and delicate. He does not want to play with others and may have slight pyrexia. Such a child is apt to be labelled tuberculous and in support of the diagnosis a positive tuberculin test and a radiograph showing heavy root shadows are produced. Morriston Davies (3) remarks: "The tuberculous child is often plump, while a poorly covered chest that suggests a wicker basket is more suggestive of the catarrhal group." He goes on to say that "the bacillus of Koch should not be made the whipping-boy for our ignorance of the common causes of delicacy in childhood"; general enlargement of the root shadows is not suggestive of tuberculosis. In severe cases of tuberculous infection the diagnosis is clear, in mild ones there may be no signs or symptoms at all. Lloyd and Macpherson (4) with the Mantoux method retested 700 children after an interval of one and a half to two years. They found that 96 per cent. of the cases originally positive at the first test remained positive. In 81 cases negative at the first test there was a positive reaction at the second without any notable disturbance in the health of the child having occurred. Seven of these 700 cases had tuberculosis when first examined, but of the remaining 693 not one developed any signs or symptoms of clinical tuberculosis during the one and a half to two years. It is not enough to diagnose

tuberculous infection, since the enormous majority of those infected do not require treatment and never develop clinical tuberculosis, and so a positive tuberculin test is not really helpful but a negative one is of great importance. If it is positive, the stomach contents should be examined for tubercle bacilli and a careful radiological examination made. It is necessary to start treatment in the earliest stages in the case of a patient who needs treatment, but it is the greatest mistake to treat as tuberculous a child suffering from some feeding or other defect. There are a number of people whose lives have been completely spoiled on account of undue treatment during childhood with long residence and perhaps education abroad as a result of a mistaken diagnosis of tuberculosis.

Armand Delille (1) gives the following classification of childhood tuberculosis.

A. Tuberculosis of Primary Infection

1. *Tuberculous Pneumonia of Limited Area.*

Occult benign form.	Initial and perinodular infiltration.
Serious form, sometimes curable.	Caseous tuberculosis.

2. *Extensive or Total Lobar Tuberculous Pneumonia.*

Clinically recognisable and sometimes curable.	(*a*) Initial tuberculosis and perifocal tuberculous pneumonia.
	(*b*) Extensive primary tuberculous pneumonia.
Serious form, rarely curable.	(*a*) Caseous broncho-pneumonia.
	(*b*) Caseous lobar pneumonia.

3. *Primary Cavities Secondary to Caseous Tuberculosis or Caseous Pneumonia.*

4. *Disseminated Nodular Tuberculosis.*

Localised clinical tuberculosis often associated with tuberculous meningitis.	Microscopic or punctiform granulations.
Acute clinical tuberculosis.	Miliary tuberculosis.
Afebrile tuberculosis with slow-forming local lesions secondary to caseous pulmonary focus.	Disseminated tuberculosis.

5. *Glandular Tuberculosis.*

Benign form.	Tuberculous infiltration of tracheo-bronchial glands.
Serious form.	Massive caseation of bronchial glands.

B. Tuberculous Re-Infection

Exogenous or secondary endogenous to an arrested primary pulmonary glandular lesion.

I. *Spleno-pneumonia* (*Epituberculosis*).

(*a*) Lobar.

(*b*) Limited.

II. *Fibro-Caseous Pulmonary Tuberculosis.*

A. Initial Manifestations.

(*a*) Lobar with acute onset.	Lobar tuberculous pneumonia with necrotic areas. Cavities.
(*b*) Lobar with insidious onset.	Fibro-caseous.
(*c*) Insidious form with localised lesions.	Discrete apical infiltration.
(*d*) Multilobar.	Acute caseous broncho-pneumonia with disseminated lesions.

B. *Proliferative Form*, following one of the four above-mentioned forms.

Insidious secondary lesions.
Acute pneumonic.
Interstitial tuberculosis.
Sub-acute caseous broncho-pneumonia with disseminated lesions.

III. *Disseminated Nodular Tuberculosis.*

Acute miliary.
Chronic miliary.
Sub-acute miliary.

It was formerly assumed that infection was by inhalation, and Koch, after the discovery of the tubercle bacillus, thought it took place by inhalation of particles of dried sputum containing the bacilli. In 1903, Von Behring suggested that the bacilli entered in infancy by the alimentary tract and either at once or after a latent period, possibly of many years, were carried by the lymphatics and blood stream to produce disease in the lungs or other organs.

In support of this theory experiments showed that general tuberculosis was produced by feeding animals on large doses of tubercle bacilli. But similar results were obtained by making animals inhale the bacilli, and such experiments only go to show that with very large doses of tubercle bacilli a general infection may occur, however they are introduced.

Tuberculin tests show beyond doubt that only a very small minority of those infected in childhood develop the adult form of tuberculosis, and it is difficult to understand why so many escape if the adult type is merely a late manifestation of a childhood infection. The matter is one of the greatest importance, not only in the management of children suspected of having tuberculosis but in dealing with adults known to have had some tuberculous lesion in their childhood.

Tuberculin tests show that the proportion of reactors

increases as age advances and in the early ages is much greater in children who have been in contact with the disease. Thus in the United States, Opie and McPhedran (5) found a positive reaction as follows :—

TABLE 14

Age.	No contact.	Contact.
	Per cent	Per cent.
0–5	23·3	80
5–10	35·5	80·4
10–15	46·2	91·1
20	100	100

Chadwock and Zacks (6) found positive reactions in 28 per cent. of 101,118 school children.

Lloyd and Dow (7) found the following positive reactions :—

TABLE 15

Age.	No contact.	Contact.	Contact and non-contact.
			Per cent.
0–5	13·6	71·4	27
5–10	31·2	75·9	39
10–15	54·4	76·3	58

Now the mortality rates for all forms of tuberculosis in England and Wales and, according to Lloyd and Dow's work, the number of reactors at the same age periods per 100,000 living in 1928 were :—

0–5 . . .	83	against	27,000	reactors.
5–10 . . .	31·8	,,	89,000	,,
10–15 . .	33·4	,,	58,000	,,

It is clear, therefore, that the overwhelming number of

those infected recover and only a small proportion develop clinical tuberculosis.

Contact with an open case increases the number of reactors enormously, especially in the early ages of 0–5. The mortality rate amongst those exposed is also increased but not nearly so much. Lloyd and Dow (8) found the percentage death rate within the first five years of life for children who had lived from birth in contact with a parent with positive sputum to be 2·6 per cent. They found that for an average English working-class family twice as many exposed children die of tuberculosis within the first year of life as compared with the unexposed.

Lissant Cox in collaboration with others in Lancashire investigated a number of children in 1,063 Lancashire homes, in each of which was living at least one patient (lodger, parent, or other relative) with sputum containing tubercle bacilli. He found that of those that died between birth and the age of five years 3·3 per cent. died from respiratory and 30 per cent. from other forms of tuberculosis. The corresponding percentage for all children in England and Wales who died between birth and five years being 0·6 and 3·8.

3·2 per cent. children of tuberculous mothers died within the first year of life from tuberculosis, and 11·5 from all causes. This is much lower than the result of Calmette, who found that 24 per cent. of all children born of tuberculous mothers died within their first year.

The death rate from tuberculosis amongst young children has shown a great and steady fall for many years, whereas in older children it is in recent years that the fall has been most obvious. This can be seen from the table on p. 132.

Blacklock (9) made 1,800 post-mortem examinations at Glasgow on children from birth to thirteen years of age and found a naked-eye tuberculous lesion in 283 (15·7 per cent.) of them.

The primary lesion was in the thorax in 173 (61·1 per

cent.) cases, and of these 168 (97·1) died of tuberculosis. It was found most frequently in the right upper lobe, then in order of frequency in the right lower, the left upper, left lower and right middle lobes. In the upper and middle

TABLE 16

Deaths from Tuberculosis (all Forms) in Certain Age Groups
(England and Wales)

Age	1898.		1910.		1920.		1935.	
	Deaths.	Rate per 100,000.	Deaths.	Rate per 100,000.	Deaths.	Rate per 100,000.	Deaths.	Rate per 100,000.
0–1	7,062	765*	3,509	391*	1,405	147*	335	56*
0–5	14,030	382	8,896	231	3,988	123	1,405	50
5–10	2,291	66	2,232	61	1,631	46	585	19
10–15	1,907	58	2,004	57	1,830	50	653	19

* Infantile mortality, *i.e.*, deaths per 100,000 live births.

TABLE 17

Age.	Tuberculous lesion post-mortem.
	Per cent.
0–1	7
1–2	29·8
2–3	44·8
3–6	36·3
6–9	27·8
9–10	28·6

lobes the lesion was most commonly situated in the anterior part of the lungs, whereas in the lower lobes it was usually posterior.

In 101 (35·7 per cent.) the primary lesion was in the abdomen and tuberculosis was the cause of death in 77 (76·2) of these.

The bacilli were typed in 110 cases where the primary

lesion was thoracic and found to be human in 107 and bovine in 8. They were typed in 66 of the cases where the primary lesion was in the abdomen and found to be human in 12 and bovine in 54.

In a further series of 62 cases, 10 being from post-mortem and 52 from surgical specimens obtained during life, the following results were noted :—

TABLE 18

	Cases.	Human. Percentage.	Bovine. Percentage.
Cervical glands .	28	10 .. 35·5	18 .. 64·3
Bones and joints .	26	17 .. 65·4	9 .. 34·6
Kidney . .	2	2 .. —	0 .. —
Miscellaneous .	6	2 .. —	4 .. —
Primary thoracic.	110	107 .. 97·3	3 .. 2·7
Primary abdominal . .	66	12 .. 18·2	54 .. 81·8

The high percentage of bovine bacilli found in the abdominal cases and the overwhelming majority of the human type in thoracic cases suggest that the spread of the disease tends to centre round the site of infection, so that when the bacilli enter the respiratory tract the lungs and lymphatic glands are chiefly affected, whereas if entry is by the alimentary canal the abdominal glands are involved. Bovine bacilli would be found more frequently in pulmonary cases if infection took place by way of the lymphatic and venous blood stream or if adult tuberculosis were a recrudescence of a childhood infection. Moreover, Blacklock found a primary lung lesion with involvement of the corresponding tracheo-bronchial glands in 148 of 178 thoracic cases.

That infection was air-borne and not vascular in origin was also suggested by the facts that the primary lesion was found histologically to be broncho-pneumonic and that 86 per cent. of the primary thoracic cases were in children

living in the town and so with a greater chance of air-borne infection.

In 1876, Parrot examined children aged one to seven and found a lesion in the lung in every one of 145 with tuberculous tracheo-bronchial glands and he described these glands as mirrors of the lungs. Since that time others have confirmed his opinion that these tuberculous glands are infected from a primary lung focus, which, however, may be small, and difficult to find. The primary focus is usually single, varies in size from a pin's head to a hazel nut and is often in the caseous stage, but an old calcified focus may be found. Some of the lesions were wedge-shaped with the base towards the pleura.

Histologically, the primary focus starts as a small patch of broncho-pneumonia with many tubercle bacilli, but at first no endothelial or giant cells, and it has no relation to blood vessels. It is apparently the result of inhalation of tubercle bacilli.

There are two groups of lymphatics in the lung, one deep in the lung substance and the other a superficial plexus in the visceral pleura. These two groups are practically independent but there are a few anastomoses between them with valves leading towards the pleura, so that if the deep group are blocked by disease the lymph may drain through the pleural group. Both groups drain into glands at the hilum.

Scattered through the lung and pleura are small collections of lymphoid tissue from which the lymphatic vessels originate.

Blacklock describes four sets of glands in relation to the respiratory system.

1. Broncho-pulmonary. Small intrapulmonary glands at angles of branching small bronchi and extra pulmonary glands close to branches of the main bronchus at the hilum. These glands are freely connected with each other on the same side, but there is no direct communication between the glands of the right lung and those of the left.

2. The inferior tracheo-bronchial glands consist of four or five glands on each side below the bifurcation of the trachea. The glands on one side communicate with those on the other and they drain into the superior group.

3. Superior tracheo-bronchial glands consist of three or four glands on each side close to the superior angle formed by the bifurcation of the trachea. The glands on one side communicate with those on the other.

4. Paratracheal glands along each side of the trachea and receiving lymph from the superior tracheal glands.

The lymphatic glands which drain the region of the primary focus are practically always affected (Plates VII. and VIII.). The focus is commonly situated just under the pleura and might drain into the pleural plexus through the small anastomoses between the superficial and deep lymphatics of the lung, especially if the deep lymphatics are obstructed by disease.

A primary lesion in the upper lobes drains through the broncho-pulmonary lymphatics to the superior tracheo-bronchial glands of the same side and one in the lower or right middle lobes drains into the inferior tracheo-bronchial glands. The tendency is to spread upwards into the para-tracheal glands, but glands become less and less infected as the distance from the primary lesion increases.

Blacklock never found any retrograde spread into the lungs from the glands. For example, he says when there was an upper lobe infection with extensive involvement of tracheo-bronchial glands he never found involvement of broncho-pulmonary glands in the lower lobe on the same side. When the broncho-pulmonary glands on both sides were affected primary foci were found in both lungs.

The youngest age at which a primary lesion was found was seven weeks. It was a tuberculous broncho-pneumonic patch with extensive caseous changes, only slight surrounding reaction and no fibrosis. As age advances there is more tendency to fibrosis. In only 1 out of 58 cases was any fibrosis found

in the first year of life and that was in a child of four months with a caseous lesion in the right lower lobe. Between the ages of seven to thirteen, however, fibrosis was found in 76·9 per cent. of the cases.

Similarly, the tendency for the deposit of lime salts increases as the children grow older; the earliest age at which they were found was six and a half months.

Of 101 cases where the primary lesion was abdominal 88 showed tuberculous mediastinal glands, but no ulceration of bowel, and of these 59 (71·1 per cent.) died of tuberculosis. In the other 24 cases the tuberculosis was found accidentally at post-mortem examination. In 18 of the 101 cases there was ulceration of the bowel. All these were children under five years and all died as a result of the tuberculous lesions.

Of the 173 cases where the primary lesion was in the thorax 168 died of tuberculosis and of the 5 who died from other causes the tuberculous lesion was arrested in 2 only. Cases where the primary lesion was thoracic due to the human type of bacillus showed the greatest tendency to develop a generalised disease, 86 per cent. of them having miliary tuberculosis. The least tendency was in the primary abdominal cases due to the bovine type of bacillus, and in these 40·6 per cent. developed miliary tuberculosis.

As a result of these findings Blacklock came to the conclusion that in Scotland nearly all young children with tuberculosis of the lung or tracheo-bronchial glands died of the disease, but that the tendency to heal increased as age advanced. He is also of the opinion that the adult lesion is due to a new infection contracted in later life and is not a recrudescence of an infantile lesion.

Armand Delille (1) does not take such a serious view of the fate of young children with intrathoracic tuberculosis and records a number of cases where recovery has occurred in spite of extensive disease. He regards artificial pneumothorax as a valuable method of treatment, especially in unilateral ulcerative cases, but bilateral pneumothorax may

also be employed with success even in very young children.

For diagnosis he relies chiefly on the family history, Von Pirquet reaction, radiography and examination of the gastric contents for tubercle bacilli.

Bovine Tuberculosis

Savage (10) stated that in England 6·5 per cent. of raw market milk contains live tubercle bacilli and that 23·3 per cent. of non-respiratory and 1 per cent. of respiratory tuberculosis was due to bovine tubercle bacilli. Forty per cent. cows react to tuberculin, 1 per cent. are infectious and 1 in 500 have tuberculous udders. He calculated that in 1931 deaths from tuberculosis of bovine origin in England and Wales were 1,716 (297 respiratory and 1,419 non-respiratory) and from tuberculosis of human origin 34,102 (29,361 respiratory and 4,741 non-respiratory).

In the bovine cases nearly all deaths occurred in the age period one to five.

Cows have much natural resistance but it lessens with age, so that some veterinary surgeons advocate the destruction of cows over ten years. The cow produces 1,000 to 2,000 gallons of milk each year as against the normal quantity of 150 gallons. It bears a calf a year, and it seems probable that this increase in calf-bearing and over-production of milk predisposes to tuberculosis.

Cumming (11) found that out of 14 cases of pulmonary tuberculosis due to the bovine bacillus, 10 had previously had gross glandular tuberculosis and in another series of 12 cases in which gross glandular tuberculosis was followed by the pulmonary form, 4 were due to the bovine tubercle bacillus. He thinks that air-borne infection may occur in those who work with cattle.

Lange (12) also recognises the possibility of direct air-borne infection from diseased cattle.

Gloyne (13) conducted a series of experiments with

milk and found, after excluding inconclusive results, 128 (9·1 per cent.) samples positive and 1,285 (90·9 per cent.) negative for tubercle bacilli after inoculation into guinea-pigs.

Thirty samples of condensed milk were all negative on guinea-pig inoculation. Of grade A (tuberculin tested) milk 2 (3·8 per cent.) were positive and of 49 grade A (non-tuberculin tested) 5 (10·27 per cent.) were positive.

He also made 28 tests from samples taken from milk churns and found all negative. It would appear that there has been little, if any, reduction in the incidence of tubercle bacilli in milk in England during the past twenty years, but there has been an enormous fall in infantile mortality from tuberculosis. This should not prevent the greatest care from being taken to ensure a clean and pure milk supply, but it does indicate that the problem of tuberculosis in childhood is not simply one of milk.

Griffith (14) found the following percentage of cases to be of bovine origin :—

TABLE 19

Ages :	0–4.	5–14.	All ages.
Pulmonary .	0	0	0·8
Cervical glands .	85·7	48·1	45·7
Lupus . .	57·3	47·1	48·6
Meningitis . .	34·8	31	30·1
Bone and joint .	27·3	18·5	1·8

Blacklock (9) found no case of bovine infection out of 94 autopsies of children with primary pulmonary tuberculosis, but out of 64 autopsies of primary abdominal tuberculosis 54 (81·8 per cent.) were of bovine origin.

The bovine type of disease appears to be no more and no less virulent than the human type. Munro and Walker (15) state that in Scotland 4 per cent. of pulmonary cases are

due to the bovine tubercle bacillus. They describe the case of a child of eleven who died of bovine tuberculosis and at the autopsy there was found widespread slowly growing tuberculosis of the glands of the neck, thorax and abdomen and extensive disease and cavities in the lungs.

They regard the bovine type as equal in virulence to the human type.

There has been much controversy about bovine tuberculosis in recent years and serious attempts to eradicate it are being made. The ideal method, no doubt, would be to destroy all cattle which reacted to tuberculin and to build up tuberculous-free herds. This would take a long time and involve enormous expense. In America 208,000 cattle were slaughtered in 1927 because they were tuberculous.

It has been suggested that reactors might be bought from farmers by the Government and segregated. Milk from these reactors would be pasteurised and used so that the cost would be less than in the wholesale destruction of reactors and yet in time the herds would become tuberculous free.

In Denmark Bang succeeded in getting tuberculous-free herds by isolating reactors. The milk from these reactors was pasteurised and sold. Their calves were separated and fed on milk that was free from tubercle bacilli and if they did not react to tuberculin were allowed to join the non-tuberculous animals. He found that the calf of a tuberculous cow was almost invariably free from tuberculosis at birth and that if kept away from infection it remained free.

Hamil (16) emphasises the importance of pasteurisation by the positive holder method, which he regards as much better than the flask method. Pasteurisation does not materially affect the nutritive value of milk but subsequent boiling does.

By the Milk Order, 1936, special grades of milk are described as follows. Tuberculin Tested Milk is milk from cows which have passed a veterinary examination and a tuberculin test; it is bottled on the farm or elsewhere, and it

may be raw or pasteurised. If it is bottled on the farm, it may be described on the bottle caps or cartons as Tuberculin Tested Milk (Certified). If it is pasteurised it is described as Tuberculin Tested Milk (Pasteurised). It must satisfy certain bacteriological tests.

Accredited milk is raw milk from cows which have passed a veterinary examination; it is bottled on the farm or elsewhere. It must satisfy the same bacteriological tests as raw Tuberculin Tested Milk.

Pasteurised milk is milk which has been retained at a temperature of 145° to 150° F. for at least thirty minutes, and does not contain more than 100,000 bacteria per millilitre.

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CHAPTER VIII

CLASSIFICATION AND TYPES OF DISEASE

THE Turban-Gerhardt classification divided cases into three groups according to the extent of the disease.

1. Disease affecting at most one lobe or two half lobes and of slight severity.

2. More extensive than the first but affecting at most two lobes and of slight severity, or one lobe and of great severity.

3. Cases worse than those in the second group.

Sir Robert Philip modified this by adding the degree of constitutional disturbance to it. He used S or s to represent the systemic change, according to whether it is great or small. If it is out of proportion to the amount of disease in the lung he uses a large S but small l. Thus L1 denotes little lung damage and no systemic disturbance and L3S denotes extensive lung involvement and excessive systemic symptoms.

His classification contains twelve groups.

L 1.	L 1 s.	L 1 S.	1 1 S.
L 2.	L 2 s.	L 2 S.	1 2 S.
L 3.	L 3 s.	L 3 S.	1 3 S.

The classification adopted by the London County Council for administrative purposes is :—

A. Cases in which tubercle bacilli have never been found in the sputum.

B. Cases in which they have at some time been demonstrated.

1. Slight if any constitutional disturbance. No serious complications. Signs must be limited to the upper zone in

unilateral cases and in bilateral ones they should not extend below the clavicle or spine of the scapula.

2. Cases falling between groups 1 and 3.

3. Cases with severe constitutional disturbance, grave complications or extensive physical signs.

It will be understood that once tubercle bacilli have been found in the sputum the case goes into group B, and can never be restored to group A.

The old classification of Laennec recognised three stages :—

1. Consolidation.
2. Softening.
3. Cavities.

Inman's classification :—

(*a*) Resting febrile,
(*b*) Ambulant febrile, resting non-febrile,
(*c*) Ambulant febrile,

is an excellent practical one from the clinical point of view and has the great advantage of simplicity.

The word pretuberculous should in my opinion be dropped from our classification. It has been suggested that a condition predisposing to tuberculosis can be recognised and this has been called the pretuberculous stage. Everyone is pretuberculous until infected with tuberculosis, and individuals who react to tuberculin are strictly speaking tuberculous, whether or not they have symptoms. Those who do not react may be called pretuberculous, but to do so is misleading because it implies that there exists a type which, when infected, is especially liable to develop clinical manifestations of the disease. A delicate child is often called pretuberculous, whereas it is known that the delicate child is just as resistant to infection as his robust brother or sister. The practical importance of this fact is that children who appear healthy should not be left in an infected household in the mistaken belief that because they are healthy they will not contract tuberculosis.

I have recently suggested the following classification as a rough guide to treatment (1) :—

1. Latent or non-clinical pulmonary tuberculosis. The stage of alarm. Here there is evidence of tuberculosis but not of activity. For example, after pleural effusion.

2. Early progressive or the stage of attack because here the body has the upper hand and the disease will probably yield to proper treatment.

There are three sub-divisions :—

(*a*) Acute.
(*b*) Subacute.
(*c*) Chronic.

3. Late progressive or the stage of defence because the disease is now firmly established and the individual cannot reasonably expect a cure, but can defend himself to some extent from the spread of the disease.

4. Chronic fibroid stage or the aftermath. Here the disease is not progressive or only very slowly or intermittently so. The patient suffers from damage, great or small, which has been done during the active stages of the disease.

Another classification is built on the type of the disease.

Acute :—

Acute Miliary. Broncho-pneumonic. Pneumonic.

Chronic :—

Chronic Miliary. Fibrocaseous. Fibroid.

Acute miliary tuberculosis is a general blood infection. Since the lungs are the great filters through which all the blood in the body has to pass they are always involved though they may not be badly infected until the late stages of the disease, so that the predominant symptoms are often non-pulmonary. Death usually occurs from meningitis.

Tuberculous broncho-pneumonia is an acute and fatal condition resulting from a bronchogenic spread. It is not

uncommon after hæmoptysis and is a frequent terminal event in chronic cases with cavities. It is often possible to trace the infection from a cavity through the bronchi into the lobules of the lung.

Acute pneumonic tuberculosis is not common and is usually an allergic reaction around an old pulmonary focus. The condition is inflammatory and a large portion of the lung may be affected, but it may resolve completely without aggravating the original lesion. On the other hand it may lead to activity, so that when the inflammatory zone has resolved an active tuberculous focus remains. In order to prevent this allergic reaction Wingfield (2) suggested desensitising patients by giving regular doses of tuberculin.

Chronic miliary tuberculosis was not recognised until shown by radiography to exist in certain patients in whom the disease was chronic.

Sayé (3) recognises two types :—

1. Chronic miliary tuberculosis of primary infection.
2. Chronic miliary tuberculosis of re-infection.

(*a*) Chronic generalised tuberculosis.
(*b*) Chronic miliary tuberculosis or *granulie froide.*
(*c*) Localised forms of hæmatogenous origin.
(*d*) Non-apparent forms.

1. Chronic miliary tuberculosis following the primary infection usually has an insidious onset. There may be no pyrexia or bouts of fever may be associated with fresh hæmatogenous infection. Metastases are common in bone, joint, spleen and other organs and death usually occurs from meningitis. Occasionally the condition remains localised to the lungs and the outlook is then more hopeful. If recovery occurs radiography may show no lesions in the lungs or there may be multiple calcified foci. (Plates XXV. and XXVI.).

2. Chronic miliary tuberculosis of re-infection.

(*a*) Chronic generalised tuberculosis. This is associated with extra pulmonary lesions and as a rule there are no signs in the lungs until the late stages although radiography may show the characteristic miliary infection.

(*b*) Chronic miliary tuberculosis. Onset may be with mild hæmoptysis or it may be insidious. There is slight toxæmia and often no pyrexia. Radiography shows miliary nodules in both lungs, but there are few if any physical signs. The condition lasts for two or more years. The lesions may remain confined to the lungs and resolution sometimes occurs even in the late stages when cavities have appeared.

(*c*) Localised chronic hæmatogenous tuberculosis generally resembles chronic fibrocaseous tuberculosis with generalisation only at the end. The course of this type of the disease is very chronic.

(*d*) Non-apparent forms of chronic miliary tuberculosis are sometimes found on radiographical examination of the apparently healthy. Sayé examined 1,176 students at Barcelona and found disseminated discrete nodules in the lungs of 5·6 per cent. of them. He thinks they may be merely the remains of a primary infection and of no special significance in regard to the future development of tuberculosis of the lungs.

Maxwell (4) described the case of a girl of twenty-two who began to get tired in June, 1934, and in August developed glands in the neck. Three months later she was found to have a temperature between 100° and 101° F. and to have developed a dry cough. X-ray (Plate XXIII.) of the chest showed typical miliary nodules scattered over both lungs with enlargement of the mediastinal glands. In addition to the enlarged cervical glands, one could be felt in the axilla but there was no sign of softening in any of them. A few crepitations were

occasionally heard at the right apex behind but there were no other physical signs in the lungs. There was very little sputum, but when a specimen was obtained it was found to contain tubercle bacilli in large numbers and they had previously been found in the stools. In February, 1935, she had her tonsils removed after recovering from an attack of acute tonsillitis and after this the temperature, which had previously been relapsing similar to the Pel Ebstein type, remained normal. No evidence of tuberculosis was found in the tonsils. In August, 1935, she had an attack of fibrinous pleurisy, but the temperature settled after a fortnight. Treatment throughout consisted in complete rest in bed followed by sanatorium routine and by February, 1936, all signs and symptoms had disappeared and X-ray (Plate XXIV.) showed that the miliary nodules had completely cleared, and except for a little opacity at both apices the radiographical appearances of the lungs were normal.

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CHAPTER IX

TREATMENT

General

THE principles of treatment may be summarised as follows :—

1. Attention to the general health of the patient and treatment of troublesome symptoms and complications are necessary at all stages.

2. During the acute periods of the disease the object of treatment is to check the spread of the disease and for this purpose rest is essential.

3. When the acute stage is passed the object of treatment is to restore the tone of the body to normal or to its maximum standard of fitness.

4. When the condition of the patient has been restored as far as possible the object of treatment is so to regulate his life that he may live and work within the limits of his impaired capacity.

It is important to distinguish between treatment designed to improve the general health and that designed to check the tuberculous lesion. For example, if an acute tuberculous joint is immobilised the splint is intended to deal with the actual disease, just as in pulmonary cases, to rest the lung by staying in bed, or more completely by pneumothorax, is to treat the disease. On the other hand, fresh air and regular living are intended to improve the general health and so indirectly affect the course of the disease. If a city worker is sent into the country, where he has good food and can lead a quiet regular life in the open air, his general health will improve just as will that of the consumptive under

similar conditions. It is very important to appreciate this, because there is a popular belief that the treatment of pulmonary tuberculosis consists of living out-of-doors and overeating, and a patient is apt to think that no other treatment is required. Fresh air and good food are beneficial and excess of any sort is harmful for everyone. Moderation is the secret of health and those who are already handicapped by some chronic disease like tuberculosis suffer sooner and more severely than normal people if they ignore the ordinary rules of health. It is therefore especially important for the tuberculous to lead a healthy life, but in addition to this they need special treatment for the tuberculosis.

Diet

Otto Walther introduced forced feeding in the treatment of pulmonary tuberculosis, and he certainly obtained some excellent results in his sanatorium in the Black Forest. At the present time it is not customary to advocate nearly such large quantities of food, although the value of a generous diet is well recognised. Fat people are less liable to tuberculosis than are the thin, and in any condition where there is a shortage of food (for example, the Siege of Paris, 1870, and the Great War) the incidence of tuberculosis increases. Vaile (1) points out that many people will eat butter or crisp fat, such as over-cooked bacon, but dislike beef or mutton fat. He has collected a series of cases from which he concludes that it is these latter fats which protect against tuberculosis. In families where several members are tuberculous, it certainly appears that those who eat animal fats are more resistant than the fat-shy. It is undoubtedly a fact that, as a general rule, a gain in weight may be taken as a good sign and a loss is often the first indication of a breakdown. It is a mistake, however, to overfeed a patient, and provided he reaches his normal weight and maintains it no further gain in weight is required.

The diet should consist of good plain food, and as far as possible one should avoid giving special articles of diet which the patient will not be able to obtain when he resumes his normal life. In ordering a diet it is necessary to take into consideration the type of food a patient has been in the habit of taking in the past and will have to go back to in the future.

Fat should be encouraged: mutton fat, bacon, butter, dripping, etc., may be advised, and most patients are able to take fat in some form or other. Milk is a good food, but there are substitutes, and it should not be forced on those with whom it disagrees. It is a mistake to get a patient into such a habit of taking extra milk that he is dependent on it.

Cod-liver oil is widely used and can be obtained in a tasteless form. It contains a fat-soluble vitamin of great potency, is very nutritious and especially useful in chronic cases who are under weight. It is quite unnecessary to use it as a routine, and it has been found at Midhurst Sanatorium that patients who are taking cod-liver oil regularly do no better than those who are not, although in special cases its value is admitted.

The amount of food which a stomach can digest is variable not only in different individuals but in the same one according to his state of health. It is obviously harmful to put into the body a bigger quantity of food than can be assimilated however small that quantity may be. One often finds a patient who begins to improve and gain weight when the quantity of food is reduced, for he assimilates more and loses the chronic dyspepsia produced by over-eating. A weak digestion may be improved by fresh air, cheerful companionship during the meal, the inclusion in the diet of a little appetising food, even if not in itself nutritious, such as smoked salmon, a bitter tonic or, still better, a little alcohol. The question of alcohol and tuberculosis is a difficult one, for the disease is very chronic, and patients are so

liable to periods of depression that the alcohol habit may well be formed. I do not think alcohol should be used in tuberculosis as readily as in certain acute diseases, where its action as a food alone, apart from its other effects, renders it a most valuable drug in helping through a crisis. Apart from the danger of developing a habit, alcohol in moderation is good. It undoubtedly assists digestion, and the addition of a glass of stout or port to the diet will often turn the scale in favour of the patient. Some years ago the routine treatment of pulmonary tuberculosis was rum and milk, and I am told that patients thrived on it. As a general rule, one would say that if patients are doing well without alcohol so much the better because of the real danger of producing a habit, and that spirits should be forbidden except in the case of elderly patients who are accustomed to them. On the other hand, one should never forget that alcohol often does good and it should not be withheld as a matter of routine.

During the acute febrile stage the diet should be as generous as is consistent with the patient's powers of digestion. Milk is often well tolerated but may lead to diarrhœa with offensive stools, in which case a more solid diet should be given and milk stopped.

It is in the subacute or sanatorium stage that diet is chiefly important, for the patient is trying to regain his lost strength and energy. Towards the end of sanatorium treatment, however, extra or fancy diets should be dropped as far as possible so that the patient may not suffer when he returns home and has to live on an ordinary diet. Special diets have been advocated for tuberculosis but their value is doubtful, and although the patient should learn to eat wholesome food and avoid excess it is usually a mistake to encourage fads about foods. Some diets are designed to supply calcium and assist in calcification, or to make up for a calcium deficiency which has been noticed in tuberculosis. It has been suggested that a salt-free diet should be given as salts tend to produce an excess of fluid in the body.

Gerson, of Bielefeld, Westphalia, advocates a salt-free diet. A substance consisting of potassium, calcium and magnesium, and which he calls " mineralogen," is substituted for sodium chloride. Tuberculosis involves exudation and sodium salts are excluded because they tend to hold up liquid in the tissues. In the general wasting of tuberculosis there is a loss of inorganic salts, and these are given to make up the wastage, in addition to which some have a definite diuretic action. Gerson also gives fresh uncooked food with a high vitamin content, and 2 oz. of phosphorated cod-liver oil daily. Carbohydrates are restricted, and there is no overfeeding. The proportion of food is protein 90 grm., fat 160 grm., carbohydrate 220 grm., in a diet of 3,000 calories. It is suggested that carbohydrate favours the spread of tuberculosis, whilst protein lessens the sensitivity and fat raises the resistance.

Strauss (2) thinks the value of the diet is solely due to the limitation of sodium salts. Professor Sauerbruch has employed this treatment extensively for cases of bone and joint tuberculosis and lupus. Special kitchens have been made in some German hospitals in order that the food may be properly prepared and salt avoided in the cooking. There are, however, two practical objections to it. In the first place it is very expensive, and secondly, impairment of the appetite is common amongst the tuberculous. Many patients have to be tempted with dainties, and would find it impossible to take such a diet.

Mayer and Kugelmass (3) describe the result of treating 20 patients for six months at Saranac with a salt-free diet rich in fats and vitamins, but low in protein and carbohydrates. They were all cases of advanced pulmonary tuberculosis, and were losing ground under ordinary sanatorium treatment. After six months, 8 had gained weight, 10 had less sputum, though tubercle bacilli were still present, 4 became apyrexial, and 8 showed fewer physical signs. Two developed pyrexia during the treatment.

Insulin has been used in certain cases when the patient is under-nourished in spite of increased diet. An ounce of glucose, best taken in the form of barley sugar which contains 85 per cent. glucose, is given with meals and from 5 to 10 units of insulin are injected afterwards three times a day. Banyai and Jurgens (4) describe forty-three patients treated in this way. It caused a gain in weight in 54·1 per cent. of the moderately advanced and in 47·3 per cent. of the advanced cases. They consider it safer to begin with 5 units three times a day after ordinary meals.

It is necessary to stimulate the appetite in certain patients, especially those with advanced disease. In these cases there is usually an accumulation of mucus in the stomach, and 30 gr. of sodium bicarbonate in half a tumbler of hot water taken the last thing at night and the first thing in the morning will often give relief. As there is a deficiency of acid in most cases, a tonic such as

Liq. Strych. Hyd. . . .	℥iij.
Glycerin.	ʒss.
Ac. Nit. Hydroch. dil. . .	℥xx.
Inf. Gent. Co. ad . . .	ℨss.

should be given before meals.

Nausea is often due to cough which begins as soon as the patient starts to eat. For this reason it is wise to make him cough up as much sputum as possible as soon as he wakes. There is usually no difficulty in doing this, and a bout of coughing can be brought on by stooping down or by change of posture. If the cough is hard and dry, a teaspoonful of eupnine or lemon and hot water taken before breakfast may loosen the phlegm.

Loss of appetite will often be improved by change of environment. If, therefore, a patient is moved from one sanatorium to another, his appetite is likely to improve, even if the food is not so good. Many will do better on inferior food well served than on the very best if it is not

made to look tempting. The arrangement of the table or tray, flowers, the quality of the plates, the way the napkin is folded, etc., are details which go to make a big difference.

Congenial friends or a cheerful nurse with pleasing conversation will often stimulate an appetite. We all know how much nicer dinner is if our horse has won the big race. It will be seen, therefore, that alteration of the actual diet is not the sole, nor indeed is it the most important, way to deal with patients who are difficult to feed.

Of course the food must be made to suit the patient. The addition of something with a strong taste will often help the appetite. For example, anchovy paste, pickles, onions added to the salad, etc. Patients who can whip up an appetite in this way are helped by a little alcohol with or before meals. Raw meat sandwiches are very often taken by such patients, who often get to like them, and find they increase the appetite. Ice cream may be accepted when no other foods tempt the patient at all.

It is not always the most delicate dish that appeals to the patient. Many who will turn from the most daintily cooked omelette will eat with relish kippers or tripe.

Whilst it is necessary to tempt those who are very ill and to advise a special diet under certain circumstances, it cannot be too strongly emphasised that for the patient who is doing well, ordinary food is best. Overfeeding is bad, and the patient should be encouraged to get used to the type of food that those in his walk of life are accustomed to take.

Rest

If one had to rely on one method only in the treatment of tuberculosis that method would have to be rest, for it is so vastly more important than all the other treatments. By resting the diseased part the lymph flow is impeded, there is a tendency to fibrosis and the disease becomes localised. These changes are seen in tuberculosis of any part of the

body. The immediate improvement in a tuberculous joint when a splint has been applied, or in acute pulmonary tuberculosis as soon as the diseased lung has been collapsed, is very striking. In any case of acute pulmonary tuberculosis an initial period of three or four months in bed is advisable. Before going to Frimley Sanatorium patients are admitted to Brompton Hospital, and I have found that those who have a long preliminary course of rest in the hospital do much better than those who go straight on to the sanatorium.

Jaquerod (5) insists on the necessity of a long period of rest in order to obtain a natural cure. He points out that artificial pneumothorax, thoracoplasty and phrenic evulsion are merely methods of giving more rest to the diseased lung, and are often unnecessary provided a sufficient period of rest in bed is given.

Even when a practical cure has been obtained, the importance of taking life quietly should not be forgotten, especially in these modern days. Dr. Jaquerod believes that the motor bicycle with its vibration is specially liable to reactivate a tuberculous lesion.

Wingfield (6) describes seven stages of rest :—

1. No physical or mental exertion allowed, the patient being treated like one with typhoid fever.

2. Patient allowed to feed himself and talk, but still no reading, writing or visitors.

3. Talking, reading and visitors allowed in short and increasing periods. Still uses bed-pan and is washed.

4. More latitude in reading, writing and having visitors. Allowed to wash face and hands himself and to sit upright for meals.

5. Allowed to sit in a lounge chair for half an hour once a day, and gradually longer. Allowed to use bedside stool.

6. As above, but allowed to walk to lavatory.

7. Allowed to wash out of bed and go to the bath, but to stay in bed for the rest of the day.

If all goes well the patient may reach the last stage in three months, and then begin a period of gentle training.

As improvement takes place the patient requires a certain amount of exercise, and success of treatment is largely a matter of correct balance between rest and exercise. This is discussed in Chapter XI. dealing with sanatorium treatment.

Fresh Air

The value of fresh air is discussed in the same chapter. Although it is important at all stages of the disease, it should be regarded as part of the general treatment and not as a method peculiar to tuberculosis. Broadly speaking, one may regard the value of fresh air as twofold :—

1. Those who work in the open air are, on the whole, more healthy than those who have indoor employment, provided they are physically fit to do their work.

2. Those who lead an open-air life (*i.e.*, have windows always open and avoid stuffy atmospheres) are less likely to contract catarrhal conditions partly because they have become accustomed to cold, partly because they do not meet concentrated infection and no doubt partly because they tend to be in better general health than those whose lives are spent largely in stuffy rooms. A few hours in an overheated or crowded place of amusement is not bad for the consumptive and much harm is done psychologically by forbidding the patient to go to an entertainment. It is entirely a matter of common sense, and the principle should be to encourage the patient to lead a healthy life, for which an abundance of fresh air is necessary. But a certain number of people are unable to stand cold and are harmed by a fresh-air life.

Treatment of Symptoms

Cough. In many cases this is largely due to habit and patients can easily be trained not to cough. In the dining-

room of any large sanatorium, where a number of consumptives are gathered together, a cough is seldom heard. Paroxysms of cough, which are due to difficulty in bringing up sputum and are especially liable to occur in the mornings, are often relieved by a simple mixture such as :—

Sod. Bicarb.	gr. xv.
Sod. Chlorid.	gr. v.
Sp. Chlorof.	℥x.
Aq. Anisi. ad	℥ss.

in an equal part of hot water. Patients with chronic disease who have tried many remedies often ask for this, as they get more relief from it than from any of the more complicated mixtures.

Other mixtures which help to loosen the sputum and relieve cough are :—

Ammon. Chlorid. . . .	gr. xx.
Ext. Glyc. Liq.	℥i.
Glycerin.	℥xx.
Aq. ad.	℥ss.

or,

Pot. Iod.	gr. iii.
Pot. Bicarb. . . .	gr. xv.
Aq. Camph. ad . . .	℥ss.

In the case of continuous cough which interrupts sleep a linctus should be given, such as :—

Oxymell. Scillæ . . .	℥ss.
Ac. Hydrocyan. dil. . . .	℥ii.
Morph. Acet.	gr. $\frac{1}{16}$.
Aq. ad	℥i.

or, if this fails, heroin will often give relief, and the following is a useful linctus :—

Heroin Hydroch. . . .	gr. $\frac{1}{16}$.
Glycerin	℥x.
Syr. Picis Liq. ad. . . .	℥i.

It often happens that a patient gets accustomed to a linctus and it is then best to change it for a time. The following prescriptions may be found useful :—

Bromoform	℥$\frac{1}{2}$.
Terpin Hydrate. . . .	gr. i.
Alcohol (90 per cent.) . .	q.s.
Heroin Hydroch. . . .	gr. $\frac{1}{40}$.
Tinct. Pruni Virg. . . .	℥iii.
Glycerin ad	℥i.

or,

Nepenthe	℥x.
Oxymell. Scillæ . . .	℥xx.
Syr. Pruni Virg. ad . . .	℥i.

For the useless, irritating cough, which does not produce sputum, a lozenge will often be sufficient. The Brompton Cough Lozenge (Trochisci Glycyrrhizæ, each containing Extract of Liquorice, gr. iii., and Anise Oil, ℥$\frac{1}{2}$) is efficacious, and the fact that patients who have tried many other lozenges frequently write for it shows that the patients themselves believe in its value. Cough is often caused by smoking, but under these conditions many patients prefer the cough.

Sputum. Special treatment, such as artificial pneumothorax and sanocrysin, may be indicated if there is excessive sputum, and the quantity of sputum is often reduced if the patient gives up smoking. If there is sputum, however, it should be expectorated, and it is a mistake to give drugs such as opiates to suppress it. A quantity of sputum suggests breaking down lung tissue or cavitation, and treatment must be directed to the cause.

Night Sweats. These occur in the early acute stages or

during an acute exacerbation in chronic disease. As the patient improves they almost always disappear, and it is uncommon to find them after he has had a few days' rest in bed. In some cases, however, they persist and may be so severe that the patient has to change his night clothes two or three times during the night. In such cases the patient should be given a tumbler of milk just before he goes to sleep, and if he wakes in the night he should take a little more milk, a few biscuits or some other food. This usually prevents night sweating, but in bad cases it may be necessary to have a special nurse to wake the patient periodically and give him a little warm milk. Omnopon, gr. $\frac{1}{3}$, given the last thing at night, will sometimes allow the patient to sleep without sweating. A common remedy is a pill, such as zinc oxide gr. ii., Extract. Belladonna Sicc. gr. $\frac{1}{4}$, but in my experience this has only occasionally relieved the sweating. In many cases the patient has too many bedclothes or too warm a room.

Pyrexia. The best treatment for fever is rest. If rest in bed is not sufficient some further rest of the diseased part by artificial pneumothorax or strapping the chest wall may succeed. It is easy to reduce the temperature by drugs, but to do so is useless except for the mental effect on the patient who worries about his temperature, for it will rise again as soon as the drug is discontinued. Cryogenin gr. v., or if this fails gr. x. given after lunch, will usually reduce the evening temperature, which, however, rises again as soon as the drug is stopped if the activity continues.

Dyspnœa. This may be due to the extent of the pulmonary lesion or to the cardiac condition. In some cases when there is extensive fibrosis and the heart is much displaced relief may follow phrenic evulsion. In this case it is probably due to the fact that during inspiration a large part of the lung is unable to expand and the force of the negative pressure affects the thoracic organs, including the heart which dilates. In some unilateral cases of fibrosis or obstruc-

tion to a large bronchus the heart does appear to dilate during inspiration. When therefore the diaphragm is paralysed it can rise during inspiration, thus saving the heart and relieving the dyspnœa. Ephedrine sometimes gives relief in chronic fibroid cases, but it is apt to produce palpitations or other cardiac symptoms. Inhalations of oxygen will also help to relieve the symptoms, but treatment is almost entirely a matter of rest.

Gastro-intestinal Symptoms. These may be due to overfeeding and always necessitate a review of the patient's diet. Poor appetite and a weak digestion are very common and sometimes the initial symptoms of pulmonary tuberculosis. In a series of cases in which I had fractional test-meals done I found a deficiency of hydrochloric acid in the stomach in the majority, and I have obtained benefit by giving before meals a mixture containing :—

Liq. Strych. Hyd. . . .	℥iii.
Glycerin	ʒss.
Ae. Nit. Hydroch. dil. . .	℥xx.
Inf. Gent. Co. ad . . .	ℨss.

In most cases, however, the gastric symptoms disappear as the patient improves, and an abundance of fresh air is the best stimulant to the appetite. Diarrhœa may be due to toxæmia without any ulceration of the intestine, or there may be actual ulceration, in which case there will be blood in the stools. In some cases, especially in the terminal phases of the disease, the stools become very offensive and loose, and this is usually due to the milk diet which is so commonly ordered in these cases. If the milk is altogether stopped and a solid diet substituted the stools will cease to be offensive, even in the worst cases, and the diarrhœa is usually considerably relieved. I have found no benefit from dimol, hyd. c. cret. or other intestinal disinfectants in these cases, nor have I obtained any results from the use of calcium chloride given intravenously.

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CHAPTER X

TREATMENT—*Contd.*

Medicinal

PREPARATIONS of gold have been used for many years in the treatment of pulmonary tuberculosis, and sanocrysin is the form most commonly employed at present. Sanocrysin is a double thiosulphate of gold and sodium which was prepared by Moëllgaard, and was believed to destroy tubercle bacilli *in vivo*. It is given intravenously, and is frequently followed by a severe reaction resembling that seen after an injection of tuberculin. At first it was thought that sanocrysin killed a large number of tubercle bacilli, and that reaction was due to the endotoxins which were consequently set free. This view was supported by the undoubted fact that tubercle bacilli did disappear or become very scanty in the sputum after injections of sanocrysin. An antitoxic serum was prepared and given before sanocrysin in order to counteract the reaction, but it proved of very doubtful value, and has now fallen into disuse. In 1890 Koch discovered that a preparation of gold cyanide had a bactericidal action on tubercle bacilli, but it was subsequently shown that, although a dilution of 1 in 1,000,000 prevented the growth of the bacilli in culture, it required a dilution of 1 in 25,000 to prevent their growth in serum. In the same way the action of sanocrysin on tubercle bacilli is affected by serum. Sweany and Wasick (1) tried the effect of sanocrysin on tubercle bacilli that had been kept in cultures for four years. There was blackening of the bacilli after about twelve days in all dilutions up to 1 in 20,000. There was complete inhibition of growth in dilutions up

to 1 in 200,000, partial inhibition up to 1 in 500,000, and slight inhibition up to 1 in 1,000,000. But in a more recent strain of bacilli some colonies grew in dilutions of 1 in 20,000. After tubercle bacilli had been exposed for two hours to sanocrysin as strong as 1 in 1,000 in salt solution and serum, they grew on Petroff's medium quite well, and even after sixty days' exposure to 1 in 2,000 sanocrysin the culture injected into guinea-pigs produced tuberculosis. Wright (2) found sanocrysin 1 in 250 had no effect on tubercle bacilli in blood. Sanocrysin may produce shock by its poisonous action on the endothelium, but not by killing tubercle bacilli and producing a tuberculin reaction from the endotoxins.

Dead tubercle bacilli produce similar lesions to those produced by living ones and Atkin (3) conducted experiments to show the effect of sanocrysin on these lesions. In this way the complicating factor of the bacilli multiplying and producing further lesions was eliminated. Atkin inoculated rabbits with a dose of dead tubercle bacilli sufficient to produce lesions and a state of allergy which led to a fatal reaction following the intravenous injection of old tuberculin. He found in one experiment that 5 out of 7 rabbits protected by sanocrysin but only 1 out of 8 controls survived the tuberculin reaction. In another experiment he found all 6 rabbits protected by sanocrysin survived but 4 out of 5 of the controls died within 24 hours of the tuberculin reaction. The intensity of the allergic reaction increased with the severity of the lesion produced by the tuberculous toxin. Those rabbits treated by sanocrysin had much smaller histological lesions in the liver than had the controls. Atkin came to the conclusion that sanocrysin counteracted the chemical changes caused by tuberculin toxins so that the size of the lesion was reduced and consequently the intensity of allergy lessened.

At first when sanocrysin was used there were some disastrous results. About 16 per cent. died as a direct result of

the treatment either from shock or acute metal poisoning. Antitoxic serum was used because it was then believed that shock was due to setting free endotoxins from killed bacilli. It was not successful, however, and its use has now been abandoned altogether by most physicians. As a matter of fact, the serum did not prevent genuine tuberculin shock. By decreasing the doses and lengthening the intervals between them these severe reactions were prevented. Professor Faber (4) advised dosage of 0·5 grm. intravenously for the first dose, and 1 grm. for subsequent doses, with intervals of three days between the first two doses and five days between the subsequent doses. If a reaction occurred he stopped the treatment until all signs of the reaction had gone. Smaller doses are now frequently used, but personally I have seen no benefit from very small doses, which in my experience are as likely as larger ones to produce reactions. If a reaction occurs it is my practice to wait until all effects of it have passed, and then to repeat the same dose. I do not increase the dose if the previous injection has caused any reaction. In the case of a well-built patient with good general condition and no pyrexia I gradually increase the dosage up to 1 grm., which I repeat three or four times. My initial dose is 0·1 grm.; after three days I give 0·25 grm., and then at weekly intervals 0·5, 0·75 and 1 grm. A course, therefore, would last about six or seven weeks, and consist of 5·6 grm. or 6·6 grm. of sanocrysin. With this system of dosage I have obtained the best results and have had no serious complications. It is, however, too large for routine use and in delicate patients I start with 0·1 grm. and increase more gradually to a maximum of 0·25 or 0·5.

Some authors claim good results from very small doses, such as 0·05 grm., 0·01 or even less.

In patients who are acutely ill, a small dose may produce reactions, for sanocrysin is specific for tuberculosis in the sense that it produces an effect on a tuberculous patient with

a dose that has no effect whatever on a healthy person. In this way it resembles tuberculin, but in both cases minute doses are in my experience useless.

In some acute cases I have given daily intravenous injections of 0·1 grm. sanocrysin for a week, and then after a week's interval repeated the course. In most cases this has had no effect for good or bad, and in the few where improvement has occurred I have not convinced myself that this was due to the sanocrysin rather than to the rest in bed.

Sayé (5) varies the dose according to the weight and general condition of the patient but generally gets to a dose as big as 0·8 grm., and in some cases the total given in a course was as much as 20 grm.

Mayer (6) advocates the more extensive use of gold salts and thinks crisalbine the best form. He uses distilled water as a solvent unless there are reactions or the patient is febrile, when he substitutes 10 c.cm. of a 10 per cent. solution of calcium gluconate (gluconyl). He starts with a preliminary dose of 0·1 grm. followed a week later by 0·15 grm. If there is no reaction he gives 0·25 grm. in 5 c.cm. distilled water intravenously at weekly intervals for four months. Then if the patient is doing well he continues the treatment for another four months when dosage is reconsidered. If there have been reactions he substitutes gluconyl for distilled water.

If, after four months, the patient has not improved or has become worse but has not had reactions he increases the dose to 0·5 grm. and uses gluconyl as solvent. Failing improvement in eight months he abandons the treatment.

In febrile cases he advocates gluconyl as solvent, and failing improvement he increases the dose and may even reach 1 grm.

He thinks it better to give a long course of many months rather than a series of treatment with rest periods and he advises an X-ray examination every two months to check the progress of the disease and guide treatment. Most of

his patients attend the Laennec Hospital as out-patients for their injections and he treats some 300 weekly. Of a series of 404 he classifies 184 as much improved or cured, 131 as improved and 139 as unaffected.

He has treated pregnant women with benefit and the children have all been born fit.

In my series of sixty I divided the cases into five groups (7). All these patients had tubercle bacilli in the sputum before treatment.

1. Chronic fibro-caseous. This group contained 28 patients, and of these 19 improved, 8 remained unchanged, and 1 appeared worse as a result of the treatment. Fifteen lost tubercle bacilli from the sputum.

2. Cases where artificial pneumothorax had been induced for disease in one lung, and after an initial improvement relapse had occurred owing to spread of disease in the other lung. In this group were 10 patients; of these 9 improved and 1 became worse. Five lost tubercle bacilli from the sputum.

3. Cases with active tuberculosis in both lungs. The worse lung was collapsed by artificial pneumothorax, and sanocrysin was given for the other lung. In this group were 6 patients, of whom 5 improved and 1 remained unchanged. Four lost tubercle bacilli from the sputum.

4. Acute pulmonary tuberculosis treated by sanocrysin without pneumothorax. There were 13 cases in this group. Five of them improved, and 2 became worse. In 6 the course of the disease showed no change. Only 1 lost tubercle bacilli from the sputum.

5. A case of spontaneous pneumothorax and 2 of pleural effusion, with active tuberculosis in the non-compressed lungs. In 2 of these there was no change, and 1 became worse.

Clarke (8) has written a full account of the uses and dangers of sanocrysin and described the result of treatment in 18 cases. In common with other observers, he noticed that the most

striking effect was diminution in the quantity of sputum and the number of tubercle bacilli found in it. Sputum ceased altogether in 8 of his cases, was diminished in 4, and in the rest there was no change. He wrote :—" Generally one or two tubercle bacilli could be found after prolonged search as long as any sputum was coughed up. On the other hand, there is no doubt that the number of bacilli tends to diminish very markedly. Two patients who ceased to have sputum for two months relapsed. When they began to produce sputum again tubercle bacilli in fair numbers were present."

Clarke also noticed that in some of his cases there was an eosinophilia during the treatment, but it subsided after two or three months. In one there was an eosinophil count of 84 per cent. In many of his cases there was a fall in the Arneth count after an injection of sanocrysin, but in favourable cases the count rose after 24 to 48 hours, and remained high for some months. Those who did not show this fluctuation in the Arneth count did badly.

The sedimentation test is affected by sanocrysin. Trail (9) mentioned eleven cases treated with sanocrysin because they were not doing well and still had tubercle bacilli in the sputum in spite of four months' treatment. The initial result of this treatment was a rise in sedimentation rate followed in satisfactory cases by a fall, which was as much as 20 per cent. in one case. Six of the eleven cases became T.B. negative. Heaf (10) found a similar initial improvement in the sedimentation rate, but in over 75 per cent. the rate increased again when the treatment was stopped. Houghton (11) found that the eosinophils often increased after using sanocrysin especially if the patient developed a rash. He applied his blood index (see p. 50) to patients undergoing sanocrysin treatment and came to the conclusion that those with a low index (under 100) did not improve, but the higher the index the greater the chance of benefit from sanocrysin.

The same fluctuation is seen in the vital capacity. After a sanocrysin reaction the capacity falls, but, if the patient is doing well under the treatment, it gradually rises and reaches a higher level than previously. The improvement after sanocrysin is often only temporary, and when a relapse occurs the vital capacity will gradually fall as the disease spreads.

Indications. It often happens that a patient improves under sanatorium or other treatment, and loses all symptoms except sputum containing tubercle bacilli. In these cases sanocrysin is especially useful, and will frequently cause the sputum to disappear after two or three injections, although relapses are common. In one case a man of fifty-five had chronic fibroid tuberculosis of seven years' duration, and there was X-ray evidence of cavities in both lungs. He was not febrile, and his general condition was good, but the sputum was loaded with tubercle bacilli. After the second injection of sanocrysin the sputum was much lessened in quantity, and tubercle bacilli were found with difficulty. After the fourth injection no bacilli were found, but otherwise his condition was quite unchanged. Two months later the sputum again contained numerous tubercle bacilli.

Patients with chronic pulmonary tuberculosis often have periods of comparative good health, alternating with periods when the disease progresses. Sanocrysin does not seem to prevent these relapses, but it does seem, in many cases, to check an exacerbation of disease and bring about a period of arrest. After an initial period of improvement many of my patients relapsed, but in some this was controlled by a second course of sanocrysin. An acute exacerbation in a chronic case is often checked by sanocrysin, but my results in the primary acute cases have been extremely disappointing.

In many patients with chronic fibro-caseous tuberculosis, the disease slowly progresses, and in these sanocrysin may be useful in checking the spread of the disease.

I have found sanocrysin helpful in combination with pneumothorax in the treatment of bilateral disease. It is

often impossible to obtain an efficient bilateral pneumothorax, and in such cases it is better to collapse one lung fully and give sanocrysin to control the disease in the other. Out of eleven cases of this type eight improved and showed no spread of disease in the uncollapsed lungs.

The report to the Joint Tuberculosis Council (12) concludes that the administration of gold does aid pneumothorax treatment.

The chief indications for treatment, therefore, may be grouped under four headings :—

1. To diminish the quantity of sputum and number of tubercle bacilli.
2. To check an acute spread of disease.
3. To treat a patient who is getting gradually worse under other treatment.
4. To treat bilateral disease in combination with artificial pneumothorax.

Complications. In my series of sixty cases the following complications were observed during the treatment :

1. Febrile reactions. These can be divided into three groups :

First, a sudden rise of temperature, starting an hour or less after the injection and lasting for a few hours. There is always a feeling of malaise during the reaction, and sometimes a rigor. This short reaction occurred in seventeen of my cases.

Secondly, a rise of temperature which may start equally suddenly, but which may persist for a few days, is more severe than the first type of reaction, and is accompanied by malaise, headache and sometimes vomiting. This type of reaction was seen in thirteen cases in my series.

Thirdly, a long reaction which occurred in four of my patients. The temperature did not begin to rise until after the third injection of sanocrysin. The rise

was gradual, and lasted for five days or more. In one case it lasted seventeen days, accompanied by a slight amount of albuminuria, but with little or no malaise.

2. Albuminuria. A very faint trace of albumin was found in most patients, but in only twenty-three was there any definite amount. There is a greater tendency for albuminuria to occur as the treatment continues, and this is one of the reasons why I do not give more than 6·6 grm. at any one course. After this quantity persistent albuminuria may begin. In most of my cases the condition was very mild and transient, and only in one case did I have to discontinue the treatment on this account. I should advise stopping after 5·6 grm. had been given in cases where more than the merest trace of albumin was found in the urine. In cases where the urea concentration test was made this was always found to be within normal limits in spite of the albuminuria.

3. Gastro-intestinal symptoms. Vomiting occurred directly after the injection in thirteen of my series, and was associated with a febrile reaction.

Looseness of the bowels was not uncommon, and a troublesome diarrhœa developed in two cases, but passed off in a day or two when the treatment was discontinued. Two patients had a mild stomatitis and eight complained of a metallic taste.

4. Aching in the limbs and rheumatic pains were present in seven cases. In one the joint pains lasted for several days, and were so severe that the sanocrysin injections were stopped.

5. An erythematous rash occurred in eleven of my series, and in three of these it was severe. In all cases where a rash appears I now stop the treatment. In one case a mild rash developed, and a week after it had subsided I gave another injection of 0·1 grm. sanocrysin, which was followed by a severe eczematous rash. The bad cases went on to a

desquamative dermatitis, and, although in the end all did well as regards their pulmonary condition, the complication was by far the most serious in my series, and for this reason I should not advise continuing the treatment or starting another course in a patient who had developed a rash after a previous injection. The rash may be relieved by daily injections of contramine gr. ii. in 1 c.cm. of sterile water. In severe cases potassium permanganate baths give relief.

6. Chrysiasis is a grey discoloration of the skin which occurs in patches as a result of gold administration. It becomes worse when the part is exposed to the light.

Sayé (5) regards small rises of temperature, transient albuminuria or erythema as focal reactions. Such complications as dermatitis, persistent albuminuria or gastro-enteritis as metallic poisoning. By decreasing the dose or lengthening the intervals between them he finds it is usually possible to overcome these complications.

Cruden (13) in a series of 50 cases noted pyrexial reactions in 10, rash in 10, stomatitis in 6, malaise or metallic taste in 6, rheumatism in 6, gastro-intestinal symptoms in 3, albuminuria in 2, jaundice, syncope and purpura in 1 each.

Conclusions. In all my cases the patients were having artificial pneumothorax, rest in bed or some other form of treatment, and it is impossible to give figures which would be of value in forming an opinion as to the exact results of sanocrysin treatment. If two patients treated by sanocrysin improved considerably and eventually the sputum cleared up altogether, but whereas one remained well the other relapsed after a few months, it would not necessarily mean that sanocrysin had succeeded in one case and failed in the other. In one case the defensive forces of the body were not able to take advantage of the check in the disease caused by the sanocrysin and make a permanent arrest, but the actual effect of the drug may have been exactly the same in both cases. Relapses are very common after this treatment, and in the majority of my patients the loss

of sputum was only temporary. There can be no question, however, that sanocrysin does have a very distinct immediate effect on many cases. I have come to the following conclusions on the subject :—

1. Patients with chronic pulmonary tuberculosis who are failing to improve or becoming slightly worse under simple routine treatment do, in many cases, show a remarkable improvement as soon as they are given sanocrysin, and the most striking change is the diminution in the quantity of sputum and tubercle bacilli. In most cases of chronic pulmonary tuberculosis there are periods of quiescence and activity. Sanocrysin seems to cut short that period of activity and to precipitate one of quiescence or arrest, but it does not prevent the liability to relapse.

2. In acute cases sanocrysin may aggravate the condition and if used at all should be given in very small doses and then with very little hope of success.

3. Sanocrysin should be regarded as a drug which, in certain cases, aids other methods of treatment. It is not a cure, and is never more than part of the treatment.

4. The dangers and complications of sanocrysin treatment are not great. Some patients are susceptible to the drug and react to small doses. If they can stand a small dose they are not likely to react to a moderate one. Doses up to 1 grm. are safe provided a full week is left between each injection. It was with still larger doses given at frequent intervals that serious forms of nephritis or enteritis occurred when sanocrysin was first introduced.

5. Sanocrysin does not act by killing the tubercle bacilli *in vivo*. It probably has two actions, one which occurs only when sufficient has been given to produce a reaction or shock, and this is comparable to the action of other substances, such as T.A.B., or serum. The other, which is not connected with the ordinary result of shock, consists in stimulating certain parts of the body to destroy the bacilli. How this result is produced is not known, but sanocrysin does

certainly differ from other substances so far employed in that when given intravenously to a consumptive patient the tubercle bacilli tend to disappear from the sputum in a very short space of time.

There are other preparations of gold now on the market and crisalbine in my experience gives results in every way comparable to those obtained from sanocrysin. Allocrysin is being used chiefly in Switzerland at present, and has the advantage that it can be given intramuscularly, but I have not seen the same results as with sanocrysin or crisalbine.

Solganal is another substitute, but I have found it less satisfactory than sanocrysin. Saegler (14) describes its use. The action of another preparation known as triphal is described by Melion (15).

Oily suspensions of gold such as oleo-sanocrysin or solganol B. have been advocated as being less toxic. It is said that the gold is more slowly absorbed and so produces a prolonged action. Giraud (16), however, has recorded cases where toxic reactions followed their use. In my experience they have no advantage over the ordinary preparations in aqueous solution.

Other metals have been used in the treatment of pulmonary tuberculosis besides gold. The intravenous injection of copper produced results similar to sanocrysin, and certainly did good in some cases, but the reactions were too severe, and it has fallen into disuse. Moxey (17) describes the results of 850 patients treated by antimony, but, as with arsenic, there is no direct effect on the tuberculosis. Arsenic has a tonic effect, and sodium cacodylate is an old remedy. Liquor arsenicalis, however, is of equal value as a tonic.

Walburn (28) has investigated the effect of certain heavy metals on animals suffering from various infections. He has experimented with barium, aluminium, cerium, selenium, cadmium, molybdium, ruthenium, lanthium, platinum and eridium. This long list is given to show how thorough the work on the heavy metals has been. Of them all, apart

from gold, cadmium appears to be the most likely to be of use. Heaf (29) has made valuable experiments on the use of cadmium and after trying various salts found that cadmium sulphide 1 per cent. emulsion in sterile olive oil was the best form in which to give it. He has described 27 cases with the following results :—

Clinically quiescent	4
Improved . .	17
Stationary . .	2
Worse . .	4

In 13 out of the 27 cases tubercle bacilli disappeared from the sputum. He concludes that heavy metals have a definite place in the treatment of tuberculosis, that cadmium in the form of the sulphide or glycine is worthy of further consideration and that cadmium is not only cheaper but probably equal in effect to gold.

Serum. Antituberculous serum has been used in the attempt to counteract tuberculous toxæmia, but has had no success. Various preparations of serum have been tried by different workers, but, although some have reported encouraging results, there is no real evidence that any particular serum has produced definite benefit, and the use of serum in tuberculosis has fallen into disuse.

As with tuberculin, if the dose of serum is increased to the extent that it produces shock, benefit may follow. I have, however, obtained the same result with normal horse serum.

Fig. 13 shows a case treated with antituberculous serum. The patient, a girl of twenty-one, had an acute exacerbation of pulmonary tuberculosis, and had six subcutaneous injections of the serum in 10-c.cm. doses without effect. When 40 c.cm. were injected she had a reaction, followed by a lowering of the temperature. After a second dose of 40 c.cm. a similar reaction occurred, and a week later it was decided to give her a third dose of 40 c.cm. On this occasion,

however, when 20 c.cm. had been injected she suddenly collapsed, stopped breathing, was pulseless, lost control of her bladder and rectum, became livid and appeared to be dead. She recovered, however, and from that time steadily

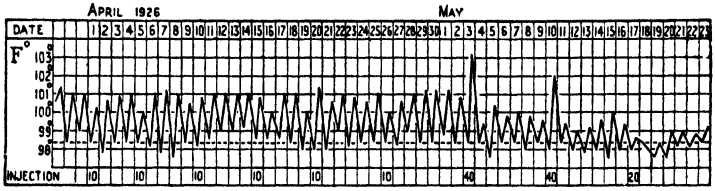

FIG. 13. Temperature chart in a case of pulmonary tuberculosis treated by injections of serum. The figures at the bottom of the chart show the number of c.cm. of serum given on the various days.

improved. The temperature became normal, the pulse fell from 120 to 80, tubercle bacilli disappeared from the sputum, and six months later she was in a sanatorium walking five miles a day.

In this case I believe the improvement was due to the shock, and not to any specific action of the serum.

Tuberculin. When tuberculin was first introduced there was a wave of enthusiasm, and it was regarded as a cure. The results were disappointing, however, and it appeared to make matters worse in many cases. Opinion began to swing to the other extreme, and tuberculin was looked upon not only as useless, but as actually dangerous. Since that time it has practically fallen into disuse, except by a few disciples, some of whom are lukewarm and others ardent in its praises. It is, however, a fact that after nearly fifty years the believers have failed to convince the world that tuberculin has any real value in treatment, and even in veterinary work it has not established its position except for diagnosis. The arguments in favour of tuberculin may be summed up :—

1. Whatever may be the theories or results in animal

experiments, tuberculin has been found to do good to cases of pulmonary tuberculosis in man.

2. Patients with incipient pulmonary tuberculosis treated with tuberculin fail to develop the disease, and a strong hypersensitiveness is corrected, so that after the treatment the patients fail to react even to large doses of tuberculin.

3. Some patients who have failed to improve under sanatorium or other treatment do improve with tuberculin.

On the other hand, it is stated :—

1. The great majority of investigators have failed to observe any real benefit from tuberculin. Tuberculosis tends to cure itself, and a certain proportion of patients do get well without any treatment at all. Most observers have failed to convince themselves that this proportion is increased by the use of tuberculin.

2. Patients who are hypersensitive to tuberculin do not necessarily develop clinical pulmonary tuberculosis. The incipient type of case is just the type that does well under any treatment.

3. Some patients certainly do improve with tuberculin, but others do not. It is possible to find a patient who has improved under any one treatment, though several other treatments have failed. Pulmonary tuberculosis is a disease that ebbs and flows. There are patients who failed to improve with tuberculin, but became rapidly better when it was stopped, and some other treatment started. When a patient with acute febrile tuberculosis goes to bed the temperature usually settles down quickly. If it does not do so the addition of tuberculin to the treatment does not cause it to fall. After a tuberculin reaction there is a tendency for the temperature to fall, but the same is seen after a reaction following non-specific protein such as T.A.B., or normal horse serum. In other words, the beneficial effect of certain treatment such as rest in bed, is plain, whereas the benefit of tuberculin is still a matter of debate.

I have seen many cases of tuberculin reaction, and in my

experience the effects soon wear off, leaving no damage even after a severe reaction. Sometimes a serious and even fatal flare-up of the disease may be initiated by tuberculin, but the chances of this are remote, and it should not occur if the tuberculin is properly used. Any drug is dangerous in the hands of those who have no knowledge of its effects. I have seen good effects follow a reaction, and shall refer to this matter under Shock Therapy.

There are four substances which may be obtained from tubercle bacilli.

1. A protein derivative which will produce skin reactions in tuberculous patients.

2. A phospholipin which is an antigen and forms immune substances.

3. A saturated fatty acid which stimulates connective tissue cells to produce monocytes and tubercles.

4. A polysaccharide which will kill a tuberculous animal.

Halliday Sutherland (18) says the ideal tuberculin might well consist of the first three fractions. The protein fraction is used for diagnosis and Purified Protein Derivative may now be obtained in tablets and used for the intracutaneous test. It does not contain any extraneous proteins.

There are many tuberculins on the market.

O.T. Old tuberculin is an extract of the growth of human tubercle bacilli boiled down to one-tenth its volume and filtered. It contains 50 per cent. of glycerin.

P.T. is prepared in the same way from bovine bacilli.

T.R. with human and P.T.R. with bovine tubercle bacilli, are extracts of the insoluble constituents of the bacilli themselves. Each c.cm. contains 2 mg. solid substance, being the residuum of 10 mg. tubercle bacilli. It is twenty times stronger than O.T. or T.A.F.

B.E. is an emulsion of the entire human and P.B.E. of the bovine tubercle bacilli. Each c.cm. contains 5 mg. of pulverised tubercle bacilli. It is two and a half times stronger than T.R. and fifty times stronger than O.T. or T.A.F.

T.A.F. or albumose-free tuberculin is similar to O.T. but is grown in a culture fluid of citrates, inorganic salts and asparagin and contains no albumoses or peptones.

B.C.G., Bacille Calmette Guerin, is made by culturing and subculturing bovine tubercle bacilli for many years so that a living but non-virulent culture is obtained.

P.P.D. is Purified Protein Derivative.

Halliday Sutherland says that the most suitable case for tuberculin treatment is the early non-febrile type. This is just the type that is regarded as most suitable for sanatorium treatment, but he claims that tuberculin treatment gives better results and at much less cost. The patient is often able to carry on his ordinary occupation throughout the course of treatment. He suggests that the sanatorium should be used for febrile patients needing rest and then graduated exercise until they have become suitable cases for tuberculin treatment at home.

He thinks that if tubercle bacilli are not found in the sputum, diagnosis can be confirmed by the subcutaneous test which has been described on p. 47. He uses T.A.F. for the test and suggests that the initial dose of B.E. which he uses for treatment should be 100 times smaller than the dose of T.A.F. to which the patient reacted. Thus if the patient reacted to 0·25 c.cm. of dilution 4 (*i.e.*, 1 in 10,000) T.A.F., treatment should begin with 0·25 c.cm. D_6 B.E. The dose is doubled every three or four days until a reaction occurs. Thus if the first dose were 0·25 D_6 the next would be 0·5 D_6 followed by 0·1, 0·2, 0·4, 0·8 D_5, and 0·16 D_4, and so on. When a reaction occurs he repeats the same dose until there is no reaction and then increases more gradually, adding a half, quarter, fifth or tenth part of the preceding dose to the next one according to how much the patient can stand without reaction. The highest rate of geometrical progression which does not cause reactions should be used. The larger the dose the longer should be the intervals between the injections, and he thinks it is a useful rule not to give an injection until

the subcutaneous lump produced by the previous dose has entirely disappeared. With pure B.E. the interval may be 8–12 weeks. The largest amount of tuberculin that a patient can tolerate without reaction varies for each individual. If he can be immunised to 2 c.cm. B.E. a high degree of immunity is obtained; many cannot go beyond 0·2 c.cm. B.E. Treatment should continue until symptoms have gone and the patient should be retested annually.

Wingfield (19) advocates immunising patients after sanatorium treatment, not to treat existing disease but to prevent allergic focal reactions which he thinks are liable to stir up activity.

Dilutions should always be freshly prepared because, although undiluted tuberculin retains its potency for four or more years, a dilution of 1 in 10 loses some in a week and 1 in 1,000 loses 40 per cent. in 10 weeks. Halliday Sutherland describes six types of febrile reaction which may follow tuberculin:—

1. Immediate reaction and fall by crisis. If under 102° F. repeat the same dose until there is no reaction. If over 102° F. reduce the next dose.

2. Immediate reaction and fall by lysis. If under 102° F. halve the next dose. If over 102° F. give a tenth for the next dose.

3. Delayed reaction and fall by crisis. If under 102° F. the next dose should be a fifth and if over 102° F. a tenth.

4. Delayed reaction and fall by lysis. The next dose should be a tenth.

5. Progressive reaction and fall by crisis. This indicates a focal reaction and the next dose should be a hundredth.

6. Progressive reaction and fall by lysis suggests tuberculous broncho-pneumonia and should not occur as a result of tuberculin.

Gillespie (20) has treated a large number of patients and his results with tuberculin are very much better than those when some other method was employed.

Shock Therapy. When tuberculin or live tubercle bacilli are injected into a sensitive animal there is an allergic reaction. A reaction may also occur when certain other substances are injected; for example, non-specific vaccine (such as T.A.B.), copper, gold, normal horse serum, etc. (21). These reactions are similar to anaphylaxis. In the case of tuberculin it requires but a small dose to produce a reaction in those who are sensitive to it, just as a specific protein will produce a reaction in a patient (with asthma, for example) sensitive to it. A tuberculin or allergic reaction is specific because it is not obtained in those who are non-sensitive, but the reaction does produce a shock very similar to that produced by copper or other substances, and very similar to anaphylaxis. I must emphasise that I do not deny that tuberculin has a specific action, and I do not suggest that shock or reaction are the only effects of its administration. My suggestion is that the shock by itself does have some effect on the disease. I have already described the case of a girl (p. 178) who had anaphylaxis whilst antituberculous serum was being given. The following cases will show under what various conditions a shock may occur and prove beneficial:—

Case 1. A man of twenty-two developed signs of pulmonary tuberculosis early in 1921, and at the end of the year went into a sanatorium. He then had signs of disease all over the right lung, sputum loaded with tubercle bacilli, and an evening temperature of 102° F. Right artificial pneumothorax was started in December, 1921, but he remained ill and febrile until May, 1922, when he had a sudden reaction with increase of fever, and pleural effusion developed.

The sudden nature of this reaction and the fact that the intrapleural pressure went at once from — 6 to + 8 suggested that a perforation of the lung had taken place. Within a month not only did the effects of the reaction wear off, but the temperature became normal, his whole condition gradually improved and he left the sanatorium free from symptoms

in May, 1923. The pneumothorax was kept up until May, 1924, and since then he has kept perfectly well. He is still free from symptoms, and leading a normal life.

Case 2. A woman of twenty-six with extensive signs in the right lung, tubercle bacilli in the sputum, temperature 101° F., and signs of toxæmia was being treated by artificial pneumothorax. During the fifth refill, when 300 c.cm. of air had been introduced and the intrathoracic pressure was — 6 — 1, she suddenly became very cyanosed, gasped for breath and lost consciousness. She recovered in half an hour and afterwards began to improve. Fig. 14 shows the

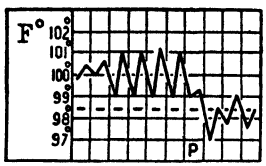

FIG. 14. Temperature chart showing the effect of pleural shock occurring at P.

temperature. In this case the improvement did not last long. She relapsed and died about a year later.

Case 3. A man had a tuberculous sinus from the transverse process of one of the vertebræ. This showed no sign of healing, in spite of prolonged treatment. He was having mild sunlight treatment, and one day, after an exposure to the sun, which was more than double the usual, he had a reaction and high temperature, and felt that he had done himself harm. But the sinus began to heal after this, and has shown no sign of breaking down since.

Similar good results may be seen after a reaction of any origin. I have heard of improvement following the shock of an accident in which a patient broke his leg. May not the reaction which follows too large a dose of tuberculin do good quite apart from any specific action?

Shock, however, is a two-edged weapon, for these reactions

may be extremely dangerous. Fig. 15 shows the temperature of a patient following only 10 c.cm. of serum. He never rallied and died in a few weeks. This is no isolated case, and a reaction, whether specific or not, may lead to disastrous results. For this reason most physicians try to avoid reactions, whatever drug or vaccine they may be using. In some of the cases I have described a violent reaction occurred, and it does not seem to me to be justifiable to cause such a reaction intentionally. The two most dreaded and fatal accidents in pneumothorax treatment are pleural shock and rupture of lung, yet in Case 2 the patient had a definite period of improvement after pleural shock, and in Case 1

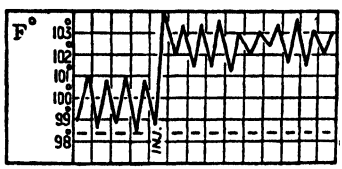

FIG. 15. Chart showing the effect of an intramuscular injection of 10 c.cm. serum.

the patient suddenly began to improve and made an uninterrupted recovery after reaction resulting from rupture of the lung.

It is not only in cases of tuberculosis that improvement may follow shock. Intravenous injections of colloidal gold will produce a reaction, often with a rigor, and bring down the temperature in typhoid fever. Mercurome has been given intravenously in septic conditions and severe cases of pneumonia. Intercurrent disease may be beneficial; thus the asthmatic may be free from asthma during an attack of scarlet fever. May not malarial treatment come under the same category ?

For these reasons when a patient with pulmonary tuberculosis is rapidly or steadily losing ground and other treatment has failed, I think it is good treatment to increase the dose of

sanocrysin, serum, or whatever is being given until a reaction is produced. The most striking case of improvement from this method occurs in young patients of fourteen to eighteen years of age with acute pulmonary tuberculosis. It is, however, a method that must be used with the greatest caution, and one should always start with small doses to avoid a severe reaction in a patient especially sensitive.

Calcium. Calcium has been advocated in the treatment of tuberculosis, especially to control hæmoptysis or diarrhœa. Its effect is distinctly disappointing, as theoretically it should be of great value. Not only does calcification play an important *rôle* in the cure of tuberculous lesions, but there is a calcium deficiency in the blood of tuberculous patients. Moreover, workers in lime are notoriously free from tuberculosis. Half the calcium chloride injected intravenously is excreted in the urine within three hours, and the remainder is excreted within the first three days, so that a calcium deficiency cannot be overcome by periodic injections of calcium chloride. It is possible, however, to overcome the deficiency for a time, and thus tide over a dangerous period, as in a case of hæmoptysis. Prest (22) described a series of cases treated with collosol calcium. He gave 0·5 c.cm. hypodermically at intervals of a week or longer. In a few cases there was some reaction, but no serious complication arose. In many patients he noted distinct improvement, such symptoms of activity as night sweats disappearing readily under the treatment.

Ringer and Minor (28) have described the treatment of intestinal tuberculosis by injections of 5 to 10 c.cm. of a 5 per cent. solution of calcium chloride. They formed the opinion that this treatment gives great relief in cases of tuberculous diarrhœa. When there is actual tuberculous ulceration of the intestine it is difficult to understand how calcium can influence the condition, for post-mortem examinations show that the ulceration remains even after prolonged calcium treatment. However, as these authors point out, a drug

which does good, though we know not how, is of more value than one which fails to relieve, although in theory it should do so.

In my experience calcium given by any method is disappointing in the treatment of diarrhœa due to tuberculous toxæmia and useless if there is actual ulceration of the intestine. Diarrhœa associated with offensive stools in cases of pulmonary tuberculosis can usually be controlled by diet, and if milk is withheld and a more solid diet substituted, it is almost always much relieved if not quite stopped.

Maendl (24) describes the results of treating 250 cases with intravenous injections of calcium, and he notes improvement and disappearance of symptoms of activity. For hæmoptysis he gave 5 c.cm. of a 10 per cent. solution of calcium chloride intravenously, and repeated the injection eight-hourly. The result of this treatment was very satisfactory, and the hæmorrhage sometimes stopped with remarkable rapidity. H. Elving (25) also found calcium chloride a successful hæmostatic. He gave as much as 8 grm. (20 c.cm. of 15 per cent. solution) of calcium chloride intravenously without ill-effect, and found that the coagulation rate of the blood in one case was increased from five and a half to half a minute, and there was always a considerable increase.

I have given weekly subcutaneous injections of 1 c.cm. collosol calcium to thirty sanatorium cases, and did not observe any change which could be attributed to the treatment. Most of the patients improved, as most do in a sanatorium, but it was impossible to say from the symptoms or course of the disease which patient had calcium and which had not. Sandoz calcium, which is calcium gluconate, may be given by the mouth, intramuscularly or intravenously. Large doses such as 10 c.cm. of a 10 per cent. solution are best given intravenously and may stop mild hæmoptysis or clear sputum which has been bloodstained. I have not

found it have any effect on the tuberculous condition either in acute or chronic cases.

Given intravenously I have seen no effect except an occasional slight reaction such as so frequently accompanies other drugs injected intravenously. I have tried daily intravenous doses of 5 c.cm. of a 10 per cent. solution in acute cases without any result. Calcium may be given in the form of ostocalcium tablets each of which contains 7½ gr. of calcium sodium lactate and 500 units of vitamin D.

For hæmoptysis calcium may well be tried. It will not, of course, control a large hæmorrhage, but it may check continuous slight bleeding, and, moreover, it is a perfectly harmless method of treatment.

Cod-liver oil or some preparation rich in vitamin D may with advantage be combined with calcium treatment. Ellman (26) gives a full account of calcium metabolism and parathyroid function.

Iodine. Iodine has been used in the treatment of lung diseases, especially if associated with bronchitis. For tuberculosis the nascent iodine treatment had a vogue some years ago. It consists in giving 10–20 grains of potassium iodide in an ounce of water at 7 a.m. and two hours later an ounce of chlorine water in four ounces of lemonade. The free iodide liberated does not get into the circulation, for in the blood an iodide is formed, so that potassium iodide is probably as good a method as any of giving this drug, and it certainly is useful in cases complicated by bronchitis. I have not found iodine in any way harmful nor have I found any bad results follow the introduction of lipiodol or neo-hydriol into the bronchi in cases of tuberculosis.

Cod-liver Oil. This is an old remedy, and is still considered by some to have a specific effect on tuberculosis. It is undoubtedly of the greatest use in certain cases, but should not be given as a routine. It is rich in vitamins A and D, and recent knowledge of vitamins and the effect of ultra-violet light suggest an explanation of the value of cod-liver

oil in treatment. Concentrated vitamins may now be obtained, and vitamins A and D may be supplied by adexolin or advita. Avoleum is a concentrated preparation of vitamin A. Halibut-liver oil and viosterol can be obtained in capsules of three minims. The preparation, which is known as haliverol, contains irradiated ergosterol with vitamin D of a potency 250 times and vitamin A 60 times that of cod-liver oil.

Sodium Morrhuate. This is a sodium salt of cod-liver oil, which has been advocated in the treatment of pulmonary tuberculosis. It is given by subcutaneous injection in doses of from 0·1 to 1 c.cm. of a 3 per cent. solution. In forty cases treated at the Brompton Hospital with doses of 1 c.cm. twice a week, I found no benefit, and there was no evidence of any more gain in weight than occurred in similar patients not having the treatment. I have frequently given a course of injections to out-patients who were losing weight or doing badly, and have found it without effect.

Jessel (27) treated seventeen male patients at Peel Hall Pulmonary Hospital, and his conclusions were " no evidence that subcutaneous injections of a 3 per cent. solution of sodium morrhuate in doses of 0·1 to 0·9 c.cm. have any material influence upon the course of tuberculosis in the chronic or advanced stages."

Turtle Vaccine. This is a culture prepared from acid-fast baccilli obtained from the lungs of deep sea turtles. Fowler (30) has recorded a series of cases treated with it, but failed to find any benefit from it.

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CHAPTER XI

TREATMENT—*Contd.*

Sanatorium

SANATORIUM treatment consists of many factors of varying importance to the combined action of which results are due. It may be said to have developed out of the " open-air therapy," but it must be clearly understood that fresh air is but a small part of the treatment, and so-called " sanatorium treatment at home " usually fails because it is not possible to produce at home all the factors which are present in a sanatorium. Kingston Fowler (1) said : " The so-called ' open air ' method was first systematised and carried out in a sanatorium, and although the name is not free from objections, I think it is better to call it the ' Sanatorium treatment ' ; we shall at any rate, by so doing, avoid the risk of its being supposed that a patient with tuberculosis of the lungs, who is merely being exposed to the open air, is being treated according to any approved method." It may be said that there are two main factors in sanatorium treatment : First, to teach the patient how to live under his altered circumstances ; and, secondly, to train the body to as high a condition of physical fitness as possible. The factors which go to make up this treatment are rest, exercise, fresh air, diet, routine life with regular habits and treatment of symptoms. Most febrile cases or those with early progressive disease need rest in bed and consequently are not suitable for sanatorium treatment, though they may be treated in a sanatorium. On the other hand, an advanced chronic fibroid case may get a new lease of life from sanatorium treatment. Absolute rest in bed, artificial pneumo-

thorax, thoracoplasty or other special methods of treatment such as sanocrysin are not part of sanatorium treatment although it may be convenient to carry out one or more of them in a sanatorium. There is a large group of patients who get completely cured and, indeed, are best treated by simple sanatorium routine alone, although others, of course, do need the addition of some special method of treatment. This system of treatment cannot be followed out except under constant supervision, and the degree of its success is largely dependent on the personality of the medical superintendent. Nordrach-in-Baden Sanatorium, in the Black Forest, was founded by Otto Walther, a pioneer of sanatorium treatment. It was said of him that he inspired the patients with courage and the desire to live and to work. The same may be said of Trudeau, who started the sanatorium at Saranac Lake. Those who say that sanatorium treatment has failed are ignorant of the state of things before it was introduced. They often fail to understand the type of case for which a sanatorium is intended or are unlucky or unwise in their choice of a sanatorium. The site of a sanatorium is important, but of far greater importance is the medical staff, especially the medical superintendent.

It is impossible to over-rate the value of sanatorium treatment at the proper stage of the disease, and although very few patients want to go into a sanatorium, most of them settle down when they are there. Occasionally one finds a patient whose temperament is such that he finds the routine too irksome and has to leave, but this type of patient almost always does badly. Most of them, when once they have become accustomed to sanatorium ways, prefer to stay as long as their treatment requires many restrictions. To them life as an invalid at home, with brothers and sisters leading a normal life around them, is harder than at a sanatorium with others leading a similarly restricted life. The routine in a modern sanatorium is completely different from that of a few years ago and recent advances in sanatorium

management have made the life of the patient much less irksome.

Wingfield (2) gives three main objectives in sanatorium treatment :—

1. Regaining health or resistance.
2. Consolidating this gain.
3. Education.

He points out that the best results can be obtained only after long treatment, such as nine months or a year, although in more advanced cases, where arrest is out of the question, patching up can be accomplished in half or quarter of this time. Those who have visited Swiss sanatoria must have been impressed with the length of stay in the sanatorium of some of the most successful cases. It is no uncommon thing to find a patient who has been two or more years in the institution. The co-operation of the patient is essential, and Wingfield says that a patient should be encouraged to ask, " How well can I get ? " rather than " How soon can I get home ? " He also emphasises the fact that treatment should be varied according to the patient and should not be rigidly the same for all. " What is grade 5 to an ill-developed, elderly, emphysematous clerk may be only grade 2 to a muscular young policeman."

Rest is an important part of sanatorium life and is taken lying on a bed or couch at stated hours, such as 12 to 1, 2 to 4 and 6 to 7. In most Swiss sanatoria these rest hours are called " cures," and are usually spent on couches on the balcony. The hours of getting up and going to bed, and the exact amount of rest taken during the day, will depend on the condition of the patient. Exercise is also necessary, and one of the most difficult parts of sanatorium treatment is to judge the correct proportion of rest and exercise required to give the best results.

At many sanatoria walking is the only form of exercise permitted, and it is a very good rule to allow no other form

of exercise for at least two years after the disease has become arrested. Patients should never run and should not walk quicker than three miles an hour. Relapse is more likely to occur from over-exercise, especially outdoor games, than from any other cause.

For patients who have to earn their living by manual labour a system of graduated labour may be employed when they have finished the initial period of rest. Marcus Paterson (3) developed this system at Frimley Sanatorium.

It is well known that exercise is followed by a reaction in a tuberculous patient, and Paterson believed that these reactions, if carefully controlled by the physicians ordering the right amount of exercise, were beneficial and that the patient was in effect being treated by his own tuberculin. Whether this is the true explanation or not, the results of the treatment were very good, and many patients left the sanatorium capable of doing a full day's manual labour without harm. At the present time a system of graduated labour is employed at Frimley Sanatorium. The patients at first keep to walking exercise, but when they are sufficiently strong they begin a system of grade work.

Grade 1. Carrying small gardening basket about three miles.

Grade 2. Carrying heavier basket the same distance. Light painting. Potting.

Grade 3. Hoeing and light digging. Using light roller or hand cart.

Grade 4. Digging and trenching with small spades. Cross-cut sawing.

Grade 5. Digging and trenching with full-sized tools. Garden truck work.

No rapid work should be allowed and sudden spurts are harmful. It is most helpful to have an ex-patient as foreman to supervise the working parties, for this gives great encouragement to the patients, who see in the "cured"

foreman an example of the results of treatment. There is undoubtedly more danger in employing graduated labour than merely walking exercises, and the work must be supervised with the greatest care, but amongst the working classes it has many advantages.

Cooper and Dow (4) describe certain exercise tests which were employed at Frimley Sanatorium. They took 100 patients, and those who seemed likely to respond well to treatment and to have a good ultimate prognosis they classed as satisfactory, the others as unsatisfactory. There were forty satisfactory and sixty unsatisfactory in their series. The tests consisted in the effects of exercise (such as a four-mile walk) on pulse, temperature and respiration. They classified the results of these tests as :—

Good. If the pulse-rate fell to within ten beats of the resting pulse-rate within five minutes of stopping exercise and there was no dyspnœa or rise of temperature.

Intermediate. If the pulse-rate fell to within ten beats of the resting pulse-rate within half an hour of stopping exercise and there was no rise of temperature and no marked dyspnœa.

Bad. If the pulse-rate did not fall within ten beats of the resting pulse-rate within half an hour. There may be a rise of temperature and dyspnœa.

The results of these tests were, in the forty satisfactory cases :—

Good, 82·5 per cent. Intermediate, 62·5 per cent. Bad, 5 per cent.

In the sixty unsatisfactory cases the results were :—

Good, 18·3 per cent. Intermediate, 31·6 per cent. Bad, 50 per cent.

Heaf (5) advises three classes of tests :—

1. Walking half a mile in seven and a half minutes.
2. Two miles in half an hour.
3. Four miles in one hour.

The patient should start with the first walk and go on to the second and third if no abnormality is found. He makes the patient rest for fifteen minutes before each walk and takes the pulse and temperature before they start. After the walk the patient lies down and the pulse and temperature are taken every five minutes.

A slight rise of temperature after exercise is not uncommon, but it should fall to normal in ten minutes. Temperature is the most important guide to the patient's capacity for exercise, but other signs, such as loss of weight, increase of symptoms, greater sedimentation rate or falling off of vital capacity, would suggest that a readjustment of treatment should be considered and usually indicate that more rest is required.

Diet. The principles of diet have already been discussed. Wingfield (2) is of opinion that the normal weight in health for an individual need not be exceeded and that all attempts at stuffing or over-fattening should be avoided. None the less, food is an important part of treatment, and one of the functions of sanatorium treatment is to educate the patients to take regular and sufficient, though not excessive, food. To assist digestion the teeth should always be examined and treated if necessary.

Milk is often regarded as essential; an average of 1½ pints per patient daily, ½ pint being taken with porridge for breakfast, ½ pint at the midday meal and ½ pint in the evening may be taken as a typical sanatorium allowance. In addition, milk is generously employed for puddings, custards, etc.

At Frimley less milk is given, and patients are kept as far as possible on a diet which they can afford to continue when they have returned home. Extra milk or other diet is of course ordered in special cases when required, but a patient who can keep fit only under a diet and mode of living which he cannot afford at home will tend to break down on leaving the sanatorium. Frimley is essentially a working-class sanatorium, and its chief object is to restore to the patient as

much capacity for work as possible under conditions which he can maintain at home.

When a patient has lost much weight and wants special aid to regain it, or when the digestion is bad or some other reason is present, extra milk, cod-liver oil, alcohol or tonics may be helpful, or even essential, but one should never forget that the best cases are those who can do without such extras.

Fresh Air. Sanatorium treatment has developed out of the open-air theory of treatment, and fresh air is undoubtedly a very important factor in sanatorium life. It is not a new theory but recent knowledge has led most physicians to regard fresh air as a general tonic and not a specific for tuberculosis. In 1840, George Bodington urged the treatment of tuberculosis with fresh air and a generous diet. Later, Henry MacCormac thought that tuberculosis was caused by air that was rebreathed and could not exist in pure air. He wrote several papers advocating fresh air as the most important method of prevention and treatment of tuberculosis. Even when the tubercle bacillus was discovered a few years before his death he maintained that the bacilli were the result and not the cause of the disease and upheld his view that tuberculosis was the result of contaminated air. When one speaks of fresh air one does not mean air rich in oxygen. There is the same proportion of oxygen in city as in country air. Moreover, oxygen does not inhibit the spread of tuberculosis and some authorities say it is actually harmful in cases of tuberculosis. The benefit of the Swiss air at a high altitude is said to be due to deficiency of oxygen owing to reduced pressure. The benefit of fresh air is probably the cold. Cooling power depends on temperature, moisture and the movement of air. A bracing or stimulating place has a quick cooling power by virtue of cold, dry and breezy air. A relaxing or sedative climate has air that is warm, moist and still. The object of an open-air life is to tone up the body and improve the

general health and not to treat tuberculosis. In most cases it is easy to acquire the fresh-air habit, and it has several advantages, the chief of which are a lessened liability to catch cold and an improved standard of general health. People who work in the open air but spend their evenings or leisure hours in stuffy atmospheres are often robust as a result of their work, but the changes from hot to cold temperatures are harmful, and bronchitis, pneumonia and colds are not uncommon amongst them. However, the comfort of warm rooms is such that most people are prepared to risk the various catarrhal conditions and prefer a few colds to living a fresh-air life. For the consumptive, however, the risk is much greater. A little bronchitis or even a bad cold in the head so frequently leads to renewed activity in the tuberculous lesion that it is essential for the patient to take all precautions against such catarrhal conditions. The patients and staff in a sanatorium very rarely catch cold. A common saying is, "I like fresh air, but cannot stand draughts, they always give me a cold." Perhaps they do, but as it is impossible to enjoy life without meeting an occasional draught, why not get used to them? Of course there are degrees of cold, and it is quite wrong to expose consumptives or anyone to all weathers; but overdressing, overheated rooms and screening off the slightest current of air do tend to make an individual soft and liable to catarrh and it is remarkable how quickly one gets used to cool atmospheres and begins to find stuffy ones oppressive.

It may be said that work in the open air is good for the general health and that, apart from this, a fresh-air life (by which I mean open windows and the avoidance of stuffy atmospheres) is the best way to keep free from cold and other catarrhal conditions. With regard to open-air work, however, it should be remembered that most outdoor jobs mean manual labour and only a few consumptives can stand this. A delicate city clerk who, after leaving the sanatorium, gave up his office work and got employment on a farm would

almost certainly break down with active tuberculosis, whereas if he returned to his office the disease might remain arrested permanently. If one bears in mind that there are people who are not physically fit for outdoor work, one will avoid the mistake of advising it for all consumptives, regardless of their condition, but simply because they are tuberculous. One should also remember that there are people who cannot stand an open-air life. The average young patient will quickly get acclimatised to open windows and draughts and will be all the better for it, but elderly patients, those who are very thin or delicate or have bad circulations, usually have to be protected from cold. It is a big mistake to expose such people to cold whatever the state of their tuberculosis may be, for they will do much better in warm rooms or in a warm climate.

Recreation. It is very important to provide as much recreation as possible, but a relapse is more often the result of outdoor games than of any other cause. All energetic games, such as hockey, tennis, football, cricket, must be forbidden, and even golf should be avoided until the disease is arrested or in the chronic stage. Clock golf, putting, croquet, etc., may be allowed for many patients, and such games may be used in treatment in place of gardening or other forms of graduated labour. With regard to indoor games, each patient should be watched and should not be allowed to play a game which unduly excites him. Chess, played in the evening, may lead to a sleepless night, and I have known a patient's pulse go from 76 to 140 at an exciting moment in a game. In the same way many patients get excited at cards, especially if they are playing for more money than they are prepared to lose. If the patients are watched it is easy to detect any harm that may follow a particular game, but as far as possible the patients should be allowed to choose their own recreations, and all harmless amusements should be encouraged. Never forbid any game or pastime unless it is harmful to the patient's health. He

has quite enough to bear without any unnecessary restrictions.

Routine. A regular life is an essential part of the treatment, and the following may be taken as a typical routine :—

8.30. Breakfast.

9.30—12. Rest, exercise or recreation according to medical orders.

12—1. Rest in recumbent position on balcony.

1.15. Luncheon.

2—4. Rest in recumbent position on balcony.

4.15. Tea.

5—6. Recreation.

6—7. Rest in recumbent position.

7.15. Dinner.

8—9.30. Recreation.

9.30. Bed.

This routine must, of course, be varied according to the seasons and the condition of the patient. For example, some will require more rest and others will be able to dispense with one or more of the rest-hours and go for walks or play games. Smoking should be allowed, for although like everything else, it is harmful in excess, it is a great comfort to many people, and to deprive them of it only adds to their unhappiness and leads to discontent. In some cases it brings on cough and sputum, and it will often be found that sputum disappears when a patient gives up smoking. In such cases, and if there is any laryngitis, it should be stopped, but in the great majority of cases it will be found beneficial rather than harmful.

Sometimes, especially after prolonged sanatorium treatment, a patient becomes hypochondriacal and can think or talk of little else but his temperature and symptoms. This is not unnatural in such a chronic case, but the physician must look out for the first signs of such a condition, as it is apt to leave the patient a mental wreck long after the tuberculosis is arrested. A change of environment or a holiday

away from the sanatorium for a few weeks is often enough to restore the patient's balance, but if he is well enough to work this should be advised as soon as he shows the slightest sign of becoming hypochondriacal. Indeed, it is lack of occupation more than anything that causes such a condition. More consumptives relapse from play than from work, and one can frequently see a great improvement in a patient as soon as he gets settled in some suitable employment.

It is sometimes found that a patient who keeps perfectly well and can do a full day's work in a sanatorium relapses as soon as he leaves and returns to his previous life and occupation. Such patients do very well if they can obtain work in the institution, and in every sanatorium are to be found ex-patients keeping fit and doing hard work as doctors, nurses, clerks, porters, etc. Unfortunately there are not enough sanatorium jobs for all these patients, who form a big group, and it is for this group that an after-care colony is especially useful. Papworth Village Settlement was started chiefly for this type of patient and has now grown into a large community and become a model on which similar settlements both in this country and abroad have been based. It cannot be regarded as an institution for patients and settlers are made to feel the atmosphere of ownership and companionship which exists in a large village or small town. Sir Pendrill Varrier Jones, the medical director, realised that in a large number of cases clinical methods were doomed to failure unless followed by some good system of after-care. He appreciated how important it is to consider the sociological side and avoid over-concentration on the pathological side of this problem of tuberculosis and it is to him that Papworth owes its present position. He (6) states that it is always possible to get plenty of settlers from ex-patients and the only limitation is lack of funds. The principle of after-care here is to accept as settlers patients who have finished their active medical treatment. Unmarried settlers live in hostels built on the

bungalow principle and married ones live in cottages. The children do, of course, meet infection but the experience at Papworth is that they do not acquire the disease. Varrier Jones considers the dominating protective factors to be good housing and good wages. The industries include leather work, trunk making, printing, bookbinding, upholstering, furniture making, etc., and the net sales which were under £4,000 in 1919 were over £106,000 in 1935. Every patient capable of working, even if only for two hours a day, is enabled to do so. The work provided resembles that in the outside world in every possible way, so that there is no impression of wasting time which patients so often feel when given occupation therapy.

In addition to the settlement there is the hospital side of the colony where patients in all stages of tuberculosis are received and even major surgical operative treatment can be obtained in the new surgical block. One building with four paying wards is used as a hospital for men and there is another for women. One building is devoted to cases of non-pulmonary tuberculosis such as glands, bones or joints.

The whole settlement forms a unit, fitted with all modern equipment, including a pathological department where much research work is undertaken. The great importance of Papworth is, however, in its industries rather than its medical or surgical side and it is the greatest scheme yet evolved for dealing with the after-care of tuberculous patients.

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CHAPTER XII

TREATMENT—*Contd.*

Climate and Light

Climate. A change of environment has for centuries been considered beneficial. Celsus (25 B.C.) wrote: (the patient must) "Change his climate, taking care to remove to a grosser one than that he leaves, and therefore from Italy to Alexandria is a very agreeable change."

Morland (1) says that the Romans regarded Egypt as the most suitable place for the consumptive but that the elder Pliny considered the chief benefit was due to the vomiting induced by the voyage. The same view was held by Reid in 1782 and about the same time Erasmus Darwin suggested that a less expensive way of producing the nausea was the rapid rotation of a chair.

Laennec advised the seashore and used to put seaweed under the patients' beds in the hope that the marine aroma would be beneficial. It is curious to note that modern French opinion is exactly opposite and regards residence near the sea as harmful for the tuberculous. A century ago the tropics were advised, then Madeira was fashionable and later Davos became the Mecca of the consumptive. Davos was then a quiet Swiss village where the patient could live peacefully in the cold dry air, now it is a centre for winter sports with the same air but with facilities for exercise and enjoyment beneficial for the healthy but suicidal for the tuberculous patient. Belief in mountain air for the consumptive was held by the ancient Greeks and Indians.

The modern tendency is to regard climate as a matter of

secondary importance in tuberculosis. Paterson (2) wrote "Climate, provided it is not actually unsuitable has little if anything to do with treatment." Gauvain (3) thinks that the English climate is good for the tuberculous by virtue of its variability. Continuous sun treatment is not so beneficial as alternations of light and shade or heat and cold and he thinks that during a hot spell in summer a change to a week or so of dull wet weather is often an advantage. Young (4) agrees with the benefits derived from variability and quotes Ellsworth Huntington's saying that the climate of England comes nearer to the ideal than almost any other place. Morland (1) surveyed the tuberculous mortality in different parts of the world and stated that the highest mortalities, about 655 per 100,000 living are found in Manilla and in Guayaquil. Mortalities between 300 and 500 are found in Athens, Seville, and Lisbon, between 150 and 200 in Paris, Moscow, Dublin and Geneva, about 100 in London and Berlin, 70 in New York and Cape Town and 50 in Sydney. Chile, Finland, Hungary and Iceland have tuberculosis mortalities of over 200 per 100,000, Norway, France and Spain are between 150 and 200, Switzerland, Sweden and Italy 100 to 150. The British Isles, Holland and Denmark have the lowest mortality, being under 100, and these countries border on the North Sea.

The fall in tuberculosis mortality follows the steps that are taken to combat the disease rather than the climate. As Morland remarks, no air in the world can make up for the absence of medical guidance and discipline which a well-conducted sanatorium provides. And yet the popular belief in the value of climate is so strong that one must not disregard its psychological effect and it is generally best to allow a patient to go where he wishes provided he can get proper treatment there and the place he selects is not actually unsuitable for him. The belief may, however, prove harmful as it was in the case of a young man who lived with his mother in a London flat not far from the office in which

he worked. He contracted pulmonary tuberculosis and after treatment, in a sanatorium was told that the disease was arrested. His mother insisted on leaving the flat and taking a house in the country with the result that he had a journey of forty-five minutes to his work. Nothing would make her appreciate the harm of this journey taken every morning and again after the days' work in a crowded train in the heat of summer and cold or fogs of winter and she simply repeated that country air was essential for him. His subsequent breakdown was said to be due to the stuffy office.

It must not be thought, however, that climate has no place in treatment, but the essential factor is the patient's power of response. Metabolism is increased by the low temperature, dryness and movement of air which constitutes a bracing climate and benefits those who are strong enough to respond to it. A relaxing climate where the air is moist and still and the temperature higher is more suitable for the delicate or elderly patient or one with poor circulation who feels the cold.

Gordon (5) found that tuberculosis was more common in districts exposed to rain-bearing winds. Parts of Devonshire protected by the Dartmoor hills and again those districts protected by the Exmoor hills from the westerly rain-bearing winds have a tuberculosis death rate considerably lower than that found in neighbouring districts not so protected. Rogers (6) stated that 60 per cent. of the tuberculosis deaths in India occurred during the hot season and that they were increased by rain-bearing winds rather than actual rainfall.

J. A. Miller (7), discussing climate in the treatment of pulmonary tuberculosis, points out that there is no universally ideal climate. Selection of a suitable locality, he says, is " an individual problem for every patient, depending upon his temperament, tastes and individual reaction to environment as well as the character of his disease." He also says that contentment and reasonable comfort are essential, and

that the patient must be able to afford to stay a sufficient time and have the necessary medical supervision.

Moreover, any change of climate involving fatigue of travel is contra-indicated in acute cases with fever or those with hæmoptysis, or in those with advanced disease.

Roughly it may be said that acute cases are better at home in some institution where they can be under medical supervision, and any complication or development in the disease can have the appropriate treatment. There should be good ventilation with an abundance of fresh air, but it is not necessary for the patient to be uncomfortably cold, and some patients, especially those who are thin or have poor circulation, may suffer from too much exposure. In this acute stage it is usually a mistake to send the patient abroad, and he should certainly not be sent to a high altitude.

During the subacute stage there is an advantage in sending the patient to a climate suitable to his powers of response, but there is no need to send him abroad, and, as has already been said, a sanatorium is indicated at this stage. The choice of an English sanatorium should depend on the patient, and, on general principles, the robust type usually does best in a bracing climate, whereas the delicate one may get on better in some warmer place in the South or West of England.

In the chronic or after-care stage the choice of climate is usually a matter of the patient's financial position. Many who have to work get on quite well, even in the big cities, and one must remember that there are certain advantages in living in a city. Light work in the country—for example, secretary to a golf club in a health resort—is ideal; but such jobs are difficult to find, and country work usually entails hard manual labour, which, above all, is bad for this type of patient. A city clerk who is used to a regular sedentary life is far less likely to break down in a city office than if he went to work on a farm in the country, though if he had the offer of transfer to some branch office in the country he would be wise to accept, not so much on account

of the climate but because, as a rule, life is quieter and less of a rush in the country than in a city.

For those who do no work and can live where they like, I should advise gravel soil in a place protected as far as possible from rain-bearing winds. The exact place must depend on the patient individually, and I always advise him to stay there for a few months to see if it suits him before taking a house.

Many patients like to leave home for the winter, and, in England, Falmouth or Torquay are suitable for those with extensive fibrosis, or whose condition is complicated with bronchitis. For the robust patient who can take plenty of walking exercise the East Coast is more suitable, and for the intermediate type of case Bournemouth or the Isle of Wight is to be preferred.

Switzerland. The climate of the Swiss Alps is most suitable for those who have just finished the subacute or sanatorium stage. Provided a patient is well enough to take regular walking exercise and is moderately robust, he may derive great benefit from spending a winter in the Alps. The choice of a Swiss resort or sanatorium is a matter of great importance, as they vary enormously in efficiency and routine, so that a patient should never be sent to one unless it is known to be satisfactory. The best months for these resorts are December to April, and it is a mistake for a patient to return to England before the winter is over. One knows the great benefit which people derive from a winter holiday in the Alps, and, of course, the consumptive gets the same benefit, provided he is strong enough to take advantage of the facilities offered.

The summer season is delightful and I think the habit of spending the winter in Switzerland and the summer at home is wrong. If the patient starts treatment there it is generally wise for him to remain until the disease is arrested.

The reduced pressure of oxygen in the altitudes has a

stimulating effect on the blood-forming tissues so that the blood is quickly regenerated after hæmorrhage. It also produces a rise in serum calcium. This diminished oxygen supply and the excess of ultra-violet light are the two important factors in the Swiss altitudes that cannot be supplied in this country.

Morland says that the temperature falls about 1° F. for every 300 feet ascended and that at Montana which is 5,000 feet he has rarely known the temperature to be above 70° F., though in the Rhone valley some two miles away it is 85° or 90°F. In January, with the thermometer just below freezing point, the cooling power at Basel was 21·2°, whereas at Davos with 12 degrees of frost it was only 13·5°. He says that at sea-level only one quarter of the water lost by the body comes from the lungs; at an altitude of 8,000 feet the evaporation from the lungs and skin is equal. The low barometric pressure also stimulates metabolism and the increased ventilation of the lungs is carried out by deeper rather than by quicker breathing. At 5,000 feet the basal metabolism is increased by 20 per cent.

He thinks that the effect of altitude on the nervous system is important and that nervous people do not stand altitude well. Patients require sleeping draughts at 5,000 feet two or three times more frequently than they do at sea-level.

Patients whose vital capacity has been much reduced by fibrosis do not get on well at altitudes; for most patients an initial period of three to four weeks in bed is necessary to enable them to get accustomed to it.

Some patients are sent out by those who have no knowledge of Swiss conditions and of what type of case is suitable. Many are just told to go to Switzerland and may find themselves in the hands of anyone. There, as here, success of treatment depends on the doctor and it should be impressed on every patient sent to Switzerland that the climate is only part and a very small part of the treatment.

There is undoubtedly more temptation in the average

foreign institution than in an English one, and, although a patient can obey rules and live a suitable life, it is easy for him to live unwisely. The disease is a very long and irksome one, and a number of bored young people living together are apt to get into bad habits, especially if, as so often happens, they feel well in themselves, and believe that because they are in Switzerland and "taking the cure," no other treatment or precaution is necessary. For this reason it is important to choose the institution from personal knowledge, and also to consider the temperament and character of the patient. Another danger is that a patient may get a habit, so that he cannot, or thinks he cannot, winter anywhere except in Switzerland. This does not matter for a few who are well-to-do but for the majority it is impossible. In general terms one may say that :—

1. Acute or advanced cases usually are harmed by treatment in high altitudes.

2. Subacute or sanatorium stage cases get no special advantage, but may do well.

3. Patients whose disease is nearly arrested may get enormous benefit from a winter in Switzerland. It is an excellent method of putting the finishing touches to a cure.

French and Italian Riviera. Patients with pulmonary tuberculosis do not, as a rule, find the Riviera suitable. There are undoubtedly many days of bright warm sunshine, when the patient can spend most of the day out of doors, but it becomes very chilly when the sun goes down, and catarrhal conditions are common. Some patients with chronic disease like the Riviera and have become accustomed to it and in these cases it is usually best to let them go. On the whole, however, it is not a suitable climate for the consumptive.

Egypt and the North Coast of Africa. Here the atmosphere is dry and pure, and there is an abundance of sunshine. Some places, however, are dusty, and there is a great difference between the day and night temperature. There are many

places on the North Coast of Africa which are much preferable to those on the other side of the Mediterranean, but the physician would be well advised to recommend only those places that he knows from personal experience.

Canary Islands. In these islands the climate is as nearly perfect as possible for many cases, and the only disadvantages are that to get to them necessitates a sea voyage, and life there is somewhat dull. This latter reason may not always be a disadvantage.

Lucas (8) has written an interesting description of the islands, and he gives the following table, showing the

TABLE 20

	Oct.	Nov.	Dec.	Jan.	Feb.	March	April.	May.
Mean shade temperature	71·4	67·6	64·7	62·3	63·9	63·0	64·6	66·5
Surface sea temperature	72·4	70·1	67·5	65·9	65·1	65·4	65·9	67·3
Amount of rain in inches .	1·06	1·75	1·57	1·68	0·57	0·79	0·40	0·55
Sunshine in hours . .	189	165	161	168	183	189	190	218

Meteorological records, Las Palmas, Grand Canary.

meteorological records from Las Palmas (Grand Canary) during the months of October to May.

It will be seen how very equable is the climate. The variations between the day and night temperature is never more than 10° F. At Orotava (Teneriffe) the rainfall is slightly higher and the sunshine less than at Las Palmas, and this is said to be due to the elaborate system of irrigation in connection with the banana industry, which requires a generous supply. At Las Palmas is a British hospital with private rooms available for visitors, and staffed by English nurses. There is also a good social club, a lawn tennis club and an eighteen-hole golf course.

Distant Health Resorts. There are many excellent places in Australia, New Zealand, S. Africa, America, etc., but none of them present any special advantages which cannot be obtained nearer home and all of them have bad as well as good seasons. Some of the country round Bloemfontein, for example, is ideal during certain months, but during others is either a desert of dust or a bed of mud. To visit such places during the proper season may be good, but to take a farm and settle down is usually unwise. Again, many distant places that sound attractive are suitable only for those who can do hard manual work, and are quite impossible for the tuberculous patient who has to earn his living.

Sea Voyages. For a patient in fair general health who wishes to avoid the English winter a sea voyage to S. America, S. Africa, Australia, or some such trip south, may be a very healthy way of spending the winter. The good food, the rest, and the opportunity for fresh air and sunshine are all satisfactory.

In the treatment of active tuberculosis, however, a sea voyage is almost always a mistake, and more likely to do harm than good, quite apart from the fact that the patient may not be allowed on the ship or to land at certain ports of call.

Before advising a patient to go abroad, it is important to find out the laws as to the reception of consumptives in that particular country, or he may be refused permission to land. As these rules vary from time to time, inquiry should be made through the Colonial or Foreign Office in each case.

Although people who, through illness or age, are liable to become a public charge on the country are refused admission, they are often accepted if they have private means or can show that friends or relatives will support them. In most cases provided the patient can support himself, he will be allowed to have treatment or reside, but he should be warned of the possibility of being sent home, and careful inquiries should be made as to the laws and customs of the country he wishes to visit.

In Jamaica consumptives are allowed to enter only if they are visiting the island for the purpose of treatment and have the means to maintain themselves. In Northern and Southern Rhodesia a permit may be obtained by a tuberculous patient. In some colonies and countries the laws prohibit the landing of persons suffering from contagious, communicable or dangerous diseases, and in others permission to land is refused if the Medical Officer of Health certifies that for medical reasons permission should not be granted.

In conclusion it may be said that climate is of secondary importance in the great majority of cases of pulmonary tuberculosis. People get accustomed to their own home conditions, and it is usually a mistake to send them for long periods to a totally different climate if, after treatment, they have to live at home again.

The ideal spot for a consumptive to live might be some country district where there are no extremes of heat or cold in summer and winter, where there is protection from rain-bearing winds and the house is on gravel soil. But even under such conditions it is doubtful whether the patient would have a longer expectation of life than one who chose his home to suit his own happiness, his business and social convenience.

For the consumptive the important question is, " How shall I live ? " not " Where shall I live ? "

Phototherapy. Crocket (9), discussing ultra-violet ray treatment in tuberculosis, says that rays with therapeutic properties may be divided into four classes, all electro-magnetic and differing only in the length of wave :—

1. Those from radio-active substances, such as radium. Here the rays are only from 0·015 to 0·15 Angström units. There are three kinds, called alpha, beta and gamma. The alpha rays do not penetrate, the beta have slight, and the gamma considerable penetrating power.

2. Röntgen rays range from 0·15 to 12 Angström units, and have many gamma rays with great penetration.

3. The so-called ultra-violet rays measure from 2,100 to 3,800 units. They lie between the luminous spectrum and the area of Schumann and Lyman. Strictly speaking, of course, all rays beyond the violet in the visible spectrum are ultra-violet, which therefore include X-rays and radium.

4. Rays of the visible spectrum. These are from 3,800 to 7,600 units. The violet end of the spectrum has considerable chemical but little heating action, and the red end has great heating but no chemical powers.

The sun has ultra-violet rays, but none shorter than 2,900 Angström units, as the shorter ones are filtered out by the smoke and moisture in the atmosphere. The purer the air, therefore, the more ultra-violet rays are present, so that the pure rarefied air of a mountain may contain twice the amount of ultra-violet rays as the atmosphere of a valley. The short rays do not penetrate as much as the longer ones, but they are more irritating and bactericidal and are filtered out before the longer rays. Water and snow reflect the rays, and increase their power considerably, and for this reason people get bronzed so readily at the seaside. It is not the warmth of the sun, but the chemical action of the violet rays which produces sunburn. The pigment protects against the ultra-violet rays, so that when well sunburnt one can stand much exposure to the sun without reaction.

The effect of the rays is also increased by the movement of the air, and hence they are not so effective if given artificially in a room where the air is still and warm. Hill (10) emphasises the importance of cool air passing over the skin if it is desired to get any therapeutic effect from sunlight or artificial rays. With actual sunlight there are other rays which assist the therapeutic action of the ultra-violet ones.

Crocket (9) describes the following direct effects of ultra-violet therapy :—

1. Vasodilatation and œdema of the soft tissues exposed to the light. He points out that this relieves congestion in internal organs, and no doubt accounts for the improvement

in the condition of the skin and the growth of hair and nails, which are so often noted in those exposed to ultra-violet rays.

2. Sterilisation of superficial tissues. The short ultra-violet rays are strongly bactericidal, and as these are filtered out by impurities in the atmosphere before the long ones, the rays of the sun have much more bactericidal effect at high altitudes than at low ones, especially low altitudes where there are much smoke, moisture and other impurities in the air.

3. Relief of pain and comfort. This is especially noted if there is an excess of the short, and therefore more irritating, rays, and consequently for this purpose also treatment at a high altitude will be more effective than at a low one.

4. Pigmentation. He describes two forms of skin reaction, an immediate transitory reddening which disappears half an hour after exposure, and an erythema which appears from six to ten hours after exposure, and which may go on to severe desquamation and blistering if the exposure has been too long. This "sunburn" is produced only by rays below 3,200 units. He says that although this pigmentation is protective, it changes a white reflecting surface into a dark light-absorbing one, and therefore favours the absorption of rays.

The rays also have an indirect effect, and are said to affect not only the general health, but also to cause alteration in the blood chemistry.

Spence (11) made an investigation of twelve adult tuberculous patients; of these eight had pulmonary disease with tubercle bacilli in the sputum. Irradiation was given by a Hanan mercury vapour lamp of 40 units power. It was found that the blood calcium and phosphate were not affected by the light. The blood cholesterol, however, was raised, the greatest rise being seen in the patients who showed the greatest improvement clinically. In one case after ten exposures the patient had a febrile reaction and

vomited. His blood was examined, and no change was found in the calcium or phosphorus, but the cholesterol had fallen from 160 to 140 mg. per 100 c.cm. of serum, and the irradiations were discontinued.

Crocket (9) has noted a definite improvement in the general health and mentality of the patient, and also a reduction of blood pressure when this was high. He has seen a fall of as much as 20 mm. Hg in the systolic pressure as a result of light treatment.

In certain cases of surgical tuberculosis treatment by light is undoubtedly of great value. Much depends, of course, on the way it is given and on the experience of the doctor in charge, but, provided it is properly administered, one will frequently see rapid improvement of certain conditions which are not amenable to other treatment, for example, tuberculous ulceration of the fauces.

In pulmonary tuberculosis, however, the value of light treatment is a matter of doubt. Some deny that it has any value at all, and certainly its value, if any, is not great. It may, moreover, do harm if carelessly employed. Many patients with tuberculosis of the lungs get on better in winter than summer, and any great improvement noted when fine weather comes is more likely to be the result of removing the feeling of depression which so often goes with gloomy weather than to any effect of the ultra-violet rays.

Direct exposure to sunlight or ultra-violet rays will, if overdone, produce a shock reaction in cases of pulmonary tuberculosis, and, although this may be harmful, it may actually be beneficial, and, as with shock from other causes (tuberculin, serum, vaccine, etc.), the patient's condition and temperature may rapidly improve after recovering from the initial reaction. The reaction following over-exposure to sun is very similar to that produced by tuberculin.

Gosse and Erwin (12) regard sunbathing as dangerous for patients with pulmonary tuberculosis and conclude that it should not be done after hæmoptysis or by those who are

overtired or have lost weight or by those who get a temperature of over 99° F. afterwards.

Mathieu (13) devotes a chapter to the effects of sunlight on pulmonary cases. He quotes the opinions of doctors of Leysin who have had experience of the treatment, and also the views given at the conference at Leysin in 1910. The general opinion was that no better results were obtained by light therapy than by the ordinary methods of treatment, but that properly employed it was a safe procedure. Malgat obtained good results at Nice, and it was generally noted that the general well-being of the patients was improved. Rollier (14) found that patients having pulmonary tuberculosis complicating some surgical lesion did better than those with uncomplicated pulmonary tuberculosis. He saw a patient with recurrent hæmoptysis who was cured by sun treatment tried as a last resort. Many physicians consider light treatment contra-indicated in pulmonary cases, and Mathieu quotes Jaquerod, of Leysin, as describing the case of a patient who had never had hæmoptysis until he went for a sun cure to the Mediterranean coast. Jaquerod said that light treatment did not appear to modify the results obtained by ordinary means, and was not without danger. De Reynier, at the same meeting, said that although he had never seen any improvement in the local condition as shown by auscultation, and had not seen diminution of sputum or loss of tubercle bacilli, at the same time he had never met with hæmoptysis, febrile reaction or increase of symptoms, and thought further trial should be given to the treatment.

Burnand referred to the similarity which existed between the reactions following over-exposure to the sun and those resulting from tuberculin injections. He admitted that the general stimulant and tonic effect of heliotherapy was unquestionable, but thought that its focal effect was less certain. Its action depends largely on the state of the lung, and in cases of great activity Burnand considered it

unwise to start a form of treatment capable of causing rapid exaggeration of congestion already excessive.

Crocket (9) gives the following summary of his results from seventy-two cases of pulmonary tuberculosis treated with light treatment :—

1. The general condition and the mental outlook were improved in practically every case.

2. The lung lesions showed fewer indications of activity.

3. The cough was relieved in 92 per cent. of the cases. The sputum was much reduced or ceased in 54 per cent. of the cases and in 20 per cent. was unaffected.

4. Wheezing, often indicative of congestion, is frequently removed by light treatment.

5. The appetite was usually improved. Of the cases with anorexia or dyspepsia 76 per cent. received definite benefit. It is not advisable to give light treatment for one hour before meals, as patients usually complain that it interferes with their ability to enjoy their food.

6. Weight was not benefited as often as expected. Thirty per cent. of the cases did not increase in weight.

7. Sleep was often greatly improved, though in nine cases it was apparently affected unfavourably.

He says that of these seventy-two cases improvement occurred in 85 per cent., whereas, apart from light treatment, 67 per cent. of the patients in the same institution improved.

Hæmoptysis occurred in four cases in the series, but Crocket thinks that it would have occurred apart from the treatment, and certainly it does not seem unduly frequent. In ten cases which had been running an intermittent temperature for several months, this subsided during the treatment. He gives the following contra-indications.

1. Cases of progressive disease.

2. Cases in which destruction of tissue is marked and progressive.

3. Cases with obvious cachexia and amyloid disease, or other toxic manifestations.

4. Cases of sepsis with pyrexia.
5. Cases of hyperthyroidism.

Trail found that at Midhurst the sedimentation rate improved as the patient's condition improved, but there was no change in the sedimentation rate resulting from light therapy.

Most of those who use sun or artificial light treatment try to avoid reactions. When a reaction does occur, its result is very similar to that following tuberculin, serum, etc. Apart from the effect of such a reaction or shock it may be said that light treatment in pulmonary tuberculosis probably has no direct action either for good or bad. It has, however, two indirect effects which may be of considerable benefit to the patient :—

1. Light, and especially a bright sunny day, has a stimulating and cheering effect on every one, including the consumptive patient.

2. The mental effect of feeling something definite is being done, and of seeing the skin gradually becoming pigmented, added to the improved appetite and feeling of well-being which usually goes with bright weather may assist the patient's recovery to a very great extent.

The method by which light acts is not clearly understood. Some think that the action is specific, and it produces an auto-tuberculinisation. This view is supported by the undoubted fact that an over-exposure to the sun produces a reaction which is very similar to that following the use of tuberculin, but one must not forget that the same result can be obtained by the intravenous injection of T.A.B. or other non-specific substances. Rollier suggests that the pigment transforms the short-wave rays into the longer and more penetrating ones.

If light treatment is employed in pulmonary tuberculosis the actual sun is better than artificial light, for the other rays besides the ultra-violet found in sunlight, combined with the fresh air, play a large part in the effect. For

artificial light treatment the tungsten arc lamp, or the quartz mercury vapour, are best for ultra-violet rays, the tungsten arc being especially rich in short-wave rays; the carbon arc has, in addition to the violet rays, a large proportion from the red end of the spectrum.

In order to avoid reactions it is best to start with a short exposure of a limb, and if this produces no reaction the length of exposure and amount of the body exposed is gradually increased day by day. When the patient is thoroughly bronzed it is possible for him to remain exposed to the direct rays of the sun for hours without getting a reaction.

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CHAPTER XIII

TREATMENT—*Contd.*

Artificial Pneumothorax

MANY methods of treatment and so-called cures of pulmonary tuberculosis have been advocated, but very few have stood the test of time. Artificial pneumothorax was suggested on theoretical grounds more than a century ago and now is employed all over the civilised world. It is generally recognised by those with experience of tuberculosis that the basis of treatment is rest of the diseased part. The spongy consistency and continual movement of the lung make it easy for tuberculosis to spread, and it is not surprising therefore to see the rapid improvement which so often follows the induction of a pneumothorax.

The principle of the treatment is to relieve tension, produce lymph stasis and to rest the lung, thus preventing spread of the disease and allowing nature to effect a cure. The lobes of the lungs collapse independently in the absence of adhesions so that on the right side one can see by X-ray three, and on the left two lobes clearly separated after pneumothorax has been induced (Plate XXVIII.). As with every form of treatment there are enthusiasts who regard pneumothorax as the whole of the treatment, advocate it on any and every occasion, and think no other treatment necessary; on the other hand there are those who think it is of very little value and rarely, if ever, recommend it. If one appreciates the purpose of the treatment it is possible to avoid either of these extreme views and to find in artificial pneumothorax one of the most valuable and effective

weapons against pulmonary tuberculosis, and at the same time to recognise its limitations.

The mild case does well and the advanced one badly with any form of treatment. It is in the intermediate stage, where there is early exudative disease with tubercle bacilli in the sputum and physical signs of infiltration and often of cavitation, that one sees such dramatic results from artificial pneumothorax especially if only one lung is involved.

Briefly, one may say that artificial pneumothorax is indicated in cases of acute proliferative disease, whereas patients with good resistance as shown by the presence of fibrosis are more suitable for thoracoplasty if some form of collapse treatment is required.

I do not agree with those who advocate inducing a pneumothorax in the very early stages of the disease before physical signs have appeared, unless of course there is a special indication for it. Most of these cases do quite well with simple medical treatment and one must remember that there are certain complications associated with artificial pneumothorax. At the same time the treatment is more often started too late than too early and it should not be withheld too long.

When the disease has progressed to that stage when crepitations are to be heard it is no longer early and pneumothorax is usually indicated. One should bear in mind that the longer it is delayed the greater the chance of finding adhesions which prevent a satisfactory collapse so that the patient becomes exposed to a subsequent thoracoplastic operation. When in doubt, therefore, it is almost always safer to err on the side of inducing a pneumothorax too early than too late.

Heaf (1) admits the value of artificial pneumothorax, but thinks it should not be lightly undertaken. There are definite dangers and complications, and one should ask oneself " Can this patient recover without any form of interference ? " and not " Can a pneumothorax be induced

in this patient?" He mentions four types which he considers unsuitable.

1. Young adults with no tubercle bacilli in the sputum and no sign or symptom except slight infiltration, or even a small cavity shown by X-ray. This type does well with routine treatment.

2. Young adults with slight unilateral infiltration and tubercle bacilli in the sputum, but no cavity and no pyrexia. Try for three months routine treatment before inducing a pneumothorax.

3. Patients over forty-five with recent infiltration superimposed on an old fibrotic lesion.

4. Patients with a small lesion in the better lung and low resistance as shown by bad sedimentation rate, high monocyte and low lymphocyte count, absence of eosinophils or a bad family history. Pneumothorax in these cases is liable to cause a rapid spread of disease in the better lung.

Selection of Cases (2).

A. In the unilateral case.

1. The acute case. In acute disease some of the signs are catarrhal, and clear up quickly with rest in bed, but the danger of spread to the other lung is so great, that in any acute febrile case with definite signs confined to one lung it is safer to induce a pneumothorax. Cavitation occurs early in these cases but it is a mistake to defer pneumothorax until signs of one appear. Plate XXIX. shows the X-ray of a child on January 12th, 1925. She then had harsh breath sounds at the root of the left lung and a high intermittent temperature, but very slight cough and no tubercle bacilli were found on examination of the sputum. She was admitted into hospital on February 4th, 1925, and Plate XXX. shows how the disease had spread. At this time crepitations were heard over the upper part of the left lung, and she had developed purulent sputum containing large numbers of tubercle bacilli. Her temperature was swinging between 98° F. and 108° F., but fortunately the condition remained unilateral. The lung

was at once collapsed, after which the spread was stopped and she made a good recovery.

2. The subacute case. If, in spite of rest in bed for six weeks in a subacute case, the activity persists or improvement is not satisfactory. As a common example one may quote the patient whose condition does not become stable and the slightest exertion, such as getting up for a few hours, causes a rise in temperature or some other symptom. The alternative to artificial pneumothorax in these cases is prolonged rest in bed and if the patient is prepared to submit to this and is of the right temperament it will give him almost, if not quite, the same chance of recovery.

3. The chronic case. In a chronic case, if the disease is unilateral and tubercle bacilli are present in the sputum, it is usually best to try to induce artificial pneumothorax, but these cases usually belong to the fibro-caseous type and it is often found that adherent pleura prevents the collapse of the diseased portion of the lung. There is almost always some cavitation in these cases even if it is not shown by X-ray, and in any case the prognosis is bad. It is unusual for such patients to live more than a few years unless some form of collapse treatment renders the sputum free from tubercle bacilli.

4. The patient has to earn his living or for some reason is unable to undergo prolonged treatment. There is no doubt that with the affected lung collapsed a patient is able to lead a more active life with much less risk of a breakdown than one not so treated.

5. There is repeated or severe hæmoptysis.

6. There are cavities. A cavity is always a potential danger even if it is dry. In the early and subacute stages cavities often close after a period of simple rest in bed, but it is almost always wise to close them by pneumothorax if possible. The chronic fibroid cavity will not close by rest nor will pneumothorax succeed except in a very few cases, but sometimes apicolysis or thoracoplasty will succeed.

7. In children. Pneumothorax should always be induced in children between the ages of two and fourteen if they develop unilateral tuberculosis of the lung. In bilateral cases the treatment usually leads to rapid spread of disease in the other lung. At the age of puberty the disease is usually acute and pneumothorax should be induced if it is unilateral. The older the child, the less important is it to regard some disease in the better lung as a contra-indication. Armand-Delille (3) has described the results of pneumothorax treatment in fifty children, and his work does much to dispel the old idea that pulmonary tuberculosis in young children is universally fatal.

8. Pregnancy. It is usually wise to terminate pregnancy in cases suitable for artificial pneumothorax and it should not be advised instead of abortion unless the pregnancy has advanced beyond the fourth month. If the disease is not detected until after this period it is safer to induce a pneumothorax in order to reduce the risk of an exacerbation of symptoms which occasionally follows parturition.

9. A tuberculous pleural effusion should be converted into a pneumothorax if there is active disease in the underlying lung.

10. There are certain complications, such as tuberculous laryngitis. If there is actual ulceration of the intestine pneumothorax is, of course, useless, but in many cases of diarrhœa or other gastro-intestinal symptoms resulting from toxæmia, the symptoms may be completely relieved when the diseased lung is collapsed.

B. In bilateral cases the indications for inducing artificial pneumothorax are affected by the following considerations :–

1. The disease is being treated in only one lung by unilateral collapse ; if, therefore, there is any active disease in the other lung this also must be treated. In other words, unilateral pneumothorax is affecting only part of the tuberculous lesion in a case of bilateral disease.

2. If one lung is much worse than the other collapse may remove so much of the toxæmia and cause so great an

improvement in the patient's general condition that the disease in the better lung also improves.

3. On the other hand collapse of one lung may increase the activity in the other. This is especially the case when there is early acute bilateral disease.

From this it follows that artificial pneumothorax may be considered under the same conditions in bilateral as in unilateral cases of pulmonary tuberculosis provided one remembers that it is impossible to get complete relaxation of both lungs and unless the diseased parts of both lungs can be collapsed it may be better to obtain a full collapse of one lung and treat the other by sanocrysing. And moreover, that collapse of one lung may aggravate the disease in the other.

In coming to a decision as to the wisdom of inducing pneumothorax in a bilateral case it is more important to consider the degree of activity than the actual amount of disease, as determined by physical signs, in the better lung.

Bilateral Artificial Pneumothorax. There is a large amount of reserve in the lung so that life can continue even after the greater part of it is destroyed. In most cases of bilateral disease there are adhesions which prevent the lung from collapsing sufficiently to endanger life, and it is remarkable how much collapse can be obtained in both lungs without any apparent ill-effect on the patient. There are, however, two great difficulties in producing successful pneumothoraces simultaneously on both sides. The first is that one cannot obtain complete collapse on both sides at the same time, and it is in cases of complete collapse that one obtains the best results. The second is that in acute cases, when there is considerable disease in both lungs bilateral collapse is apt to produce so much dyspnœa that the treatment has to be stopped, whereas in the more chronic cases with adhesions bilateral collapse is well borne, but usually unnecessary. The ideal in bilateral cases is to obtain a selective collapse so that the

diseased portions of both lungs are collapsed and the healthy portions maintain their functions.

The type of case where the most striking results of collapse therapy are seen is the acute case with high fever and rapidly spreading disease, but remaining confined to one lung. This type of case is common in girls of about fourteen to eighteen years, and to a somewhat less extent in boys from about seventeen to twenty-one. Unfortunately, these cases are almost always bilateral, and in acute cases simultaneous bilateral pneumothorax usually produces increasing dyspnœa and has to be stopped before the treatment has had any effect on the spread of the disease. It may however be successful and is worthy of trial.

In more chronic bilateral cases a simultaneous collapse of both lungs is often beneficial especially if there are cavities which close as the tension is relieved. In unilateral cases the results of those where an efficient collapse is obtained, and those where the collapse is incomplete, are very different, and it is not surprising therefore to find less satisfactory results in bilateral cases where only partial pneumothorax is obtained. Bilateral collapse is, however, of definite value and is now often practised. Many patients carry on their occupation with a bilateral pneumothorax and it is remarkable how free from dyspnœa or other symptoms they may be.

One patient, a woman of twenty-six, who had diabetes for which she was being treated with insulin, developed acute tuberculosis in September, 1935. Radiography showed extensive disease and cavitation in the upper half of the right lung and a little in the middle zone of the left. In December, 1935 a right artificial pneumothorax was induced, but the disease continued to spread in the left lung which was therefore collapsed by artificial pneumothorax in June, 1936. On the right side there were some adhesions which prevented a satisfactory collapse of the upper zone and held open a cavity and her sputum continued to contain tubercle bacilli. The adhesions were cauterised in July, and four

hours after the operation she went into a diabetic coma and for three days her condition was critical. She had no margin so that even a slight increase in her insulin produced hypoglycæmia. She recovered however and at present her condition is very satisfactory. She has no cough or sputum, very little dyspnœa and she leads a quiet but practically normal life. She travels fifty miles by train for her refills after which she goes to a theatre and stays the night in London. Plate XXXI. shows her present condition and the last few refills have been as follows :—

20th January, 1937.					
Left.	−12	−6	500	−5	−0
Right.	−10	−4	500	−4	−1
27th January.					
Left.	−10	−6	500	−4	−1
3rd February.					
Left.	−12	−4	500	−5	−1
Right.	−16	−8	500	−5	−0
10th February.					
Left.	−10	−5	500	−5	−1
17th February.					
Left.	−12	−7	500	−4	+1
Right.	−18	−8	500	−4	−0

The interval for the left refill was now increased to a fortnight.

3rd March.					
Left.	−12	−7	500	−4	−0
Right.	−20	−10	400	−5	−0
17th March.					
Left.	−14	−8	550	−5	−1
Right.	−18	−7	400	−6	−2
31st March.					
Left.	−12	−8	500	−5	−1
Right.	−20	−8	400	−6	−1

Sometimes it happens that during pneumothorax treatment active disease appears in the non-treated lung and begins to spread rapidly. In such a case it is often necessary to collapse this lung without waiting for the other to re-expand. Plate XXXII. shows a left pneumothorax with adhesions holding out a large cavity and with disease in the right lung. Plate XXXIII. shows the condition when the adhesions have been cauterised and a right selective pneumothorax started. Plate XXXIV. shows a left pneumothorax and XXXV. the same case two years later when the left lung has re-expanded and a right pneumothorax is being kept up.

Bilateral collapse has been practised for many years, and Forlanini (4) had two cases which he published in 1911.

Burnand (5) induced a right artificial pneumothorax in a patient in June, 1915. In October, the patient had lost the tubercle bacilli from the sputum and was free from symptoms. In April, 1918, he had influenza followed by hæmoptysis and pyrexia. In July he had tuberculous laryngitis; signs of active disease appeared at the left apex, and tubercle bacilli were present in the sputum. The pneumothorax treatment was stopped and the right lung re-expanded. In January, 1919, a left artificial pneumothorax was induced carefully, a negative pressure being left after each refill. The patient was very dyspnœic, but was able to get up and walk about quietly. He died suddenly in May, 1919.

Burnand's second case was a patient for whom a left artificial pneumothorax was induced in January, 1918. The patient improved at once, but the symptoms returned in October, and signs of active disease on the right side were found. The treatment was stopped, and the right lung became gradually worse until June, 1920, when a right pneumothorax was induced. The dosage varied from 200 to 500 c.cm., and the intervals between each injection were about eight days. A negative pressure was left after each refill. Sixteen refills were given. There was some improvement, but at the end of September right pleural

effusion developed, and there was much dyspnœa. The heart became very weak, and the treatment was discontinued.

In one of my patients there were extensive signs of active disease over the left lung, and over the right lung as far as the third rib in the mid-clavicular line in front, and the angle of the scapula behind. She had just returned from a sanatorium, where she had become much worse, the physical signs having extended very rapidly. A left pneumothorax was induced, and four refills were given. The treatment was then stopped for seven weeks, when a right pneumothorax was made and five refills given. The patient was very dyspnœic, but there was less sputum, and the cough was not so troublesome. The activity of the disease was checked and the case developed into one of the chronic fibroid type, the patient being still alive eight years later.

Saugman (6) describes seven cases in which he made a bilateral pneumothorax. One had the left lung collapsed from November, 1912, to July, 1913, and the right lung from January, 1914, to June, 1915. Since then he has been in good health and was known to be still at work in 1919. All the other six cases died.

Contra-lateral Artificial Pneumothorax. When, owing to adherent pleura, the diseased lung cannot be collapsed, partial pneumothorax has been induced on the healthy side with the object of giving some rest to the bad lung. It is necessary to have a mobile mediastinum and the pleura on the healthy side must be free from adhesions. In certain cases where the fibrosis in the affected lung exerts a pull on the mediastinum some benefit may result by a partial contra-lateral pneumothorax which, by making the intrapleural pressure less negative, assists the mediastinum to bulge towards the affected side. Apart from this I do not regard this treatment as of any value.

Contra-indications

1. In the absence of any of the special indications

mentioned, pneumothorax should not be induced if the lesions are healing under ordinary treatment and the patient can afford the time to allow arrest to take place.

2. When there is so much adherent pleura that it is possible to produce only a partial pneumothorax which is not sufficient to rest the diseased part. Sometimes a very partial collapse will have a beneficial effect on the disease, but if it has no effect it is not wise to maintain it. One should remember that a satisfactory collapse is often obtained although physical signs, X-ray or a history of repeated attacks of pleurisy, or even pleural effusion may suggest that there are extensive adhesions. One should never assume that pneumothorax is impossible until one has tried to induce it. A partial pneumothorax may sometimes be converted into a complete one by cauterisation of adhesions.

3. Patients with a highly neurotic temperament do badly with pneumothorax as with any other treatment. I have had to discontinue the refills owing to the mental state of the patient.

4. It is obviously unwise to induce pneumothorax when the tuberculosis of the lungs is in a very advanced state or is a terminal phase of some intercurrent disease. At the same time, intercurrent disease need not of itself be a contra-indication. For example, many patients with diabetes and tuberculosis do extraordinarily well with insulin and pneumothorax. Asthma and emphysema are not always absolute contra-indications, although in such cases one often has to stop the treatment owing to dyspnœa, but I have known asthmatic consumptives successfully treated by pneumothorax.

5. Active bilateral disease is a contra-indication only if collapse of one lung aggravates the disease in the other and collapse of both lungs either simultaneously or alternately is not feasible.

Apparatus

There are many different kinds of apparatus (2) and (15) designed to induce artificial pneumothorax, and after a time the physician gets accustomed to a certain type of instrument, some liking one and others another, but really it is a matter of small importance as to which is used. When asked to advise in the choice of a pneumothorax apparatus I always recommend the simplest for hospital and institutional use, and for private work the most portable. The apparatus devised by Drs. Lillingston and Pearson and shown in Fig. 16, is very simple and satisfactory. It consists of two bottles

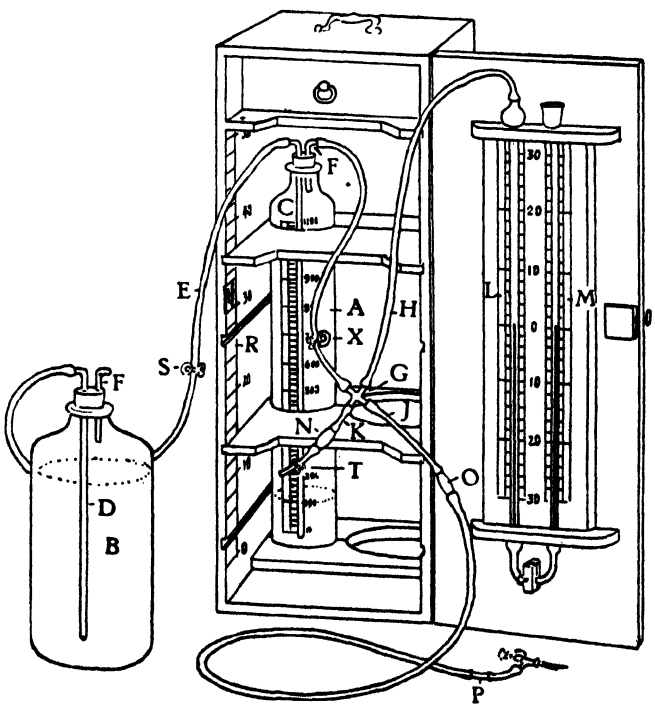

FIG. 16. Lillingston and Pearson's artificial pneumothorax apparatus.

A and B. A is graduated from 0 to 1,100 c.cm., and is tall and narrow, so that the graduations are easily read. Both bottles are fitted with rubber stoppers. A glass tube C passes through one stopper to the bottom of bottle A and a similar glass tube D passes through the other stopper to the bottom of bottle B. These two tubes are connected by a rubber tube E. Shorter glass tubes FF pass just through both stoppers. Now if one bottle and the tubes CDE are filled with water the water will flow into the other until it is on the same level in the two bottles. Air will enter through tube F in the emptying bottle and be forced out through tube F in the other bottle. Tube F from the graduated bottle A is connected by a rubber tube with one limb of a cross-shaped glass tube G. The other three limbs of this glass tube are fitted with rubber tubes, one of which H, is connected with the manometer, another J, is connected with the needle and, the third K, is a short tube used only for filling the apparatus with air, oxygen or other gas.

If the tube K is clamped and the bottle B raised so that water flows into A, the air in A will be forced out through the needle at the end of tube J, but if J is also clamped, the air will force down the column of liquid in the limb L of the manometer and the liquid in the limb M will rise. NO are filters of sterilised cotton-wool. P is a short glass tube to act as a window in the tube J near the needle. R is a scale by which the pressure can be read in centimetres of water. STX are clamps. When the apparatus is not in use the open ends of tubes J and K are closed by sterilised glass rods. The apparatus is very simple, the bottles can easily be cleaned and the tubing boiled. The limb of the manometer should be long and fitted with bulbs at the tops so that the liquid may not be forced out of M by a high positive pressure or sucked into tube H by a negative one. I use water coloured with a little red ink for the manometer liquid. Mercury is too heavy.

Before using the apparatus it is important to see that there

is no leak. This can easily be done by making a small negative or positive pressure recorded on the manometer and after clamping the tubes seeing if any air escapes.

In order to use the apparatus, bottle B is filled with 1 in 100 carbolic solution slightly coloured to make it more visible. A little air is blown with a Higginson syringe through F into bottle B to force the liquid through tubes DEC into bottle A, and thus start a syphon action. When the liquid reaches 0 in bottle A, the tubes are clamped at S, X and T, so that the needle is in connection only with the manometer. The trocar is removed from the needle and the tap at the top turned off. Clamps S and X are now unfastened and the end of the needle is put into a little methylated spirit through which bubbles of air should pass if the apparatus is working properly. Liquid should be allowed to flow from bottle B into bottle A until its level is equal in the two bottles, and the pressure in the tubing is atmospheric. Thus, air cannot be forced out through the needle under pressure with the risk of causing gas embolism. The tubing is now clamped at S and X (T is already clamped), the needle is dried over a spirit flame and the apparatus is ready for use. For the initial induction of pneumothorax I use a Riviere needle, as this has a blunt end when the trocar is removed, and is less likely to injure the visceral pleura than the pointed Saugman needle, which I use for giving refills. I do not think any other instrument is better than this for hospital use; besides being effective it is cheap and simple, and any part that happens to get broken can easily be replaced. For private work, however, when the instrument has to be carried from place to place and taken in trains and cars it is rather awkward and heavy, and the liquid is apt to spill. Maxwell has designed a portable apparatus which is very satisfactory. It is shown in Fig. 17 on p. 230, and has the advantages of having no liquids in any part and being light and portable. It consists of a metal cylinder containing

a piston which can be drawn up to the top of the cylinder and allowed to fall by its own weight. The cylinder holds 200 c.cm. of gas and a graduated scale shows the amount used. At the top is a metal tube and tap so that it can be filled with air or other gas, and opposite is an outlet which leads the gas through a valve which regulates the rate of flow and then through a simple filter to the tube connected with the

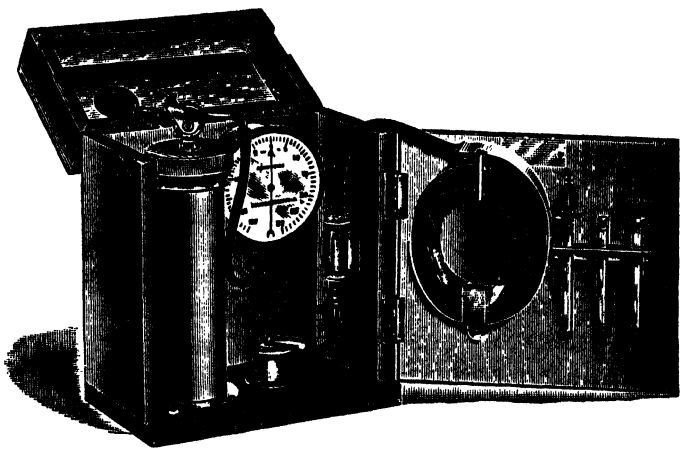

FIG. 17. Maxwell's artificial pneumothorax apparatus.

pneumothorax needle. A manometer is in direct connection with the pneumothorax needle.

Choice of Gas. I always use air for ordinary pneumothorax work not only because it is easy to get, but because it approaches most nearly to the alveolar air and therefore less interchange of gas takes place in the pleural cavity. Rist and Strohl (7) concluded that there is a balance between the gases of the pneumothorax cavity and the gases in the venous blood. When either air or nitrogen is introduced there follows a short period during which the total gas in the pneumothorax cavity is increased before interchange of gases establishes a balance. Tobiesen (8) found on analysis N_2 90 per cent., CO_2 6·7 per cent., O_2 8·4 per cent. in the

pneumothorax cavity whether nitrogen, carbon dioxide or oxygen had been used at the previous refill. Oxygen is absorbed rather more rapidly than air or nitrogen, and is therefore the best gas to use if a rapid absorption is required, as, for example, in certain cases of pleural effusion. It has been used at the initial induction of pneumothorax on the assumption that it is less likely to cause gas embolism owing to its rapid absorption. For a similar reason carbon dioxide, which is readily absorbed by the blood serum, has been used. With proper technique gas embolism should not occur and the choice of gas in this respect makes no real difference. Helium has been tried, but was found to have no advantages and to be absorbed as quickly as air or nitrogen. Nitrogen is still used by some, but I think air is better. Perhaps the American was right when he said that the difference between the two was only a matter of dollars.

The following figures suggest that there is no appreciable difference in the rate of absorption between the two gases even when a large quantity of gas is given and high pressures are maintained :—

1. One case having refills at three-weekly intervals.

March	10th	.	−6	−0	...	700 air	...	+4 + 9
,,	31st	.	−6	−1	...	750 ,,	...	+4 +10
April	21st	.	−5	−0	...	750 N_2	...	+3 + 9
May	12th	.	−6	−0	...	700 air	...	+4 + 9
June	2nd	.	−5	−1	...	700 ,,	...	+4 + 8

2. Another having refills at four-weekly intervals.

April	7th	.	−8	−2	...	1,000 air	...	+2 +12
May	5th	.	−8	−2	...	1,000 ,,	...	+1 +11
June	2nd	.	−7	−3	...	1,000 N_2	...	+2 +11
,,	30th	.	−8	−3	...	1,000 air	...	+1 +11

The rate of absorption varies enormously in different individuals and, as a general rule, it is absorbed more quickly in the early stages of pneumothorax than in the later ones.

Technique of Inducing Artificial Pneumothorax

The mental attitude of the patient is of the greatest importance. In a hospital or sanatorium where several patients are having pneumothorax treatment the immediate benefits are so obvious to all, and the inconveniences of the refills so slight, that patients often ask to have it and become very disappointed and depressed if told they are unsuitable. Nowadays it is so widely known that the majority of patients expect that it should at least be mentioned as one of the possibilities of treatment. There are still a few, however, who regard pneumothorax as an operation only to be considered as a last resort or as a kind of experiment, and who work themselves up into a state of nerves so that there is a considerable rise of temperature on each day the refill is due, even if for some reason it is not given. A little dyspnœa becomes serious gasping for breath, relieved at once by morphia, a little pain becomes agony and a feeling of tightness in the chest may lead to most alarming symptoms of distress which are never seen in the patient with confidence. On two occasions I have had to stop the refills owing to the mental condition of the patient. In such cases it is almost essential for the patient to have a preliminary course of sanatorium treatment and, after a week or so, if he does not himself suggest pneumothorax, he will usually welcome rather than dread the idea of it. Unfortunately this type of patient is the one who so often refuses to go into a sanatorium, and it may be quite impossible to get him into the right frame of mind for any form of real treatment, though a serious attempt should be made to influence him and gain his confidence before starting a pneumothorax.

In any case, the procedure should not be in the nature of a surgical operation and should be done in the patient's room and not in an operating theatre, although, of course, all instruments should be boiled and the strictest precautions taken against sepsis. It is unnecessary to make any special

preparation of the patient or to give him a preliminary dose of morphia or sedative. In case of accidents, brandy, pituitary extract and other stimulants should be in readiness and hot water bottles at hand, but these precautions should be taken without the patient's knowledge and to him the whole procedure should be made to appear no more than an ordinary hypodermic injection.

The site of election for puncture is the fifth intercostal space in the mid-axillary line. If the pleura is adherent in this area, so that the lung will not collapse, an attempt should be made in the sixth space in the posterior axillary line, and, failing this, in the seventh space, just below and internal to the angle of the scapula. If the pleura is adherent to all these sites, I try in the first intercostal space below the clavicle. Should the pleura be adherent here also, it is not necessary to make any further attempts to induce a pneumothorax, for with such widespread adhesions any collapse obtained would be dangerous rather than beneficial.

If the skin and pleura are properly anæsthetised with novocain and sharp needles are used, the punctures are painless, and it is possible to make all four attempts at one sitting. In the case of a patient who shows signs of nervousness it is sometimes best to stop after the second attempt, and to postpone the other punctures for a day or two; it is hardly ever necessary to do this, however, and the great majority of patients prefer to get the whole proceeding over at one time. Only a few patients get a satisfactory collapse after failure to produce pneumothorax at the first attempt, and a successful collapse after a second failure is rare.

The axillary region is the site of election for making the first attempt. Here the ribs are far apart, and there is little muscle tissue covering the chest. Moreover, adhesions are less likely to be found in the axilla than at other sites.

In a case where much pleural adhesion is suspected, but where a friction rub is heard, an attempt should be made

over the area of friction, for this indicates the absence of adhesions. Whatever the clinical or X-ray findings, one should never assume the presence of too many adhesions to allow a pneumothorax, for one often obtains quite a good collapse in the most unlikely cases. When trying to induce a pneumothorax it is best not to puncture the pleura over a large cavity or over an area of extensive caseation.

The patient should be arranged comfortably in bed, supported by pillows so that the site of the puncture is uppermost and the ribs in that region are as far apart as possible. The area selected for puncture is then cleaned and ½ c.cm. of a 2 per cent. solution of novocain injected intracutaneously so that a small lump is raised and the skin anæsthetised. The hypodermic needle is then inserted into the middle of this lump and pushed slowly down between the ribs, novocain being injected all the time, the last ½ c.cm. against the pleura. This procedure is perfectly painless if the needle is small and sharp. I use a Record needle, size No. 20.

The pneumothorax apparatus, which has previously been got ready and tested as already described, is then arranged at the patient's bedside. If Lillingston and Pearson's apparatus is used (Fig. 16) clips SXT are fastened so that when the chest is punctured the pleural cavity will be in connection only with the manometer and not also with reservoir A. For a refill the liquid in A should stand at 0, but for the initial induction of pneumothorax the liquid in A should be at the same level as that in B, so that on releasing clips S and X air can be sucked from A into the pleural cavity by negative pressure, but cannot be forced in by the rising liquid in A.

For refills Saugman's needle, which has a sharp point, is satisfactory, but for the initial injection it is better to use Riviere's needle, which consists of a blunt-ended cannula, through which passes a sharp-pointed trocar. In choosing a needle I should avoid one with too small a bore, as the

movements of the manometer are not so readily registered with small-bore needles. Some physicians however prefer a small-bore needle and Morland's model is a good one. With this it is possible to give a refill which is practically painless without using any local anæsthetic and occasionally patients prefer this method.

For the initial induction of pneumothorax a Riviere needle is fixed to the end of tube J and pushed gently through the anæsthetised area of skin between the ribs and down to the pleura. Provided the needle is sharp, this is quite simple, and it is not necessary to make a preliminary incision of the skin with a tenotome. On reaching the pleura the trocar is withdrawn into the stuffing box and the tap at the top of the needle turned off. The blunt end of the cannula is then gently pushed through the parietal pleura, and when it is in the pleural cavity the manometer will show a negative pressure with oscillations corresponding to inspiration and expiration. At this stage the patient must keep absolutely quiet and the needle be held perfectly steady or it is liable to slip out of the pleural cavity. When, from the oscillations, it is certain that the needle is between the visceral and parietal layers of pleura, clips S and X are unfastened and about 50 c.cm. of air allowed to be sucked into the pleural cavity; this allows a small air space to collect round the needle since the patient has been so arranged that the site of puncture is uppermost. If the manometer still shows a negative pressure with oscillations, it is now safe to raise bottle B and allow some 800 c.cm. of air to enter the pleural cavity. The readings of the manometer are then noted and the needle withdrawn. The exact quantity of air which is introduced will depend on the intrapleural pressure, but for the initial injection 800 c.cm. may be taken as a good average as it is not enough to cause a reaction and yet sufficient to leave an air space which lasts until the first refill on the following day. It is best not to put any dressing over the small puncture which will then heal without leaving a scar.

Sometimes when the needle enters the pleural cavity there is registered a negative pressure, but the oscillations soon stop. This is because the visceral layer of pleura tends to obstruct the end of the cannula. At first the small quantity of air in the needle and tubing is sucked into the pleural cavity, producing a bubble, but as this spreads away, the viscera pleura blocks the end of the cannula. It sometimes happens that during inspiration the pressure becomes more negative, but during expiration the visceral pleura blocks the end of the cannula, so that there is a valve action which practically makes the manometer a minimum pressure one.

If the lung is punctured by the needle, air may escape from the alveoli and produce an air space so that oscillations occur as in an ordinary artificial pneumothorax. These small punctures soon heal and, as a rule, do no harm, but they are especially liable to occur if a sharp needle is used for the initial injection.

When the needle enters the lung or a bronchus or cavity there may be oscillations registering a negative pressure during inspiration and a positive one during expiration, but with the mean pressure at zero. If air is introduced it will not affect the pressure in such a case, and I have seen one where 2,000 c.cm. of air were injected and yet the oscillations of the manometer ranged a point or so above and below zero, because, of course, the air was not entering the pleural cavity, but escaping through the bronchi.

Should the needle pierce the diaphragm and enter the abdominal cavity the oscillations are the same as for the chest: that is to say, a low pressure will be registered with inspiration and a higher one with expiration.

Sometimes the manometer registers a positive pressure which becomes gradually higher. This may be due to the needle being in a blood vessel, when blood will pass up the tubing and be seen through the glass tube P. It is in such a case that there is a danger of gas embolism if air is forced in under pressure. I have had cases where the

needle has entered a large vessel or the heart, and blood has rushed up the tubing, but no ill effects have followed.

Refills. The object of artificial pneumothorax is to relieve the tension on the lung, not to compress it. It is on careful spacing of the refills and giving the correct quantity of gas that success or failure depends. When a lung is collapsed no special skill is required to put a needle into the pleural cavity and inject gas, but it requires considerable experience to judge how much to give and when to give it. The X-ray is the most accurate method of finding the exact degree of collapse and screening should be done before and after each refill. Sometimes air is retained for weeks or even months so that refills are needed only at long intervals. In other cases, this so-called *pneumothorax insatiable*, air is absorbed in a few days, even after large refills. The rate of absorption may vary from time to time in the same individual, but as a rule it is fairly constant.

A common mistake is to leave too long an interval between the refills so that the lung is alternately collapsed and expanded. I call this the concertina method (9). The following case is an example of this faulty procedure: a patient who had been under treatment by artificial pneumothorax for two and a half years was having 1,500 c.cm. of air every five weeks, at the end of which time the lung had completely re-expanded. It was found that 600 c.cm. left a pressure of −6, but caused a good collapse which lasted for a fortnight when the lung began to re-expand quickly. The correct treatment therefore was to give 600 c.cm. every fortnight.

If the lung is allowed to expand sufficiently for the visceral and parietal pleura to come in contact, adhesion is likely to take place. Firm adhesion may occur in three days or less, and no amount of intrapleural pressure will separate the adherent surfaces. Even at operation it is often found impossible to do so without tearing the lung, so firm are the adhesions. The process gradually spreads until the whole of the pleura becomes adherent and the pneumothorax

cavity obliterated in spite of maintaining high intrapleural pressures or injecting oil into the pleural cavity. Lillingston (10) describes a case in which the refills were being given once a month, but were left once for an extra two weeks, when it was found that 300 c.cm. of gas caused a pressure of +8. Frequent refills were given and the pressure taken to +16, but the pneumothorax cavity became obliterated. This closing of the cavity is especially liable to happen if there is an effusion. I (11) have referred to it as obliterative pneumothorax and sometimes it leads to fibrosis and is a satisfactory termination of the treatment, but if it is desired to maintain a pneumothorax it is of the utmost importance to prevent the visceral and parietal pleura from coming into contact with each other.

Perhaps the most common mistake is to maintain the wrong degree of collapse. Some patients do best with a very partial collapse, and if this is increased they begin to get thin, develop dyspnœa and lose ground. Others fail to improve until a complete collapse is obtained and large and frequent refills are given. Some of my best cases had regular refills of over 1,000 c.cm. with full collapse of lung and often considerable displacement of the mediastinum. In the majority of cases, however, small refills are more suitable, and I rarely give more than 300 c.cm. for the initial injection and 600 for refills. The first refill should be given twenty-four hours later, and subsequent small refills every few days, so that the desired collapse is obtained in about a fortnight. The following may be taken as two typical examples of the first few refills :—

1. June	5th .	−12	− 7	300 c.cm.	− 8 − 5
„	6th .	−12	− 7	400 „	− 6 − 8
„	8th .	−10	− 5	400 „	− 6 − 8
„	11th .	−10	− 6	400 „	− 5 − 1
„	14th .	− 8	− 8	400 „	− 5 − 1
„	18th .	− 8	− 4	500 „	− 5 − 2
„	23rd .	− 7	− 8	500 „	− 4 − 1

2.	Nov. 14th	.	−15	− 9	300	„	−12 − 6
	„ 15th	.	−14	− 9	400	„	−10 − 4
	„ 17th	.	−12	− 7	400	„	− 8 − 4
	„ 19th	.	−12	− 7	500	„	− 6 − 2
	„ 22nd	.	−10	− 6	500	„	− 6 − 2
	„ 25th	.	− 8	− 4	500	„	− 5 − 1
	„ 29th	.	− 6	− 3	500	„	− 4 − 0

All pressures are centimetres of water.

A good collapse should be obtained at the end of about a fortnight after the initial induction and it is then necessary to find the optimum degree of collapse which is that most suitable for the individual patient; some do best with a very partial and others with a complete collapse. The next thing to discover is how long this optimum degree of collapse will last without a refill, and this can be done by watching the degree of re-expansion of lung by X-ray, and by observing any return of symptoms.

If, after a refill leaving the intrapleural pressure at −2, the patient is free from symptoms, but with any further collapse he does not do so well and begins to lose weight or have dyspnœa, we may regard −2 as the maximum pressure which should be reached for that patient at that stage of the treatment.

In the same way the minimum pressure may be found, a lower one producing a return of symptoms or failure to give sufficient rest to the lung. Suppose −12 is the minimum pressure, then in this case the optimum degree of collapse is that which exists between −2 and −12. The refills and the quantity of gas required can be arranged accordingly. X-ray control is essential.

When a lung is completely collapsed by pneumothorax it appears as a ball projecting from the hilum if there are no adhesions and the healthy part of the lung will re-expand before the infiltrated part, so that there are three ways of conducting treatment.

1. To arrange the interval between the refills and the

quantity of gas given so that the lung is kept completely collapsed. By complete collapse I mean as much collapse as can be obtained by the lung's own elasticity. This must not be confused with compression of the lung, which may be produced by a positive intrapleural pressure.

2. To wait until the healthy part of the lung has almost re-expanded and then to give a refill and collapse it again. The advantages claimed are that it prevents the healthy portion of the lung from adhering to the chest wall and thus, in the event of its becoming infected, a full collapse can be maintained. Moreover, its partial re-expansion does no harm, whilst it is healthy and allows a longer interval to be left between refills than would be possible if a complete collapse was maintained. The diseased portion of lung is kept collapsed all the time.

3. The third method is to allow the healthy portion of the lung to expand and give small refills which are just sufficient to prevent re-expansion of the diseased portion of the lung. The affected part is kept at rest whilst the remainder is allowed to function. This is true selective collapse.

In my experience, the first of these methods gives the best result and should be employed whenever possible. In certain cases, however, selective collapse has advantages.

Adhesions may prevent a satisfactory collapse and lead to high intrapleural pressures after only a small quantity of air has been given. If it is decided to go on with the treatment, and try to stretch the adhesions, small frequent refills are better than large ones at longer intervals. High intrapleural pressures in such cases are useless when the adhesions are firm, and dangerous when they may be stretched, but are also liable to rupture. Moreover, a high pressure is very quickly reduced, as shown in the following cases :—

	Intrapleural pressure.		Six hours later.
1.	+18	+30 ...	+1 +8
2.	+12	+20 ...	0 +6
3.	+22	+28 ...	+1 +5

If high pressures are obtained it is quite common for a little surgical emphysema to develop around the site of puncture from escape of the gas, and this, of course, also lowers the intrapleural pressure.

When liquid develops during pneumothorax treatment the gas is less quickly absorbed, and the weight of the effusion empties the alveoli completely of air, so that the lung is often collapsed for a long period without a refill. It is important, however, to keep these cases under careful observation, for adhesions are liable to form under the effusion and to draw out the base of the lung, thus starting a process of obliteration of the pneumothorax cavity. To prevent this, aspiration or gas replacement should be performed if there are any signs of re-expansion at the base of the lung.

In a straightforward case, therefore, it may be said :—

1. That the lung should be allowed to collapse gradually.

2. That when it has been collapsed the optimum pressures should be determined for each individual case and thus correct dosage of gas and intervals between refills ascertained.

3. That the patient must be kept under careful X-ray supervision not only because the rate of absorption tends to vary as time passes, but also because it may be affected by such complications as spread of adhesions or pleural effusion.

Duration of Treatment. Professor Saugman (6) wrote : " The pneumothorax being complete I now normally let the patient be treated for about five years. If then all is normal, and if he wishes it, I let the pneumothorax close, choosing the summer season to allow the lung to re-expand, for the first months, observing the patient very closely, if possible in the sanatorium ; and no sooner do symptoms of relapse in the treated lung appear than I renew the injection of gas, but only in a very few cases have I seen reason to do so." He states that of forty-three patients discharged 1907-1917, and at work in 1919, the pneumothorax lasted—

½ year in 1 case.
¾ „ „ 2 cases.
1 to 2 years in 11 cases.
2 to 3 „ „ 17 „
3 to 4 „ „ 7 „
4 to 5 years in 2 cases.
5 to 6 „ „ 1 case.
6 to 7 „ „ „
Unknown in 1 case.

In a large number of cases the termination of the treatment is settled by various factors such as obliteration of the pneumothorax cavity by adherent pleura, development of active disease in the other lung, etc.

If, however, the patient is doing well and keeping free from symptoms and at work I think the collapse should be maintained for three years and then the lung allowed to expand gradually during the summer. Before allowing complete re-expansion I give two or three refills at long intervals, using only sufficient gas to prevent the visceral and parietal layers of pleura from coming into contact, for by this means it is possible to re-collapse the lung fully should a return of symptoms make this advisable.

If the disease is very slight, healing will probably take place in two years or less but it is safer to allow an extra year if possible. The greater the extent of the disease when treatment is started the more chance there is of a relapse afterwards but if there is still activity after three years collapse, healing is not likely to occur and I think it is usually best to abandon the pneumothorax and perform thoracoplasty, for by this time there is probably sufficient fibrosis to make this operation the better method of collapse.

When there are areas of adherent pleura or adhesions which cannot be cauterised so that the pneumothorax is partial and inadequate it is a mistake to maintain it too long. It is always unwise to keep up a small useless pneumothorax which is a source of danger, especially if it is near an unclosed cavity which may rupture into it.

Dufault and Laroche (12), in a discussion of the difficult problem of when to terminate pneumothorax treatment,

conclude that to allow re-expansion of lung too soon is to risk the recurrence of activity and may necessitate thoracoplasty if the pleural surfaces adhere. To maintain the pneumothorax too long is to risk thickening of the pleura and the dangers of a permanent pneumothorax. In their opinion a small infiltration without cavities will heal quickly and so the lung need not be collapsed for so long as one with old fibro-caseous lesions which may require seven years of collapse before healing occurs.

Selective Collapse. It was suggested by Parry Morgan (13) that a partial pneumothorax would produce the same results as a complete one by resting the diseased part; he pointed out that during inspiration the normal parts of the lung expand against the chest wall, so that a small quantity of air in the pleural cavity would tend to collect over the less expansile or diseased portions. In practice the matter is generally complicated by adhesions which are especially to be found over the diseased parts, but there is no doubt that in the absence of adhesions a small quantity of gas introduced into the pleural cavity does tend to collect over the diseased area, and that if a complete pneumothorax is produced the normal portions of lung re-expand more readily than the others when refills are stopped.

Selective collapse is useful to collapse an apical lesion or cavity if full collapse of the lung is contra-indicated by disease in the other, or in combination with a pneumothorax or thoracoplasty on the other side.

In my experience the best results are obtained by a full collapse, by which is meant complete relaxation of the lung so that it collapses by its own elasticity. Selective collapse has its uses and often yields good results, but if the lung is insufficiently collapsed the disease will spread.

Riviere (14) was not impressed by the results of partial collapse and pointed out several practical difficulties. The small air space is apt to close and is difficult to maintain

in many cases. Frequent refills and constant X-ray control are needed so that he said the treatment is one for millionaires or the very poor.

In an ordinary acute or subacute case a full collapse should be obtained if possible. Even if a selective collapse is sufficient for the lesion subsequent spread into the uncollapsed portion of the lung may occur and if the pleura has been allowed to adhere a full pneumothorax is impossible.

A selective pneumothorax may be obtained by giving small refills with careful X-ray control until the diseased part of the lung is sufficiently collapsed. Refills large enough to maintain the selective collapse but not large enough to collapse the rest of the lung are then given.

When possible, however, I prefer to produce a complete collapse at first and after a few weeks allow parietal re-expansion until the desired degree of collapse of the diseased part is obtained and then maintaining this selective collapse by small refills. This method has the advantage of enabling the physician to study the condition of the patient with a full collapse and compare it with that under varying degrees of collapse. Plate XXXVI. shows selective artificial pneumothorax with solid collapse of the upper zone.

Intrapleural Pressure. Intrapleural pressure is measured by the manometer. Coloured water is generally used, as mercury is too heavy, spirit gives large oscillations but water is quite satisfactory unless a very small needle is used. All pressures given in this book are centimetres of water.

The size of the needle is important, as the oscillations of the manometer are not well shown if too small a needle is used. If two needles (one being a large pneumothorax needle and the other a small hypodermic one) connected with the manometer by means of rubber tubes and a T-shaped glass rod are put into a distended rubber bladder, it will be found that the pressure registered through the small needle is the same as that registered through the large one, but it takes longer to register through the small needle. By giving the

bladder little taps, the manometer shows good oscillations if connected through the large needle, but hardly any movement if connected through the small one. Similarly, it will be found that if the liquid in one bottle of the pneumothorax apparatus is higher than the liquid in the other and the outlets are stopped, the column of water in the manometer will show the pressure, and it will be the same whether the tube to the manometer is fully patent or partially obstructed, but it will take longer for the pressure to be registered if the tube is partially obstructed. It is important that the respiratory oscillations should be well shown, and for this reason I like the bore of the needle to be at least 1·2 mm.

Normally the intrapleural pressure is negative owing to the elasticity of the lungs and becomes more negative during inspiration. The difference of pressure between full inspiration and full expiration varies in different patients, but it is often considerable and, of course, is largely dependent on the depth of respiration.

Posture has a big effect on intrapleural pressure and this should always be borne in mind when giving a refill to a strange patient, and he should be put in the same position for the refill as previously. Some physicians give refills with the patient sitting on a chair, others with him lying flat on his back, and others with him lying completely or partially on one side. If one has a record of previous refills it is usually possible to tell in what position they were given by putting a needle into the pleural cavity and moving the patient until the usual pressure is found.

If the patient lies on the pneumothorax side, the weight of the viscera will render the pressure in the pneumothorax cavity higher than if he lies on the healthy side. In one of my cases the pressure was −4 +2 with the patient lying on the left (pneumothorax side), but −10 −0 when lying on the healthy side. After a refill of 450 c.cm. the pressures were +1 +8 and −5 +2, respectively. In other words,

when this patient was lying on the healthy side he could raise the pressure in the pneumothorax cavity as much by turning on to the treated side as by having a refill of 450 c.cm. It is possible, therefore, to use posture if we wish to stretch adhesions, for by making the patient lie on the healthy side there will be a continual drag on the adherent surfaces. Posture will, of course, have a greater effect if the mediastinum is freely movable and if the lung is not bound to the chest wall by adherent pleura.

The intrapleural pressure is sometimes lowered by shifting of the mediastinum, and this may occur suddenly. With a very movable mediastinum it is often difficult and sometimes impossible to obtain a good collapse of the lung without displacing the mediastinum and producing symptoms by interfering with the function of the other lung and heart.

Rupture of a pleural adhesion sometimes occurs, and this may affect the intrapleural pressure.

As air is allowed to enter the pleural cavity, the pressure approaches that of the atmosphere, and then if more air is forced in to compress the lung the pressure becomes positive.

The following table shows how the intrapleural pressure rises as air is introduced :—

TABLE 21

	Case 1. *Initial Operation.*	Case 2. *Initial Operation.*	Case 2. *Three Months Later.*
Initial pressure .	−14 −12	−7 −5	− 3 − 0
After 100 c.cm. air .	−10 − 9	−7 −2	− 3 − 0
„ 200 „ „ .	−10 − 8	−7 −2	− 2 + 2
„ 300 „ „ .	− 9 − 6	−6 −2	0 + 2
„ 400 „ „ .	− 8 − 6	−6 −1	+ 1 + 4

The next two cases show the change of pressure when large refills are given. Some years ago it was the custom to introduce large quantities of air and the results were often

remarkably good in spite of the fact that it usually caused considerable displacement of the mediastinum. At the present time such large refills are seldom if ever used.

TABLE 22

	Case 3. *Refill.*	Case 4. *Refill.*
Initial pressure . .	−12 −8	−6 − 0
After 600 c.cm. air .	− 3 −0	—
„ 700 „ „ .	− 2 −0	—
„ 800 „ „ .	+ 2 +2	−2 + 4
„ 900 „ „ .	+ 4 +2	−2 +10
„ 1,000 „ „ .	+ 4 +6	+2 +10
„ 1,100 „ „ .		+4 +10
„ 1,200 „ „ .		+6 +11

In these four cases collapse was not interfered with by adhesions. If adhesions or pleural effusion reduce the size of the pneumothorax cavity, it will sometimes be found that there is a considerable negative pressure at first, but that it rapidly changes. For instance, in one of my cases in which the pneumothorax cavity was gradually becoming obliterated by the spreading of the pleural adhesions, a negative pressure of −24 −12 was found, but it became +20 +26 after 250 c.cm. of air had been introduced.

The following table shows the variation of intrapleural pressure at the initial operation when there are adhesions.

TABLE 23

	Case 1.	Case 2.	Case 3.
Initial pressure .	−4 − 0	− 4 − 0	−6 −0
After 50 c.cm. air .	+8 +12	—	—
„ 100 „ „ .	—	+19 +23	—
„ 200 „ „ .	—	—	+2 +6

The next table shows the variation of intrapleural pressure after the formation of pleural effusion :—

TABLE 24

	Case 1.	Case 2.	Case 3.
Initial pressure .	− 6 + 4	− 2 + 4	− 3 − 0
After 100 c.cm. air .	− 1 + 6	—	—
„ 200 „ „ .	+ 8 +14	—	—
„ 300 „ „ .	+16 +22	—	—
„ 400 „ „ .	—	+20 +22	—
„ 500 „ „ .	—	—	+10 +14

When the liquid is replaced by air the quantity removed must be greater than that of the air added if the intrapleural pressure is to be lowered. For instance, in one of my cases the pressure in the air-space above the liquid was −3 +1; after removing 700 c.cm. of liquid it was −14 −6, but on adding 700 c.c.m of air it became +3 +8.

The following cases show the effect of replacing liquid with air on the intrapleural pressure:—

1. Pressure −2 +4. After removing 1,650 c.cm. of liquid and introducing 1,100 c.cm. of air the pressure was −4 +2.
2. Pressure −8 +3. 1,350 c.cm. of liquid were removed and 1,100 c.cm. air introduced, and the pressure was −8 +2.
3. Pressure −5 −4. 1,250 c.cm. of liquid were removed and 1,000 c.cm. of air introduced, and the pressure was −4 −2.

The lung can remain well collapsed with a negative pressure. Indeed, one almost always finds a negative intrapleural pressure when the patient attends for a refill, even if X-ray shows that the lung is well collapsed and that very little re-expansion has taken place since the last refill. A positive pressure left after a refill is soon converted into a negative one so that there is no advantage in creating a high intrapleural pressure and it usually makes the patient

uncomfortable. Dumarest (15) says high pressures compress the air and not the lung and he regards an expiratory pressure of zero as one most suitable in most cases of pneumothorax even when adhesions are present. It is not often that one has the chance of seeing how quickly the intrapleural pressure alters, because at the initial operation only a small quantity of air is given and the initial pressure is altered very little, whereas in the later stages of the treatment, when there is a positive pressure, the refills are given at longer intervals. In the two following cases, however, a large initial quantity of air was given to check hæmoptysis, and the pressures were as follows: -

1.	Initial pressure . .	−11	−8
	After 1,400 c.cm. of air .	+ 1	+5
	Thirty-six hours later .	−11	−8
2.	Initial pressure . .	−10	−8
	After 900 c.cm. of air .	− 2	−0
	Two days later . .	− 7	−5

In the following three cases there was much adherent pleura :—

1.	Initial pressure . .	− 9	− 5
	After 120 c.cm. of air .	+10	+14
	Twenty-four hours later .	− 4	+ 1
2.	Initial pressure . .	− 4	− 2
	After 800 c.cm. of air .	+ 1	+ 5
	Sixteen hours later . .	− 2	− 0
3.	Initial pressure . .	− 5	− 2
	After 500 c.cm. of air .	0	+ 3
	Twenty-four hours later .	− 5	− 2

It sometimes happens that after a refill the patient complains of dyspnœa and tightness of the chest and a needle is put into the pleural cavity to find the pressure and, if necessary, to remove some of the gas.

1. Initial pressure before the fourth refill	−9	− 3
After 700 c.cm. of air . . .	+1	+ 3
Three hours later	−5	+ 2
2. Initial pressure before fifth refill .	−5	− 1
After 700 c.cm. of air . . .	+7	+11
Four hours later	0	+ 4
3. Pressure before initial operation .	−6	− 3
After 350 c.c.m of air . . .	−4	− 1
Sixteen hours later . . .	−6	− 4

These cases do not show any evidence of a temporary increase in the volume of gas after a refill.

If a big positive pressure is reached it will sometimes be found that the oscillations recorded by the manometer are reversed—that is, a higher pressure is recorded with inspiration than with expiration. Bjure (16) describes three cases in which the pressure was increased during inspiration, but normal fluctuations were recorded when the patient was told to use costal and not abdominal respiration. The reversed fluctuations in such cases are due to the descent of the diaphragm during inspiration on the healthy side and the ascent on the other side, and this can well be demonstrated by X-rays. This is known as paradoxical respiration, or Keinboch's phenomenon.

If the pneumothorax needle is accidentally put into the abdominal cavity the readings on the manometer are the same as in the pleural cavity.

H. de Carle Woodcock (17), at a post-mortem examination, found an intrapleural pressure of −1·5 in. of water. Intestinal pressure was +2, but after freely puncturing the intestine and allowing the gas to escape, the intrapleural pressure became −3·5. It would appear, therefore, that distension of the bowels has some effect on intrapleural pressure. This may account for the curious changes of pressure very occasionally found at refills. For example, in one case the patient was having 800 c.cm. of air every three weeks,

the pressure being taken from about −8 −2 to +6 +10. At the fourteenth refill the pressure was −2 +4 and 500 c.cm. raised it to +15 +17. No liquid was seen by X-ray, and at subsequent refills the patient was able to take 700 to 900 c.cm. as previously.

It must be remembered, however, that such changes in pressure are almost invariably due either to the presence of an effusion or to extension of adhesions diminishing the pneumothorax space. The development of dry pleurisy may cause a high intrapleural pressure, although no liquid forms. Perforation of the visceral pleura often causes a very high intrapleural pressure. In one of my cases the pressure after perforation was +12 +29, and it was reduced to −6 −1 by removing 1,200 c.cm. of gas.

It has been stated (7) that for a short time after a refill before the gases in the pleural cavity are balanced by interchange with the gases comprising alveolar air, the actual volume of gas in the pleural cavity is increased. This would raise the intrapleural pressure and be one explanation of the dyspnœa not infrequently seen some three or four hours after a refill. It is not my experience, however, to find this rise of pressure.

In some cases, when there was but little collapse before the refill, or more especially at the original induction of pneumothorax, the visceral pleura is pierced and a little air escapes from the lungs; this may continue for some time after the refill and so increase the quantity of gas in the pneumothorax cavity and raise the pressure. I do not think, however, that this is of common occurrence, for any slight puncture of the lung usually closes up at once.

It has often been stated that alteration of the intrapleural pressure on one side of the chest affects that on the other, but this is only the case after a certain quantity of gas has been introduced, and it also depends largely on the mobility of the mediastinum. If the intrapleural pressures are taken on both sides of the chest during a refill, it will be

found that on the treated side the pressure gets higher and higher as the gas enters the pleural cavity, but that it does not alter on the other side until a certain amount of gas has entered, and then it will gradually rise as more air is introduced.

In one case of left artificial pneumothorax the intrapleural pressures were as follows :—

Amount of gas.	Left side.	Right side.
0 . . .	−6 −2 ..	−5 −3
600 c.cm. . .	+2 +5 ..	−5 −3
800 „ . .	+6 +9 ..	−5 −3

In another case of right-sided pneumothorax the following intrapleural pressures were obtained during a refill :—

Amount of gas.	Right side.	Left side.
0 . . .	−6 − 4 ..	−8 −6
200 c.cm. . .	+3 + 9 ..	−8 −6
300 „ . .	+4 +12 ..	−8 −6

Another factor affecting intrapleural pressure is altitude, and this should be remembered in giving refills to those about to go to Switzerland. It is my practice to give no refill to a patient after one full week before he goes to the Alps and then to leave an intrapleural pressure considerably lower than normal. I have known patients soon after a refill arrive at an altitude of some 5,000 ft. with great dyspnœa, necessitating removal of some gas from the pneumothorax cavity. Riviere (14) said that a rise of 3,250 to 5,000 ft. is equivalent in a full pneumothorax of 3 or 4 litres to the addition of 400 to 800 c.cm. of gas. It has been estimated that every 1,000 c.cm. of gas in the chest increase by 40 c.cm. for every 1,000 ft. rise of altitude.

Adhesions. It is important to distinguish between adherent pleura and a pleural adhesion, the one may be compared to a ship aground and the other to a ship at anchor. In the one case it is very dangerous to try to separate the adherent surfaces, for the lung will almost certainly be torn,

in the other it is not difficult to divide the adhesions. Fig. 18 shows adhesions which may be easily divided. Fig. 19 shows one with lung tissue extending into it but not as far as the visceral pleura and it could be safely cauterised. In Fig. 20

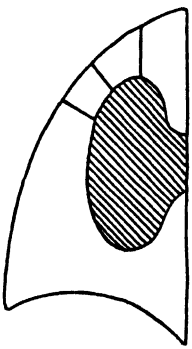

FIG. 18.

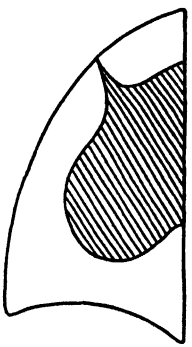

FIG. 19.

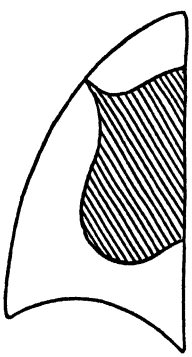

FIG. 20.

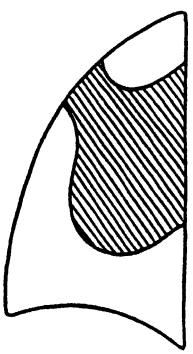

FIG. 21.

the lung tissue is seen extending to the visceral pleura and the adhesions may be enucleated but should not be cauterised. Fig. 21 shows adherent pleura.

One should never assume that there are extensive adhesions without trying to collapse the lung. I have succeeded in obtaining a good collapse in cases with much displacement

of the heart and trachea, and others with a history of repeated pleurisy or pleural effusion or conditions which would make the existence of extensive pleural adhesion most probable. There are many signs which make one feel confident that a pneumothorax will be impossible and one is generally right, but it is a mistake to discard the possibility of inducing one without trying. If there is too much adherent pleura to allow an efficient collapse the pneumothorax should be abandoned unless it is possible to convert it into a satisfactory one by dividing adhesions.

In some cases only a small pocket is found and I think it is wrong to keep on refilling it. If the adhesions are firm it is useless to do so, if they are not, it is dangerous, for there is always a chance of it rupturing into the lung, of surgical emphysema, or some other complication. The following cases are examples of such extensive adherent pleura that the pneumothorax treatment should be abandoned.

1. Male aged thirty-six. No free pleural space found at the first attempt. At the second a pocket was found with the pressure −4 −0, but rising to +19 +23 after 150 c.cm. of air had been injected. On the following day a third attempt was made in a different place and failed, but at the fourth attempt a space was found with a pressure −6 −2, but it was raised to +8 +12 by 100 c.cm. of air. No further attempts were made.

2. In this case there was practically no collapse and no benefit to the patient after ten days, so the treatment was stopped. The pressures were as follows :—

August 15th	−6 −8	300	0	+ 2
„ 16th	−4 −8	300	+ 6	+ 8
„ 17th	0 −1	200	+10	+12
„ 19th	−1 +1	300	+12	+14
„ 22nd	0 +1	300	+16	+20
„ 25th	+1 +8	200	+18	+20

When the adherent pleura is not sufficient to prevent

a useful degree of collapse the pneumothorax should be maintained as long as it controls the symptoms. If, however, the adhesions prevent the collapse of a cavity and cannot be cauterised or if the symptoms persist it is best to abandon the refills and treat the patient by prolonged rest or by thoracoplasty. It is bad practice to maintain an inefficient pneumothorax. If it is near an uncollapsed cavity it is especially dangerous as the cavity may rupture into it. Sometimes the symptoms are controlled in spite of gradual obliteration of the pneumothorax cavity as in the following case.

A woman, aged twenty-eight, had tuberculosis with slight pyrexia, signs of toxæmia and sputum containing tubercle bacilli. A pneumothorax was induced, but the upper third of the lung was found to be adherent and collapse of the base alone was possible. The temperature, however, subsided, her general condition improved and the quantity of sputum lessened so the treatment was continued. Gradually, however, the pleural space became smaller and smaller, so that less gas could be given at each refill and the pressures were higher. The treatment was kept up for eight months and had to be stopped then owing to complete obliteration of the pneumothorax cavity. She had, however, gained weight, and lost her toxic symptoms. Four years later she was keeping at work with signs of fibrosis, but apparently no active disease and in this case the pneumothorax was undoubtedly the turning point in the patient's illness.

Sometimes, however, as the lung is drawn out, the symptoms return and, in this case, a thoracoplasty should be performed if the condition of the other lung permits. In order to prevent this gradual obliteration of the pneumothorax cavity, frequent refills have been given in the endeavour to keep the intrapleural pressure high. It has already been pointed out that air under pressure in the pleural cavity is quickly absorbed and the pressure falls, so that the gradual obliteration of the pleural cavity proceeds in spite of large

frequent refills. It has been suggested that the obliteration may be delayed by injecting liquid into the pleural cavity instead of air. Some liquids, such as normal saline, are absorbed too quickly for this purpose, others set up an inflammatory reaction in the pleura which tends to hasten, rather than slow, the process of obliteration. The liquid most commonly used is a 5 per cent. solution of gomenol in olive oil. Chandler (18) uses sterile olive oil and says there is no need to add gomenol which may act as an irritant. In his opinion there are two essential conditions necessary for a successful oleothorax. One, a rigid mediastinum and the other a pleural cavity of limited capacity, and so a commencing obliterative pleurisy is an ideal condition for oleothorax because the mediastinum is fixed, and the pleural cavity limited and steadily becoming smaller. As obliteration proceeds the oil is pushed to the upper part of the pleural cavity and exerts its effect just where it is most needed in apical cases. It may keep up a selective apical collapse for years. In these obliterative cases where the alternative is thoracoplasty, oleothorax is clearly the lesser danger. The objection to oleothorax is that when under pressure the oil may break through into the lung with serious results. Plate XXXVII. shows the beginning of obliterative pleurisy and Plate XXXVIII. shows the same case with a small apical pneumothorax maintained by oil.

Adhesions will sometimes become stretched during pneumothorax treatment, and this may be helped by frequent small refills rather than by large ones and by encouraging the patient to lie on his healthy side as much as possible, as this will produce a drag on the adhesions. In my opinion these methods of stretching them should be given a good trial before resorting to surgical means. Open operation and division of the adhesions is dangerous, and I have seen two patients who died shortly after such an operation, one from hæmorrhage and the other from shock. If the adhesions are to be divided it is best to do so with the cautery under

direct vision through the thoracoscope. This method was described by Jacobæus in 1913 (19). After the exact position of the adhesions has been determined by X-ray, and a refill has been given to collapse the lung fully, a special trocar and cannula are inserted into the pneumothorax cavity. The cannula is made with a valve so that on withdrawing the trocar, no air can escape from the pleural cavity. After withdrawing the trocar a thoracoscope is introduced through the cannula, and a view of the adhesions can then be obtained. On examination through the thoracoscope the collapsed border of the lung will be seen, and leading from it adhesions pass outwards to the chest wall. Some of these are string-like and can easily be divided with the cautery, others are thick with a broad attachment to the chest wall, contain lung tissue, and cannot safely be divided, others again appear to have lung tissue in them as they leave the border of the lung, but not at their attachment to the chest. This last type is the one which most frequently holds up a large portion of the lung and is, therefore, the one in which it is especially important to beware of cutting through lung tissue. If there is any doubt as to whether or not there is lung tissue in an adhesion, no attempt should be made to divide it.

Plate XXXIX. shows a right pneumothorax with the lung and a cavity held out by an adhesion to the axilla. Plate XL. shows the condition with an efficient pneumothorax after cauterisation of the adhesion.

Although it is often possible to convert a bad pneumothorax into a good one by dividing adhesions, the operation is not infrequently followed by an obliterative pleurisy so that the pneumothorax closes as a result of adherent pleura at the base, spreading upwards. It is in this type of case that oleothorax is especially valuable.

The most serious complication is pyothorax, which usually results from injury to the lung and opening up a caseous focus. Lung tissue may be found in an adhesion the size

of a pencil. It often happens that there are several small string-like adhesions, as well as one or more large ones. If the large ones are divided and the lung is held by the small ones, they may stretch or snap. During cough there is a great chance of one of these small adhesions breaking, and if the tear takes place at its junction with the lung, pyothorax may follow. It is therefore important first to divide all the small adhesions before the bigger ones. Hæmorrhage into the pneumothorax cavity is another complication, but this can largely be prevented by coagulation with the diathermic cautery before dividing the adhesions. Provided proper local anæsthesia is used, the proceeding is painless, and it is safe if only the small string-like adhesions are divided, but this is rarely enough to free the lung, and it is when the larger adhesions are tackled that the dangers arise.

Maurer (20) enucleates the adhesion by cauterising the parietal pleura round it, having first anæsthetised it with a solution of novocain injected through a long needle under vision through a thoracoscope. Thus there is no chance of injuring lung tissue, and by this method he has considerably reduced the chance of complications. Only one of 130 cases treated by him developed pyothorax, and there were no deaths either from direct or indirect causes. In order to prevent hæmorrhage, he uses diathermy to produce a narrow zone of coagulation around the adhesion, and he then cuts through this zone with the galvano-cautery. Chandler (21) describes a diathermy apparatus which he uses in severing adhesions. A suitable portion of the adhesion is coagulated and then the coagulated portion is cut. He points out the necessity of localising the insertion of the adhesion before putting in the thoracoscope. This he does by watching the adhesion under the screen; as the patient is rotated the adhesion will appear to become longer or shorter, according to whether it is inserted in front of or behind the mid-axillary line. His thoracoscope consists of a combined endoscope and cautery, so that only one puncture is necessary.

He has described (22) the results of a series of operations for cutting adhesions (internal pneumolysis) on eighty-nine patients—

Good health	47
Fair „	11
Poor „ or relapse	6
Ill	3
Dead	15
Lost sight of	7

Effusion occurred in 24 and of these 20 recovered. Empyema followed in 7 cases and was tuberculous in 3, mixed in 3 and staphylococcal in 1. Hæmorrhage occurred in 15 and was always from the parietal stump. It was never severe.

Mac Dermott (23) states that in certain cases of spontaneous pneumothorax the tear in the visceral pleura is held open by an adhesion and will heal if the adhesion is cauterised.

Dangers of Inducing Artificial Pneumothorax. Woodcock (17) wrote in 1915: "There are dangers in connection with the production of artificial pneumothorax, but the greatest —and about this let there be no mistake—is the neglect in which it is held."

To-day collapse therapy is far more widely employed, and its value is increasingly recognised, but a few years ago it was necessary to find where the patient lived and whether the refills could be continued at home before starting to collapse the lung, and even now it is sometimes difficult to arrange for a patient's refills after he has left the hospital or sanatorium.

It is true that when pneumothorax is started the patient is embarked on a course of treatment which will probably last three or more years, but during most of this time he will be able to work and with much more safety with a collapsed lung.

The actual dangers of inducing a pneumothorax under

proper precautions are negligible, but unfortunately two of them, namely, gas embolism and pleural shock, though both extremely rare, are very serious and often fatal.

Gas Embolism. Air may enter the pulmonary veins and, if some bubbles are carried to the coronary vessels causing embolism of the capillaries, sudden death may occur. Hemiplegia or other forms of paralysis may result from gas embolism of the cerebral vessels. Transient blindness or blanching or marbling of the skin are examples of focal embolism.

In one case the patient became semi-conscious for a few moments and afterwards for several hours had partial hemianopia and hemiparesis.

In another, during induction of pneumothorax, the needle was felt to pass the pleura, but no manometer movements were recorded. Oxygen was introduced and the patient at once complained of severe præcordial pain and numbness of one arm. Convulsive movements and unconsciousness followed and the heart and respiration stopped. Recovery soon occurred after artificial respiration and stimulants had been applied, but there was facial paralysis for a few hours. The patient was well the next day.

Another patient, a woman of thirty-two, had been under treatment for nearly two years, and the pneumothorax cavity was practically obliterated. The needle was pushed into the lung, for she afterwards coughed up a little blood and the oscillations of the manometer just above and below zero were characteristic of the swing when the needle is in a cavity or lung tissue. At the time, however, it was thought that the needle was in the pneumothorax cavity and some air was introduced under pressure from the apparatus. After about 200 c.cm. had been given she suddenly became faint, complained of pins and needles in the legs and inability to breathe. She then actually lost consciousness for a few moments, but was quickly restored by stimulants. On

recovery she complained of weakness and a feeling of numbness in the right leg, but she had quite recovered by the next morning.

Death from air embolism is rare, but if it does occur it is usually quick. Recovery is the rule if the patient survives fifteen minutes (24). Death may be delayed, however, especially if a vessel remains open and small quantities of air are continuously being sucked into it. The vessel may be held open as a result of a tear in the pleura and this, I think, was the cause of death in the following case: A woman of thirty-six had a small selective pneumothorax which collapsed the upper third of the right lung. She had had refills for several years and after one (−7 −3 400 c.cm. −2 +1) which caused her no discomfort she had dined and gone to a theatre. During the performance she had pain in the right side and later developed intense vomiting. The next day she had less pain and vomiting but began to get tingling in the arms and legs, and dimness of vision. A needle put into the pneumothorax cavity showed a negative pressure and no blood was found. Breathing became sighing and she died forty hours after the refill. Autopsy showed torn pleura below the pneumothorax and about a pint of blood between its layers. There was no blood in the pneumothorax cavity.

It is often difficult to distinguish gas embolism from pleural shock, and it has been suggested that the great majority of cases described as shock are, in reality, due to embolism, but with modern methods it is very unlikely. Before the manometer was used in pneumothorax work, the needle was gently pushed into the chest until gas flowed from the apparatus, when it was assumed that the needle was in the pleural cavity. It was occasionally in lung tissue or a blood vessel, however, and if the gas entered the pulmonary veins embolism was liable to occur. By using a manometer and not allowing any gas to flow under pressure until it is certain that the needle is in the pleural cavity, gas embolism can

usually be prevented, and, indeed, it has been truly said to belong to the historical period of pneumothorax therapy. Stivelman (25) found no case of gas embolism amongst 867 cases treated by nineteen American doctors.

It has been suggested that air may enter the pulmonary veins from the alveoli when they are punctured. In cases of chronic pulmonary tuberculosis there is free anastomosis between the bronchial (systemic) and pulmonary veins, so that gas entering any intrathoracic vessels may find its way to the systemic circulation. This is not likely, however, or must be extremely rare as most gas which got into the systemic veins would pass through the right side of the heart or be blocked by the pulmonary capillaries. It is, therefore, only the pulmonary veins that are important in the matter of embolism unless the quantity of gas is very large. These contain arterial blood, so that it is not necessary to use oxygen when inducing a pneumothorax in the belief that it will be more readily absorbed by the blood and so less likely to produce gas embolism. The use of carbon dioxide has also been advocated at the initial induction of pneumothorax because it is quickly absorbed by the plasma, but it is doubtful whether this makes any practical difference and accident should be prevented by correct technique. If air enters the systemic veins, for example, during operations on the neck, a hissing sound may be heard and soon a " water wheel " murmur over the heart due to churning in the right ventricle. Dyspnœa, cyanosis and coma occur.

In rare cases air may be liberated from solution in the blood as in caisson disease. It was found that 80 c.cm. of air could be put into a vein of a dog without ill effect, but 150 usually caused death from gas in the right ventricle. In the arteries or pulmonary veins, however, a very small quantity might produce death or serious symptoms and, owing to the negative pressure of about −15 cm. water in the pulmonary veins, embolism is not unlikely if they are torn, especially in fibrotic cases where retraction of the vein is prevented.

Symptoms vary according to the quantity of air and its rate of introduction.

If the needle enters a big vessel blood is forced up the tubing and can be seen at the glass window. Any blood entering the needle quickly clots and blocks the needle. When the pleura is adherent the needle is often pushed, and sometimes deeply, into lung tissue, but no gas can enter the circulation unless it is under pressure. In order to prevent gas embolism, the following precautions should be taken :—

1. Allow the gas to be sucked in very cautiously and use no pressure until the movements of the manometer make it certain that the needle is in the pleural cavity.

2. The needle and tubing should at first be connected only with the manometer, being clipped off from the gas chamber.

3. Use a large and blunt-tipped needle for the initial induction of pneumothorax.

4. Coughing or deep breathing should be prevented, at least until the needle is in the pleural cavity and the gas flowing freely.

Pleural Shock. This rare accident may occur when the pleura is punctured or irritated or even when the pleural cavity is being washed out. I have known a case where the patient suddenly died when the chest was punctured for the purpose of aspirating a pleural effusion, and another where sudden death occurred whilst B.I.P. was being introduced into an empyema sinus. With animals it has been found that shock can be produced by irritation of the pleura and probably occurs through the vagus. It can be prevented by ligaturing the carotids, or by keeping the animal under an anæsthetic. Some have denied the existence of pleural shock, claiming that the symptoms are due to embolism, and there is no doubt that they are very similar in the two conditions. But apart from all other reasons, the fact that shock is liable to occur in the same individual, whenever the pleura is punctured, must exclude gas embolism as the cause of all cases.

The symptoms vary from a mere faintness to actual death and usually occur as the needle is entering or leaving the pleural cavity, but they may come on during a refill or even some minutes after it is finished. It is said to be more common in the early stages of disease, when the pleura is healthy, than in cases of advanced tuberculosis, and this is one reason why Riviere (14) did not advocate artificial pneumothorax in the treatment of early pulmonary tuberculosis. Pleural shock is rare, and it is not easy to determine whether or not it is less unlikely to occur in cases where there is healthy pleura, but it certainly may occur when the pleura is considerably involved, even in cases of chronic empyema sinus. The following cases are examples of pleural shock :—

1. Stivelman (25) describes the case of a patient with pulmonary tuberculosis who had a fibroma removed from his arm under cocaine and adrenalin on February 1st, and there was no ill effect. On February 20th an attempt was made to induce an artificial pneumothorax and after giving an injection of morphia, ¼ gr., the pleura was anæsthetised with 1·5 c.cm. of a 0·75 per cent. solution of cocaine and adrenalin 1 in 8,000 but before the hypodermic needle was withdrawn the patient collapsed, the pulse was only just perceptible, there was stertorous breathing and the patient became livid. Stimulants were given and he recovered after an hour and a half. Two days later he had a similar but worse attack, whilst the pleura was being anæsthetised, and went completely blind for four hours. Photophobia lasted twenty-four hours, but he was quite well in two days' time.

2. In one of my cases (26) the patient, a man of thirty-eight was very ill with tuberculosis which had followed pleurisy. The whole of the left lung was involved. He was given an injection of morphia, ¼ gr. half an hour before the operation. The track of the needle and the pleura were anæsthetised with a 2 per cent. solution of novocain, and an attempt

was made to induce a pneumothorax from the left axilla, but it failed. A second attempt was made at the back after anæsthetising the pleura as before, but again the pleural cavity could not be found. I was just about to withdraw the needle when the patient suddenly said he could not breathe. There was no movement of the manometer as would have occurred had air been sucked into the blood stream, for the tube leading to the air chamber of the apparatus was clipped and the only air which could have been sucked in was that contained in the tube between the needle and the manometer. I withdrew the needle at once and the patient sat up, gave a few gasps and died. There was no post-mortem examination.

3. In another of my cases, a woman of twenty-eight, pleural shock occurred, but not the first time the pleura was punctured, and, indeed, in this case the pleura seemed to become more sensitive as treatment progressed. During the third refill she complained of a sudden feeling of giddiness. After the needle had been removed following the fourth refill she had a sudden pain over the heart, could not breathe and lost consciousness for about two minutes. During the fifth refill, when about 150 c.cm. of air had been given, she suddenly gasped, became blue and almost pulseless. The needle was withdrawn and stimulants administered and she recovered without having actually lost consciousness. After this treatment was discontinued.

It is doubtful whether anything practical can be done to prevent pleural shock. It is said not to occur in animals under a general anæsthetic, but the danger of complications from an anæsthetic is obviously greater than the risk of pleural shock. A preliminary injection of morphia had been given in the only fatal case in my series and also in several of the other published cases. I always use novocain to anæsthetise the pleura as well as the skin, so that the puncture is absolutely painless, but in spite of this shock has occasionally occurred in some of my cases, and Stivelman (25)

found pleural shock equally frequent whether a local anæsthetic had been used or not in a series of 867 patients treated by nineteen American doctors. At the Royal National Hospital for Consumption, Ventnor, Hempson advises giving an injection of atropine before inducing pneumothorax. A series of cases treated with certain technique and without pleural shock does not indicate that shock can be prevented by following that method, for it is extremely rare whatever technique is used.

Puncture of Lung. If the visceral and parietal pleura are adherent it often happens that the needle passes straight through into the lung. In this case a negative pressure may be registered on the manometer, but the oscillations are just above and below zero, so that the mean pressure is 0. The needle usually becomes blocked with clotted blood. A little bloodstained sputum may follow this accident, but there are no other ill effects.

If the pleura is not adherent, it is usually possible to avoid piercing the visceral pleura by being very gentle and using a blunt Riviere needle for the initial operation. In some cases when the lung is punctured air may escape and lead to an increased pneumothorax cavity, and possibly a higher intrathoracic pressure and dyspnœa some hours after the needle has been withdrawn. Usually, however, a small puncture quickly closes and no air escapes from the lung.

Pneumoperitoneum. Direct pneumoperitoneum may occur as a result of the needle being inserted through or below the diaphragm into the peritoneal cavity. It is curious that, as in the pleural cavity, the pressure is lower during inspiration than it is during expiration. There may be no symptoms or only slight abdominal discomfort, but sometimes there is severe pain in the chest and shoulder which is worse when the patient is in the erect position. X-ray shows air below the diaphragm when the patient stands up. In rare cases air may pass from an established pneumothorax through the diaphragm and produce an indirect pneumoperitoneum.

Banyai (27) has described cases in which this has occurred. The symptoms are relieved by raising the foot of the bed and shifting the air to the pelvis.

Simon and Abrams (28) have recorded a case in which air got between the diaphragm and the diaphragmatic pleura on both sides in a patient being treated by left artificial pneumothorax. The only symptom was pain in the lower part of the chest after refills.

Punctures of a Large Vessel or of the Heart. Minor (29) was inducing a pneumothorax in a young woman with an old fibroid lesion. Suddenly she gasped and turned pale and blood rushed up the tubing as far as the gas chamber. The needle was at once withdrawn and there were no ill effects.

In one of my patients pneumothorax treatment had been stopped two years previously, but symptoms had returned and I was trying to re-collapse the lung. Suddenly the manometer registered a positive pressure which gradually increased and blood began to flow up the tubing and was visible at the glass window beyond the needle.

In two other cases a similar occurrence took place, but in none of these was there any pain or discomfort at the time, or any subsequent ill effect.

Pearson (30) has described a case in which the needle entered the pericardium. The manometer showed negative oscillations synchronous with the heart. There were no ill effects.

Surgical Emphysema. Superficial emphysema is not uncommon, especially if there is a high intrapleural pressure, or if the patient coughs during the refill. It is, however, quite a painless and harmless complication and quickly clears up. Deep emphysema is caused by the air entering a false passage between the pleural layers. It travels to the neck along the trachea and œsophagus, and is accompanied by pain and severe dyspnœa. There may also be difficulty in swallowing and a sensation of choking. In three cases in my series the emphysema was severe, but eventually

subsided without leaving any harmful after effect. In one patient two attempts to induce an artificial pneumothorax had failed owing to adherent pleura, and twenty-four hours after the attempt the patient complained of pain across the chest, and some surgical emphysema was found in the chest and neck. Two days later it had spread all over the chest and abdomen. Both arms and hands became affected and emphysema could be felt at the very tips of the fingers. The neck, face and scalp became enormously swollen, and he could hardly see out of his eyes. The whole body was distended with surgical emphysema, from the top of the head to Poupart's ligaments, but below this there was no emphysema at all. The patient looked most uncomfortable, and his appearance was alarming, but except for a slight choking feeling he complained of no discomfort. There was no pain or pyrexia, and the emphysema, which began to subside after the third day, had completely disappeared in a week.

In mild cases no treatment is required but if cough is troublesome it is liable to increase the emphysema, and should be checked with morphine or a linctus. In severe cases the skin should be punctured or an incision made at the site of the thoracic puncture.

Bourgeois (81) states that although surgical emphysema is usually a slight complication, severe and even fatal cases have been described. These asphyxiating cases generally occur when there is a tear in the lung and adherent pleura. The emphysema begins some five hours after a refill. The patient has bouts of cough, the emphysema may spread rapidly to the face and head, arms, fingers and down the trunk as far as Poupart's ligaments. In very severe cases cyanosis and dyspnœa appear and lead to death from asphyxia. In these rapid and asphyxiating cases he advocates making an incision down to the pleura at the site of the puncture. He agrees that multiple punctures of the skin of the neck may relieve the symptoms, but he does not

regard them as sufficient to cure the condition. He has seen a case when the cyanosis and dyspnœa caused by surgical emphysema was followed by an acute and fatal exacerbation of the tuberculosis in the lung.

Pain. The treatment itself should be quite painless if the skin and parietal pleura are properly anæsthetised. Even when inserting the large cannula for a thoracoscope there should be no pain. Patients are usually apprehensive at first, but on finding that they feel nothing, do not dread the subsequent refills. Some patients are much more sensitive than others, and in two cases I have had to stop the treatment as the patient could not endure the refills. One, a man of forty, described the procedure as agony; he clenched his fists during a refill and beads of perspiration came on his forehead although he was always given a preliminary injection of morphia. Of course, if the local anæsthetic is not given properly or if the needle is inserted diagonally so that it pierces a portion of the pleura which has not been anæsthetised there will be pain, but some few patients complain of pain and undoubtedly feel it at every refill, to which they never become accustomed. In many cases there is a large nervous element; in one case I had removed the needle after a refill and was holding a pad of wool over the site of puncture, as there was a little bleeding. The patient said the pain was so severe he could not bear it any more, and asked me to stop the refill. I said, "Certainly, I have just finished," and, pinching the skin, removed the pad as if taking out the needle. I then held another pad over the chest, but told him it was only wool, and that the needle was out. He said the pain had gone. In this case he could not feel the needle being withdrawn, he did not even know whether or not it was in the chest, yet he had a feeling of pain amounting to what he called agony as long as he thought it was in the chest. Whether genuine or imaginary, pain does sometimes occur to such an extent that the treatment cannot be continued, though in most cases if a

refill is given gently and quickly and the track of the needle is thoroughly anæsthetised there is no pain at all.

After the initial induction the patient sometimes complains of a sensation of friction which is worse on movement and generally disappears if he keeps still. In rare cases this develops into a severe pain for which it may be necessary to inject morphia.

Pain after a refill may occur, and it varies from a mere feeling of pressure to an acute pain. A really acute pain is usually due to rupture or tearing of some pleural adhesion, and a sharp pain on cough may be due to stretching of adhesions. A feeling of pressure or discomfort is often due to an excess of air in the pneumothorax cavity, and is generally the result of giving too big a refill. Sometimes, however, extra air gets into the pneumothorax cavity from the lung which has been accidentally punctured during the refill, or according to Rist and Strohl (7) there may be a temporary increase of gas in the pleural cavity before the balance is established between the alveolar air and the gas introduced.

One may recognise three types of pain following artificial pneumothorax refills :—

1. The patient first complains of tightness in the chest which lasts for a few hours after a refill. It occurs only in patients with adhesions and a high intrapleural pressure, and it wears off as the pressure falls.

2. A pain which comes on some hours after the refill and is increased by cough or deep inspiration. This is often referred to the shoulders and usually to where X-ray shows the presence of adhesions.

3. The sudden snapping of an adhesion. This may occur for no apparent reason, or with cough or during a refill. In one of my cases the patient was a man who had been under treatment for eight months. He was doing very well, was not at all nervous and had never complained of pain before, but during one refill, after about 500 c.cm. of air

had been given he suddenly complained of a sharp pain in the chest as if something had torn. It lasted a few seconds only and I was able to finish the refill. It had no effect on the intrapleural pressure. The patient compared the pain to that felt when the dentist touches a nerve. It did not recur at any subsequent refill and was possibly due to the rupture of some small adhesion.

In several cases in my series the patients felt something give inside them, but there was no actual pain. Indeed, in one of these cases the patient had a feeling of tightness and pain which was relieved at once after the sensation of something giving way in her chest.

A sudden pain accompanied by dyspnœa may result from ruptured lung, that is the occurrence of spontaneous pneumothorax in an artificial one. Dyspnœa is usually the chief symptom, and there may be no pain, but in some cases the pain is so severe that it simulates a perforated gastric ulcer.

Pain occurring before a refill is usually due to pleurisy, the expansion of the lung allowing the visceral and parietal pleura to come into contact. It should be taken as a warning and refills given at shorter intervals or adherent pleura will probably result.

Lastly, pain may rarely be due to deep emphysema and is then felt under the sternum and in the throat. It comes on a few hours after the refill and is accompanied by a sensation of choking and severe dyspnœa. It is very unusual, however, for actual pain to occur in cases of surgical emphysema.

Dyspnœa. Shortness of breath occurring during pneumothorax treatment may be due to over-collapse of the lung. In cases where one lung is much diseased or collapsed, a pneumothorax on the other side may lead to severe dyspnœa. It is surprising, however, how much collapse in both lungs may occur with little or no dyspnœa, though it is obvious that the degree of collapse which can be obtained

in one lung must depend on the amount of functioning lung on the other side. If a large pneumothorax is produced so that the mediastinum is much displaced there may be great shortness of breath, even if the other lung is healthy.

Dyspnœa may also occur when the intrapleural pressure is too low. It may be very severe after or during aspiration of pleural effusion or when air is removed from the pneumothorax cavity. Sometimes when the lung is re-expanding dyspnœa occurs and is relieved by giving a refill.

Dyspnœa may occur about three hours after a refill, and it has been suggested that this is due to an increase in the size of the pneumothorax owing to interchange between the alveolar air and the gas in the pleural cavity. It is doubtful, however, whether there really is any temporary increase in the quantity of gas, and if it does occur on some occasions it is more probably due to accidental pricking of the visceral pleura and escape of alveolar air. This type of dyspnœa is largely nervous in most cases, it starts as a little shortness of breath which alarms the patient and makes him more conscious of it until sometimes he is worked up into a state when he is gasping for breath. Reassurance or any medicine given for its psychological effect will usually relieve the symptoms at once, though occasionally it is necessary to give an injection of morphia.

Displacement of the Mediastinum. Sometimes the mediastinum is resistant and firmly held in position, so that there is no displacement, even with high intrapleural pressures and complete collapse of the lung. In other cases it is extremely mobile and is displaced even when the intrapleural pressure is negative. In such a case it may be necessary to discontinue the treatment. Phrenic evulsion has been advocated in the belief that the raised diaphragm will prevent bulging of the mediastinum but it may make matters worse by adding a loose diaphragm. Thoracoplasty also is dangerous for the patient is likely to die of mediastinal flutter. The best treatment for a flapping mediastinum is

to induce adhesions between the lower part of the lung and the chest wall and so anchor the mediastinum. In order to do this 20 c.cm. of a 50 per cent. solution of glucose may be injected into the pleural cavity and repeated at weekly intervals increasing the quantity up to 150 c.cm. if necessary. This leads to the development of an effusion and subsequent adherent pleura. Tincture of iodine has been suggested instead of glucose and I have injected as much as 10 c.cm. of a 10 per cent. solution of iodine into the pleural cavity without ill effect. There may however be pain and even the glucose injections occasionally cause so much pain that morphia has to be given. Dumarest (15) advocates gomenol in paraffin and refers to the treatment as *pleurésie provoquée*. He points out that it is impossible to foretell how serious the pleural reaction may be and a purulent effusion may result.

In most cases the optimum degree of collapse can be reached without undue displacement of the mediastinum, but even when there is some displacement the patient may steadily improve when the lung is fully collapsed and it is not necessary to interrupt the treatment simply because of a weak mediastinum. Generally, however, symptoms occur and the pressures have to be reduced. A common symptom is loss of weight as illustrated in the following two cases :—

1. A girl of seventeen was admitted to hospital with infiltration of the upper third of the left lung. Before artificial pneumothorax was induced, even whilst she was being treated with absolute rest in bed, the evening temperature was 99·6° to 100·5° F. A left artificial pneumothorax was induced on July 18th, 1921, and Table 25 on p. 274 shows the changes in the patient's weight.

On October 20th X-ray showed good collapse of the left lung with slight displacement of the mediastinum to the right. The patient was up for six hours a day and had a normal pulse and temperature. The weight continued to drop, however, and in November the patient was put back

TABLE 25

	st.	lb.		st.	lb.
July 12th, 1921	8	8	Nov. 28th, 1921	7	12¾
Aug. 9th ,,	8	9¼	Jan. 2nd 1922	7	9
,, 24th ,,	8	8¼	,, 24th ,,	7	9
Sept. 11th ,,	8	4½	,, 31st ,,	7	10½
,, 20th ,,	8	3	Feb. 14th ,,	7	12½
Oct. 20th ,,	8	0	,, 20th ,,	7	12
Nov. 7th ,,	7	12½	June 6th ,,	8	5½
,, 14th ,,	7	12¾			

to bed. This stayed the loss of weight for a time, but in January it began to fall again. At this time X-ray showed considerable displacement of the heart and mediastinum to the right, with bulging of the upper part of the mediastinum. No refill was given from December 30th until February 10th, when the mediastinum had come back towards its normal position. Refills were then given very carefully to prevent displacement of the mediastinum, and the patient began to gain weight.

2. Female aged twenty-one, with infiltration of the left apex. A right-sided pneumothorax was induced on October 27th, 1921, and the patient's weight varied as follows :—

TABLE 26

	st.	lb.		st.	lb.
Oct. 22nd, 1921	9	6¼	Feb. 14th, 1922	8	5
Nov. 14th ,,	9	7½	,, 27th ,,	8	5
Dec. 12th ,,	9	5½	March 13th ,,	8	5
,, 30th ,,	9	1½	,, 27th ,,	8	7
Jan. 9th, 1922	9	1	April 24th ,,	8	7½
,, 24th ,,	8	12	May 8th ,,	8	8
,, 31st ,,	8	8	,, 22nd ,,	8	8¾

There was the initial gain in weight which usually follows hospital feeding and rest, but this gave way to a steady loss. On January 30th the mediastinum was much displaced

and bulging to the left in the upper part, forming a pleural hernia. By lengthening the intervals between the refills and regulating the quantity of gas given (at the same time taking care that the lung did not re-expand and produce adherence of the two layers of pleura) the mediastinum returned to the normal position and the loss in weight at once stopped.

Pleural Hernia. Occasionally the pleura forms a hernia which protrudes through the mediastinum to the other side, or between the ribs forming a superficial tumour.

The common sites for bulging beyond the mediastinum are in the anterior mediastinum between the second and fourth sterno-costal articulations and in the lower part of the posterior mediastinum. These herniæ can be diagnosed only by X-ray, and cause no symptoms as a rule (Plate XLI). In one case I saw a pleural hernia in the fourth left intercostal space in the mid-axillary line. When the patient coughed there was a swelling about the size of a hen's egg in this situation. It could easily be reduced and the air could be heard and felt re-entering the pleural cavity.

Febrile Reaction. Formerly, when it was the custom to give a large quantity of gas at the initial induction of pneumothorax, reactions were common, but now it is rare to obtain even a mild one. At the same time, symptoms of toxæmia often disappear very quickly after a severe reaction, whereas, now that the lung is collapsed more gradually by small frequent refills, it takes longer for the temperature to fall and the symptoms to subside. The effect of reaction is discussed in Chapter X. under Shock Therapy.

Most authors think that reactions should be avoided, and prefer the gradual collapse of lung, and I agree that this is generally the best procedure. It is a mistake, however, to be bound by any hard and fast rule, and if the patient fails to improve with small refills, large ones should be tried and

will often do good in spite of reactions. Fig. 22 shows a series of reactions after refills of moderate amount, and it will be noticed that the temperature soon became level. A rise of temperature during treatment is usually due to

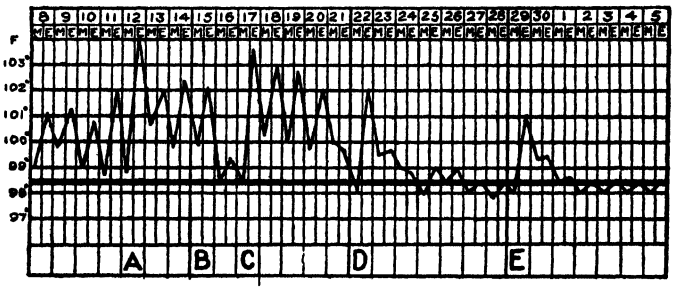

FIG. 22. A. Initial operation, 400 c.cm.; B. 1st refill, 550 c.cm.; C. 2nd refill, 600 c.cm.; D. 3rd refill, 600 c.cm.; E. 4th refill, 700 c.cm.

pleurisy, but a sudden febrile reaction may be the result of a tear in the lung. This is especially liable to happen as the result of a pull from an adhesion during cough. As a rule the tear soon heals and the temperature settles in a few days.

Infection of the Track of the Needle. A fistula may follow aspiration of a tuberculous empyema. Sometimes this will heal quickly with a little ultra-violet ray treatment, but often it persists and adds considerably to the gravity of the prognosis. The empyema should be aspirated repeatedly and the lung encouraged to re-expand. Morriston Davies (82) has had good results by injecting a 2 per cent. solution of methylene blue in absolute alcohol along the track of the sinus but it is sometimes painful.

Perforation of the Visceral Pleura. It sometimes happens that a rupture of the visceral pleura occurs and converts the artificial pneumothorax into a spontaneous one. This is a very serious complication if the perforation remains open and leads to a pyopneumothorax. The symptoms are

sudden pain in the chest accompanied by a rise in temperature which becomes hectic. The patient is acutely ill, an effusion soon forms and becomes purulent. The intrapleural pressure may be much increased if the perforation is valvular, and the patient will be very dyspnœic until some gas is removed. Breath sounds over the pneumothorax cavity are amphoric, metallic tinkling is heard, and there is usually much displacement of the mediastinum. Puncture of the lung which occasionally occurs during a refill very rarely leads to any serious consequences, for the small hole usually closes up at once. The rupture is most common where an adhesion joins the visceral pleura and a tear results from the sudden increase of intrapleural pressure during cough. Occasionally a thin-walled cavity or an area of caseating lung may rupture, and it is not uncommon for a pyopneumothorax to break through the visceral pleura and leave a gaping communication between the lung and pleural cavity.

Hutchinson (33) described a series of cases which he believes to be due to a slight rupture of lung producing an effusion, but soon healing. He remarked that an effusion is often preceded by a sudden rise of temperature which gradually falls and is very similar in type to that seen in ordinary spontaneous pneumothorax. Moreover, if the intrapleural pressure is taken in these cases, it is often found to be raised before any effusion has formed. This is a plausible explanation of the rise of intrapleural pressure, which undoubtedly does frequently precede the development of effusion in pneumothorax cases and there is no reason why a rupture should not occur and heal quickly, just as it may do in ordinary cases not treated by artificial pneumothorax. It is very different, however, from the sudden rupture with obvious signs and serious consequences, leaving an open communication between the lung and pleural cavity, and invariably resulting in pyopneumothorax. MacDermott (23) reports two cases where the perforation

was held open by an adhesion and closed when the adhesion was cauterised.

Of course, spontaneous pneumothorax may occur on the untreated side during collapse therapy and produce bilateral pneumothorax. This happened in two of my cases and both died.

In six of my first 334 cases of artificial pneumothorax (34) rupture of the visceral pleura on the treated side occurred. Two of these were non-tuberculous patients, and both recovered. The other four were cases of pulmonary tuberculosis and all died; a pyopneumothorax developed in each case. Of Forlanini's 139 cases, rupture of lung occurred in eight, and all died.

Burland (35) described nine cases. Of these three died within the first month and four in from three to six months.

It must, therefore, be regarded as a very serious complication, which not only justifies, but necessitates, drastic treatment. When the rupture occurs in a collapsed lung the perforation tends to remain open, whereas in an ordinary case of spontaneous pneumothorax the collapsing lung tends to close the perforation and allow healing to take place. The treatment, therefore, is usually that of spontaneous pneumothorax where the perforation does not heal, and is described in Chapter VI. It may here be summarised as follows:—

1. Relieve urgent dyspnœa by removing gas from the pleural cavity and, if necessary, perform intercostal drainage by leaving in the cavity a catheter connected with a rubber tube the lower end of which is in a bottle of water on the floor, thus allowing gas to leave, but not to enter, the pneumothorax cavity.

2. If healing does not occur examine with a thoracoscope to see if the tear is being held open by an adhesion which can be cauterised.

3. Clean out the pleural cavity by aspiration and, if necessary, by washing it out with 1 in 5,000 solution of

methylene blue. Solutions containing chlorine or other irritating substances must not be used if there is an open perforation in the visceral pleura.

4. Restore the general health as far as possible by glucose, tonics, fresh air, etc.

5. If the perforation remains open and the lung shows no sign of re-expanding, thoracoplasty should be performed early, as soon as the patient has recovered from the initial shock and when his general condition is good. The longer operation is delayed, the less will be the chance of a successful result.

Thickened Pleura. This may occur when there is no effusion, but is especially frequent in cases of long-standing pyopneumothorax. The collapsed lung may be so bound down by pleura that re-expansion is impossible, and in that case, whether there is an effusion or not, the lung is permanently useless and the pneumothorax cavity a source of possible danger, so it should be closed by thoracoplasty.

After pneumothorax treatment has finished, the visceral and parietal pleura are usually adherent and thickened, so that it is not possible to re-collapse the lung. This is not always the case, however, and re-collapse may sometimes be obtained even in cases where there has been pleural effusion. Vere Pearson (30) succeeded in reproducing a pneumothorax in one case two years after the last refill.

Pleurisy. A small lesion of pleurisy frequently leads to the pleural layers becoming adherent and the disease arrested. In cases of pneumothorax this cannot occur, since the visceral and parietal pleura are separated by gas, and consequently pleural effusion is a very common complication. Dry pleurisy may occur as a complication of artificial pneumothorax, and Riviere thought it was not uncommon at the site of puncture, especially if many punctures have been made at the same place. The symptoms of dry pleurisy are malaise, slight pain or aching in the chest, and there is often a little tenderness on palpation over the

inflamed pleura. If a refill is given it is found that less gas than usual is required to reach the former pressure. The treatment of such cases is to keep the patient in bed and the pain relieved by local warmth from a hot water bottle or electric pad. Veganin or aspirin and phenacetin usually give relief and Saugman believed that salicylates have a specific effect in these cases.

Sometimes intrapleural bodies are found either free or on a pedicle. They consist of coagulum of fibrosis and can only be recognised by X-ray which shows them as rounded or oval opacities and they are of no clinical significance.

Pleural effusion is the most frequent complication of artificial pneumothorax. There may be no symptoms or the patient may complain of malaise and slight pain. Sometimes there is vomiting, and the condition resembles an acute gastric disturbance. As a rule there is some pyrexia for a few days. After three or four days, however, the symptoms usually disappear, but occasionally the temperature remains high and the patient's condition becomes steadily worse. In these latter cases it is found that the tuberculosis is spreading in the lungs, and the development of the liquid seems to have been the starting-point of a general breakdown, though, fortunately, such cases are rare. The physical signs of effusion are obvious: shifting dullness can be demonstrated even if there is only a very small amount of liquid: the level of the liquid is horizontal, and there is no parabolic curve: vocal fremitus is absent over the liquid, but present over the air-space above it: breath sounds are usually absent, but often acquire a metallic quality, especially over a small zone just above the level of the liquid. The coin sound is usually well heard, and the succussion splash is one of the best physical signs, and can be heard even in the smallest effusions. The patient will often complain of feeling liquid splashing about in his chest.

X-ray shows the presence of liquid beyond doubt, but the patient must be examined in the upright position. A very

small effusion can often be detected only by X-ray examination. If examined in the upright position the effusion will obliterate the shadow of the diaphragm and it will be impossible to see the base of the lung which is often expanded under the effusion. Plate XLII. shows the base of the lung when the liquid is shifted by making the patient lie on his side. Plate XLIII. shows the base of the lung and the diaphragm with the liquid at the apex because the patient was standing on his head when the film was taken.

Various explanations have been given to account for the frequency of pleural effusion in cases of pneumothorax. Irritation by the gas, its temperature, the repeated punctures of the parietal pleura or the intrapleural pressure have all been described as playing a part in the production of effusion. Pisani and Smejkal (36) suggest that pleural effusion in these cases is due to the increased permeability of the capallaries from want of oxygen; to increased osmosis; to higher concentration of H-ions; to calcium deficiency of the pleura and lastly to the aspirating effect of a negative pressure. They suggest giving calcium just before and during pneumothorax treatment in the form of a 10 per cent. solution of calcium gluconate, and advise intramuscular injection of 10 c.cm. of this three times a week for the first two months, twice a week for the next two months and once a week during the rest of the treatment.

Dumarest (15) states that the incidence of effusion during pneumothorax treatment may be reduced by using a small calibre needle, avoiding high pressures and not giving refills to women during the menstrual period. In my opinion, however, these effusions are true tuberculous exudates due to tuberculous pleurisy, and I have found that they occur with equal frequency whether the gas is heated or not, and whether air, nitrogen, carbon-dioxide or oxygen is used, and in support of this view I would urge the following considerations:—

1. Pleurisy is a common complication of pulmonary

tuberculosis and often leads to adherent pleura. If the parietal and visceral pleura are separated, as they are in cases of pneumothorax, one would expect effusion to form when pleurisy occurred. In almost every case of tuberculous spontaneous pneumothorax effusion develops but it is extremely rare in non-tuberculous cases.

2. The cytology of the fluid is that of a tuberculous exudate and not of a transudate.

3. Tubercle bacilli are not found in the early stages in the effusion, but are almost invariably present in long-standing cases, and no other organisms have been found in any of my cases.

4. Of the first 309 cases in which I have produced a successful pneumothorax for pulmonary tuberculosis, a definite effusion formed in 128, but of my first fifty-four cases of non-tuberculous disease where pneumothorax was induced, an effusion occurred in one only, and that was only a transient and small effusion in a case of acute pulmonary abscess. A percentage of 41·4 for tuberculous and under 2 for non-tuberculous cases.

Hutchinson (33) thinks that effusion in tuberculous cases is often the result of a small rupture of lung, and supports his opinion by the facts that an effusion is often preceded by a rise in intrapleural pressure, and that the temperature which accompanies its development is very similar to that seen in ordinary cases of spontaneous pneumothorax.

A small transient effusion is almost the rule in cases of artificial pneumothorax, and if every case were examined daily with the X-ray screen it would be seen in most cases at some time or other as a small pool lying in the costodiaphragmatic angle. Such small collections of liquid, however, are soon absorbed and are not important. Here I include as cases of pleural effusion only those where it lasts at least a week and covers the diaphragm.

Pleural effusion is not a serious complication of collapse therapy and is often beneficial. It sometimes happens that

a patient, who is not doing well, starts to improve as soon as an effusion forms, and eventually makes a good recovery.

The following table shows the results of effusion on a series of 309 cases from my series. The cases are those with medium resistance who have failed to improve after other treatment; C1 included those with unilateral disease, C2 those with slight involvement of the other lung and C3 where there is involvement of a third or more of the better lung :—

TABLE 27

	C1.			C2.			C3.		
	Cases.	Died.		Cases.	Died.		Cases.	Died.	
			Per cent.			Per cent.			Per cent.
Total .	126	24	19·0	135	47	34·8	48	33	68·7
Clear fluid.	36	5	13·9	45	14	31·2	11	8	72·7
No effusion	78	13	16·6	72	22	30·6	31	22	71·0
Pus . .	12	6	50·0	18	11	61·8	6	3	50·0

From which it will be seen that the development of clear effusion does not increase the mortality, but that when the fluid is purulent the outlook is much more serious. When there is extensive disease the mortality is high in all cases.

Saugman (6) gave the following table :—

TABLE 28

	With effusion.		Without effusion.	
	No.	Per cent.	No.	Per cent.
Able to work . .	31	37·3	24	40·0
Unable to work .	4	4·8	1	1·6
Dead . . .	48	57·8	32	53·3
Ill or dead from other causes. . .	—	—	3	5·0

Here, too, there is very little difference between the dry cases and those with effusion.

The effusion usually develops in the first six months of treatment, and after twelve months it is rare.

The next table shows the number of months after the induction of artificial pneumothorax when the effusion formed in my series of 309 cases :—

TABLE 29.

1 to 3	months . . .	45 cases
3 to 6	,, . . .	43 ,,
7 to 12	,, . . .	29 ,,
13 to 18	,, . . .	5 ,,
Over 18	,, . . .	6 ,,

When liquid develops in the pleural cavity, a lung which was previously partially expanded will become completely collapsed. This is very constant, and is one reason why Hutchinson thought effusion in these pneumothorax cases was often due to ruptured lung and consequently increased intrapleural pressure.

In some cases, however, there is no rise of intrapleural pressure when the effusion forms and yet the lung becomes completely collapsed. It may be that the weight of the liquid coming into contact with all parts of the lung as the patient lies down or changes his position presses the air out of the alveoli whatever the intrapleural pressure may be. When the lung contains some air re-expansion is much easier than in the case of a lung that is completely airless.

The treatment of pleural effusion complicating artificial pneumothorax depends on many factors, and each case must be taken on its merits. It may be stated as a general rule that it is unwise to aspirate during the acute or febrile stage and that if pressure symptoms arise at this time it is better to relieve them by removing some air than by aspirating liquid. If the liquid is left it will usually cause obliteration of the pneumothorax cavity by pleural adherence and fibrosis, starting at the bottom of the pleural cavity and

working upwards. If this result is desired it should be left, but the patient must be kept under observation lest complications arise. Many cases of pneumothorax which terminate by obliteration in this way do very well, for, as the lung is drawn out, a satisfactory fibrosis develops over the diseased areas and the healthy parts of the lung begin to function once more.

In some cases the effusion does not become absorbed for months or even years or it may never be absorbed. If aspirated it reforms. These chronic effusions are usually localised collections of liquid surrounded by adherent pleura and the general health of the patient may be unimpaired. I have a patient who has had such an effusion for seven years and has kept at work and in good health all the time. About once a quarter I aspirate 30 to 40 ounces because he complains of a heavy feeling in the chest and also interrupted sleep and bad dreams at these times. Another patient had to have over two pints aspirated weekly for about eighteen months during which time he kept at work. These patients should be kept under supervision and examined with the X-ray screen every few months.

If, however, it is decided to maintain the pneumothorax, the effusion must be aspirated and replaced by air to prevent obliteration of the pneumothorax cavity. Usually after one or two aspirations it does not reform.

In replacing liquid the quantity of gas required is less than that of the liquid removed; roughly speaking, about half the quantity is usually sufficient. In one case 225 c.cm. of liquid was removed and 200 c.cm. of air given, the pressure being changed from −8 −0 to 0 + 6.

In another case the initial pressure was −17 −9. After removing 600 c.cm. of liquid it became −22 −13, and after another 200 c.cm. had been removed it was −28 −15. On adding 200 c.c.m of air the pressure at once rose to −16 −9, another 200 c.cm. of air made it −10 −6, and after a further 200 c.cm. it became −4 −0. Thus the

removal of 800 c.cm. of liquid lowered the pressure from −17 −9 to −28 −15, and the addition of 600 c.cm. of air raised it to −4 −0.

The methods of gas replacement and washing out the pleural cavity have been described in Chapter VI. I prefer to use two needles in most cases so that air can enter through one as the liquid leaves through the other, and by this means the intrapleural pressure can be kept constant, raised or lowered at will. It is not wise to let the pressure alter too quickly, and too low a pressure is apt to cause symptoms such as dyspnœa, faintness or paroxysm of cough.

If the effusion becomes purulent the case is very much more serious, although I have had several patients with large purulent effusions who made good recoveries. Plate XLIV. shows the condition of a patient in November, 1926. He had a large right tuberculous pyo-pneumothorax. Plate XLV. shows the same case ten years later (February, 1937). The right lung has completely healed and appears better than the left which shows apical fibrosis. The pus should be aspirated and the pressure kept as low as possible in order to help the lung re-expand. If the lung does re-expand the outlook is hopeful, and a satisfactory termination of the treatment by obliteration of the pneumothorax cavity may be expected. Sometimes even after failure of simple aspiration and lavage, gelatinothorax may succeed in cleansing the pleural cavity and eventually allowing the lung to re-expand. If, however, there is no sign of re-expansion the patient may remain in good health and keep on with his occupation in spite of a chronic tuberculous empyema. It is remarkable how little inconvenience this type of empyema may cause but the ultimate outlook is not good in the majority of cases. Dumarest and his colleagues (15) describe this condition as benign pyothorax or cold pleural abscess and think it should be left alone if it is well tolerated by the patient and that it often becomes absorbed even after a long time. They regard amyloid disease or other complications as rare

and advise aspiration only if the pus re-accumulates quickly and do not advocate oleothorax for these cases. Malignant pyothorax is a different condition which seriously threatens life and is often associated with a ruptured lung. The pus re-accumulates quickly after aspiration and they advise washing out the pleural cavity frequently with some simple liquid but here also they oppose using oleothorax. If the condition persists intercostal drainage should be performed. Morriston Davies (32) thinks that thoracoplasty alone is useless if there is a pulmonary fistula and the lung is much collapsed and the pleura very thick. He says that it is necessary to do an exterior decostalisation of the chest wall and in addition to remove the parietal pleura, the periosteum of the ribs and the intercostal muscles, but even if done in a series of stages this operation has a high mortality.

The Effect of Artificial Pneumothorax on Vital Capacity

Case 1. Male, twenty-eight. Onset of pulmonary tuberculosis one year ago. After five months' sanatorium treatment tubercle bacilli were still present in the sputum and there were signs of active disease at the apices of the upper and lower lobes on the left side. In March the vital capacity was 2,570 c.cm., being only 41 per cent. of normal.

July.
V.C., 2,450 c.cm. Percentage, — 44.

August 23rd.
V.C., 2,250 c.cm. Percentage, — 48·5.

August 27th.
Artificial pneumothorax. 900 c.cm. of air introduced, the end pressure, — 1. An hour later: V.C., 1,560 c.cm. Percentage, — 65.

August 30th.

Before the first refill. Pressure, — 6·5.

V.C., 2,050 c.cm. Percentage, — 53.

One hour after refill of 900 c.cm. of air and pressure, — 2.

V.C., 1,680 c.cm. Percentage, — 61·6.

September 2nd.

Before the second refill. Pressure, — 5.

V.C., 1,900 c.cm. Percentage, — 56·5.

One hour after refill of 800 c.cm. and pressure 0.

V.C., 1,400 c.cm. Percentage, — 68.

This case illustrates the effect of large initial doses. The patient's vital capacity was getting gradually worse before treatment. 900 c.cm. of air reduced the vital capacity by 690 c.cm. Three days later the vital capacity increased 490 c.cm. and fell 870 c.cm. on the addition of 900 c.cm. of air into the pleural cavity. Three days later it rose 220 c.cm., and was reduced 500 c.cm. by a refill of 800 c.cm.

Case 2. Male, aged nineteen. Two years' history of pulmonary tuberculosis. Signs of extensive disease of the right lung. Tubercle bacilli present.

September 2nd.

Before artificial pneumothorax.

V.C., 2,880. Percentage, — 45·8.

September 3rd.

Twelve hours after 700 c.cm. of air had been introduced.

End pressure, — 2·5.

V.C., 1,800. Percentage, —58·7.

September 6th.

Seventy-two hours after the first refill. Pressure, — 4.

V.C., 2,450. Percentage, — 48·6.

Two hours after 600 c.cm. of air had been introduced.

Pressure, — 0·5.

V.C., 1,700. Percentage, — 60·8.

September 8th.

Forty-eight hours since the second refill.
V.C., 2,050. Percentage, — 53.

September 9th.

Two hours after the third refill of 900 c.cm. Pressure 0.
V.C., 1,500. Percentage, — 65·5.

Analysis of Cases treated by Artificial Pneumothorax

Riviere (14) referred to the dangers of judging the results of any form of treatment in cases of pulmonary tuberculosis. He points out that no two cases are exactly alike, and that the methods of classification and analysis of the cases by different authors vary considerably, so that it is difficult to compare one series with another.

The recent report of the Joint Tuberculosis Council (87) on the survival rate of 8,021 cases of pulmonary tuberculosis treated in forty-two different hospitals by artificial pneumothorax compared with 1,829 patients in the same hospitals on whom pneumothorax had been attempted but failed, showed that the ratio of actual to expected deaths amongst the pneumothorax cases was approximately half that amongst failures. At the same time it was pointed out that a patient with an adherent pleura could not be regarded as a true control case for one who had no adhesions. The results of these two series were compared with the results in 2,750 patients treated at the King Edward VII. Sanatorium, Midhurst, of whom less than 1 per cent. had pneumothorax. The ratio of actual to expected deaths was approximately twice as high in the patients treated with pneumothorax as in the Midhurst group. If, however, ancillary methods such as gold were used, the results in the Midhurst cases were practically the same as those treated by pneumothorax together with ancillary treatment.

Although this report does not support the general opinion

as to the value of pneumothorax its findings must be regarded as inconclusive and it shows that statistics in this type of enquiry are often misleading. The first difficulty is to find a satisfactory "control." If the cases are not sub-divided according to the extent of disease, its activity, the presence of cavities or tendency to fibrosis, also the age, sex and temperament of the patient, comparisons are practically useless. If they are so divided each group contains too few cases from which to draw any useful conclusions.

One should also remember that certain methods of treatment are only employed if the patient is doing badly. Artificial pneumothorax, for example, is not often induced in the so-called "early good" case.

The following figures, therefore, are not intended to illustrate the results of artificial pneumothorax as compared with the results of other methods of treatment, but they do show the types of case for which the treatment was employed and the chances of certain dangers and complications.

The series consists of the first 500 cases in which I induced or attempted artificial pneumothorax for pulmonary tuberculosis. During the same period there were seventy-one cases of non-tuberculous disease and twenty-six cases of spontaneous pneumothorax. Treatment began between the years 1918 and November, 1925, so that it is now over eleven years since treatment was started in the last and eighteen years since it was started in the first of this series. Including unsuccessful attempts to induce a pneumothorax and refills, the pleura was punctured 10,141 times.

The following accidents occurred from these 10,141 punctures :—

1 case of fatal pleural shock.
8 cases of collapse and alarming symptoms, but recovery without bad after effects.
17 cases of faintness or mild shock.

8 cases of bad surgical emphysema; all recovered.

57 cases of slight local surgical emphysema.

4 cases where a large vessel or the heart was punctured, and blood rushed up the pneumothorax tubing, but no ill-effects followed.

2 cases had what I thought to be mild gas embolism, but both quickly recovered.

A sinus developed along the track of the needle in five cases with tuberculous pyopneumothorax. In no other type of case was there any infection of the needle track.

During the treatment there were the following complications in this series :—

Spontaneous pneumothorax on the opposite side occurred in three cases and proved rapidly fatal.

Four patients had operation for appendicitis during the treatment and one died.

Six patients were confined during the treatment. In one an acute exacerbation of disease occurred in the untreated lung, but the other five suffered no ill-effects.

Of the 500 tuberculous cases an effusion developed in 177. In 128 of these it was clear and in 54 tuberculous pus. In no case was any organism found other than the tubercle bacillus except when there was rupture of the lung.

Of the non-tuberculous cases in this series an effusion developed in one only, and this was a case of acute abscess where some clear liquid formed, but was very small in amount and was quickly absorbed.

In 46 of the 500 cases a pneumothorax could not be induced owing to adherent pleura, of the remaining 454 the pneumothorax was induced at the first attempt in 429, at the second in 17, and at the third or fourth in 8.

249 were left-sided, 289 right-sided, and 12 bilateral cases. No difference in result was noticed, whether the case was right- or left-sided.

244 were males and 256 females.

Of the 71 non-tuberculous cases artificial pneumothorax was induced or attempted for the following reasons :—

Bronchiectasis	23
Abscess of lung	14
Recurrent hæmoptysis	8
Pain of pleurisy	5
Replacing pleural effusion	9
Diagnosis	12

Of the 26 cases of spontaneous pneumothorax 17 developed pyopneumothorax, and of these 14 died. Of the 8 who survived 2 had thoracoplasty. Nine did not develop purulent effusion, and only 2 of these are dead.

In 44 cases there was too much adherent pleura to allow a sufficient collapse to produce any effect, and of these 32 are dead.

In 46 cases no collapse at all could be obtained, and of these 32 are dead.

The following table shows the incidence of effusion in the four groups :—

TABLE 30

	Total cases.	Clear Effusion.	Pus.
A.	16	5 (Of these 3 are dead.)	0
C.1	168	50 (Of these 18 are dead.)	19 (Of these 18 are dead.)
C. 2	178	56 (Of these 22 are dead.)	25 (Of these 23 are dead.)
C. 3	58	12 (Of these 10 are dead.)	10 (Of these all are dead.)

The next table shows the number of months from the onset of treatment to death in those in this series who died :—

TABLE 31

Months.	0–6.	6–12.	12–18.	18–24.	24–36.	36–48.	48–60.	Over 60.
A. . .	6	2	1	—	—	—	—	—
C.1 . .	1	1	4	2	11	10	9	5
C.2 . .	3	3	9	15	22	12	4	3
C.3 . .	10	15	7	12	5	—	—	1

I divided the tuberculous cases in this series into the following groups :—

A. Acute pulmonary tuberculosis.

C. Cases with medium resistance who failed to get on under other treatment.

1. Unilateral disease.
2. Slight involvement of the other lung.
3. A third or more of the better lung involved.

There were 500 tuberculous cases in the series and artificial pneumothorax was induced or attempted between 1918 and November, 1925. The results in 1934 in those cases where it was successful and maintained for over three months were :—

TABLE 32

	C.1.	C.2.	C.3.
Arrested . .	71 (58·7 %)	20 (16·4 %)	2 (4·5 %)
Alive but still with active disease .	5	18	1
Dead . . .	56 (42·4 %)	84 (68·8 %)	42 (93·3 %)
Total . . .	132	122	45

R. C. and R. W. Matson and Bisaillon (38) described the results of 492 cases of pulmonary tuberculosis treated by artificial pneumothorax. They exclude all cases then under treatment or treated within the last two years, so as to show

the later rather than the immediate results. They admit that certain factors influence the result; for example, the environmental and social conditions of the patient, his temperament and willingness to give loyal co-operation

TABLE 33

	Cases.	Clinically well.	Arrested.	Dead.
		Per cent.	Per cent.	Per cent.
Group I. . . .	194			
Satisfactory collapse	99	53	15	21
Partial collapse .	62	17	19	46
No collapse . .	33	15	12	54
Group II. . . .	177			
Satisfactory collapse	71	40	32	15
Partial collapse .	70	11	17	48
No collapse . .	36	5	8	61
Group III.. . .	52			
Satisfactory collapse	21	43	24	28
Partial collapse .	22	18	9	66
No collapse . .	9	0	22	44
Group IV. . . .	39			
Satisfactory collapse	13	46	7	23
Partial collapse .	17	6	6	76
No collapse . .	9	0	0	97
Group V. . . .	30			
Satisfactory collapse	7	42	0	57
Partial collapse .	12	8	16	58
No collapse . .	11	0	18	68

in the treatment, etc., but under favourable conditions they believe the end result depends on :—

1. The type of the disease.
2. The character of the pneumothorax.
3. The condition of the opposite lung.

They divide their cases into five groups :—

1. Chronic fibro-caseous disease progressive in character, but with little or no cavitation.

2. Chronic fibro-caseous disease more advanced than the first group, progressive in character and with demonstrable cavitation.

3. Chronic fibro-caseous disease with cavities, but not progressive.

4. Acute pulmonary tuberculosis. All were cases of rapidly advancing disease.

5. Chronic bilateral cases with cavities in both lungs.

Trail and Stockman (39) give the after-histories of 80 patients on whom artificial pneumothorax had been induced at King Edward VII. Sanatorium, Midhurst, between 1924 and 1930 and of 33 in which it was tried but failed owing to adherent pleura. In November, 1933, of the 80 pneumothorax patients 51 (63·7 per cent.) were fit for work and 16 (20 per cent.) were dead. Of the 33 unsuccessful cases 6 (18·27 per cent.) were fit for work and 22 (66·67 per cent.) were dead. One must, however, bear in mind the criticisms which have been made about the comparison of patients having a free pleura with those whose pleura is adherent.

Shaw (40) advocated the early induction of pneumothorax in tuberculous patients and compared 267 cases so treated between 1922 and 1930 with 1,707 treated at King Edward VII. Sanatorium, Midhurst, by simple routine between 1907 and 1916 and described by Bardswell and Thompson (41). The mortality in the two groups was as follows :—

TABLE 34

	Total.		Dead.		Percentage dead.	
	M.	F.	M.	F.	M.	F.
Shaw's cases .	139	128	30	31	21·5	24·2
Midhurst cases.	1,053	654	478	278	45·8	41·7

Grouped under the L.C.C. classification these results were :—

TABLE 35

	Total.		Dead.		Percentage dead.	
	M.	F.	M.	F.	M.	F.
Group I. . .						
Shaw's cases .	22	21	0	0	0	0
Midhurst cases	289	171	67	23	23·1	13·5
Group II. .						
Shaw's cases .	68	55	7	6	10·3	10·9
Midhurst cases	541	307	245	131	45·3	42·7
Group III. .						
Shaw's cases .	49	52	23	25	46·9	48
Midhurst cases	223	176	166	119	74·4	67·6

184 of Shaw's cases had sanatorium treatment in addition to artificial pneumothorax but this did not affect the mortality except for the females in Group II. where it was 33 per cent. less for these patients than for those who had pneumothorax treatment alone.

Horton-Smith Hartley, Wingfield and Burrows (42) investigated the expectation of survival in pulmonary tuberculosis in a series of 8,766 cases treated at the Brompton Hospital Sanatorium, Frimley. They found that the dangerous time is the first two years after leaving the sanatorium, but that after this the chances of survival improve year by year. Included amongst these patients were 701 treated by artificial pneumothorax whose chances of survival they found were materially better than those not so treated. Even B3 patients showed a higher rate of survival if treated by pneumothorax.

They regard the fundamental factor in the prognosis of artificial pneumothorax cases to be the freedom from disease

in the opposite lung, for the risk of this becoming infected is considerably lessened by collapse of the affected lung.

Bentley (48) has recently published a full report on artificial pneumothorax, including a digest of the literature and a detailed account of the results of 677 cases treated by the London County Council.

Although the results recorded by various authors differ considerably and statistics on this subject are misleading one may draw the following general conclusions :—

1. Accurate statistics are difficult to obtain chiefly owing to the impossibility of getting comparable controls.

2. Unilateral cases do very much better than bilateral ones, and this is probably due to the fact that pneumothorax prevents spread to the better lung.

3. In bilateral cases pneumothorax is successful only if the diseased parts of both lungs can be collapsed or if the disease is not active in the better lung.

4. The most dramatic results are seen in the acute exudative type of case in which spread of the disease is often stopped as soon as the lung is collapsed. The greater the tendency to fibrosis and chronicity the less likely is pneumothorax to succeed and the more suitable is the patient for some other form of collapse such as thoracoplasty.

5. Dangers and complications of the treatment are few, and against them must be set the danger of spread in an uncollapsed lung. Although they are rare dangers do exist, so that pneumothorax should not be undertaken as a routine in every early case.

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CHAPTER XIV

TREATMENT—*Contd.*

Oleothorax and Surgical Procedures

Oleothorax. This consists in introducing medicated oil into the pleural cavity. Olive oil or paraffin may be used, and some 5 per cent. oil of gomenol is generally added as an antiseptic. In the case of paraffin, eucalyptus may also be added, as it aids the solution of the gomenol. A preparation containing paraffin 94 parts, gomenol 4 parts, and eucalyptus 2 parts is a common formula. It is said that olive oil keeps the pleura soft, but paraffin renders it hard. For this reason paraffin is best if there is a superficial cavity, or if it is required to thicken the pleura in a case of displacement of the mediastinum with pneumothorax. Chandler (1) prefers pure sterile olive oil. There is no need, he says, for the addition of gomenol which may act as an irritant.

Crocket (2) advised a 2 per cent. solution of gelatin in normal saline with acriflavine added just before use to make a dilution of 1 in 2,500. He claims that by mixing in even suspension with the effusion this preparation is more efficacious than an oily one which will not mix.

In my opinion oleothorax should not be used instead of gas to keep the lung collapsed and save the necessity for refills. It would require a large quantity of oil even if the mediastinum were rigid, and if it were mobile it would be displaced by the oil before the lung became sufficiently collapsed and the treatment would be actually harmful. Nor do I advocate oleothorax in the treatment of pyopneumothorax although I have found gelatinothorax, as

practised by Crocket, valuable to cleanse the pleural cavity and prepare the patient for thoracoplasty.

Oleothorax is helpful in cases of obliterating pneumothorax or to maintain a selective collapse. It is not uncommon to find obliteration of the pneumothorax cavity starting after adhesions have been cauterised especially if there has been bleeding. The process starts at the base and extends upwards so that if oil is introduced it is forced to the apex and will maintain collapse of the upper zone for months or even years without causing any symptoms. (Plates XXXVII. and XXXVIII.). If there is an area of softening at the apex, rupture of the lung into the oil may occur and sometimes leads to disastrous results. It is largely because of this danger that oleothorax is not so frequently used in these cases. But, as Chandler points out, the risk is slight owing to the thickened pleura and it is one most patients are prepared to take since the alternative is thoracoplasty.

He regards a rigid mediastinum and a pleural space of strictly limited capacity as essentials for the establishment of a successful oleothorax.

Gilbert (8) after seven years' experience of oleothorax, gave the following classification of conditions for which it may be considered :—

1. Pleurisies of pneumothorax.

   A. Acute sero-fibrinous pleurisy.
      Here oleothorax is contra-indicated during the acute stage as the irritation of the oil would aggravate the condition. Only if adhesions follow this type of effusion should oleothorax be considered.

   B. Repeated acute pleurisy.
      Here oleothorax is indicated, not during the acute stage, but between the attacks. The effusion should be completely aspirated and some 200–300 c.cm. of oil injected.

C. Puriform effusion, or benign pyothorax.
Especially if tubercle bacilli are present in large numbers, oil containing 2–5 per cent. gomenol should be introduced after removing the fluid. If the fluid reforms it should again be replaced by gomenol solution. Several aspirations may be necessary.

D. Serofibrinous pleurisy with chronic subfebrile evolution.
In this condition there is prolonged but not severe pyrexia with constitutional symptoms, and although it sometimes subsides, there is a tendency for it to progress to a tuberculous empyema.

E. Purulent tuberculous pleurisy, or malignant pyothorax.
This is a very serious complication of artificial pneumothorax, and is accompanied by fever and toxæmia. It is associated with an acute caseous pleurisy. Aspiration of the pus and replacement with large quantities of gomenol in olive oil has the double advantage of disinfecting the pleura and maintaining the collapse of the underlying lung. This treatment may be successful, but even if it is not, and subsequently thoracoplasty becomes necessary, it will have cleared the pleural cavity to some extent, reduced the toxæmia and rendered the patient better able to undergo thoracoplasty.

F. Purulent pleurisy, secondarily infected.
In these cases the pleural cavity should be washed out with saline before the oil is injected.

2. Pleuro-pulmonary perforation.
Oleothorax may be successful in the case of a small, but not of a large perforation.
3. Cases of unsuccessful artificial pneumothorax collapse due to insufficient compression.
4. To prevent progressive adhesion of the pleura.
5. To overcome mediastinal laxity combined with an exaggerated pleural elasticity.

6. To replace air refills.

He points out, however, that as the control of an oleothorax is more delicate than that of a pneumothorax, it is hardly justifiable to substitute oleothorax for artificial pneumothorax without some special indication.

Sometimes the pleura is sensitive to the oil, and a febrile reaction follows its injection. For this reason it is wise first to inject only a small quantity, and a day or so later produce the oleothorax if there has been no reaction. Apart from this, the only complication is the formation of an effusion which together with the oil produces displacement of the mediastinum if it is mobile and for this reason a rigid mediastinum is necessary.

Extrapleural Pneumolysis or Plombage. When, owing to adherent pleura, an artificial pneumothorax cannot be induced, part of the lung may be collapsed by stripping the parietal pleura from the chest wall in the desired area and packing the space with solid paraffin, wax, fat, or muscle. Morriston Davies (4) advises breast in the case of women and pectoralis major in men.

When the apex of the lung is collapsed in this way it is known as apicolysis, and has been advocated in cases of apical tuberculosis if the apex cannot be collapsed by artificial pneumothorax.

The indications for the operation are :—

1. Active disease confined to the apices of the lungs if pneumothorax fails owing to adherent pleura and there is too much bilateral disease to justify thoracoplasty.

2. Apical cavity producing symptoms such as hæmoptysis or pyrexia if artificial pneumothorax cannot be induced.

To perform the operation, Morriston Davies (4) recommends the removal of the second costal cartilage and possibly part of the rib. When the areolar tissue between the pleural membrane and the chest wall is reached, the pleura is stripped away, thus freeing the lung, which retracts and draws the parietal pleura with it. The gap left

is filled with pectoralis major muscle or breast tissue. If pectoralis major is used, the muscle is dissected from the chest wall and humerus, but the attachments to the clavicle are left so that a pedicled graft is obtained. In the case of women, breast tissue is dissected away from the skin, nipple and muscle, but left attached by a tail in the axilla.

If wax is used it tends in time to find its way out through the skin, or it may work its way into the bronchial tubes, and be coughed up in pieces many years later. In one patient left artificial pneumothorax had been tried, and failed owing to adherent pleura, and so extrapleural collapse was induced in Switzerland by inserting 800 c.cm. of wax between the parietal pleura and the chest wall. At that time the patient was cachectic and had tuberculous peritonitis in addition to extensive infiltration and cavitation in the left lung. The immediate result was very striking, he lost his toxic symptoms, tubercle bacilli disappeared from the sputum, the peritonitis improved and he was walking about a few months later. He remained fairly well for about seven years, when symptoms began to increase and a left-sided thoracoplasty was performed and caused improvement for some time. He first began to cough up pieces of wax twelve years after it had been inserted, and this continued until his death some two years later.

In this case the treatment undoubtedly checked the rate of spread of the disease and the patient, although suffering from advanced tuberculosis at the time, lived for fourteen years.

In one of my cases where fat had been used in performing an apicolysis, the place became septic and a large abscess formed. It healed, however, and the patient was actually better, the contracting scar tissue having produced a fair collapse of the apex of the lung.

Hudson and Häberlin (5) describe their experience based on fifty cases of extrapleural plombage. They consider the most suitable case to be one of good general resistance and

a localised apical cavity. The object is not to collapse the cavity mechanically by compression but to encourage its collapse by separating the apex of the lung from the chest wall. It is not necessary to use more than 150 grm. of paraffin wax even in the largest plombage and the secret of success is to free the apex completely from the chest wall. The plugs of paraffin are pressed gently against the ribs and not against the lung. No effort is made to compress the lung and the apex when free tends to sink down by gravity.

Phrenic Evulsion. Section or evulsion of the phrenic nerve will cause paralysis of one half of the diaphragm, and consequently those movements of the lungs which are controlled by the diaphragm will cease.

Sprawson (6) injected lipiodol and took measurements of the lung during respiration. He found that the whole lung descended during inspiration, the base most and the apex least. He also found that during inspiration the apex moved outwards but the rest of the lung towards the mid-line and the bronchi became more horizontal. Phrenic evulsion affects the movements of the base considerably but those of the apex very little.

Formerly it was the custom to divide the phrenic nerve, and this often failed to produce paralysis of the diaphragm owing to the frequent presence of accessory phrenic nerves. Now the nerve is twisted and some six or more inches are removed. In one case Morriston Davies (4) removed 49 cm. (20 in.) of the phrenic nerve, 29 being the trunk, and 20 cm. the terminal filaments. He found recovery of the diaphragm in only 1 out of 105 cases, and in this one the muscle was paralysed and showed paradoxical movements a month after the operation, but five months later it was functioning normally. In this case 4 in. of nerve had been removed.

If only temporary paralysis is required the nerve may be crushed or injected with alcohol. Alexander (7) crushes the phrenic nerves and resects some 2 cm. of its accessory

branches in order to produce paralysis of the diaphragm on that side for about six months. This temporary paralysis may be made permanent at a subsequent operation if necessary.

Morin (8) regards paralysis of the phrenic nerve as preferable to artificial pneumothorax because there are fewer complications, it does not necessitate frequent visits to the physician for refills and it often gives the lung sufficient rest to effect a cure. Moreover, a pneumothorax can always be induced later if necessary. He has treated 321 patients with artificial pneumothorax and 174 with phrenic nerve paralysis with the following results, but many more of the pneumothorax cases than of the phrenic paralysis ones became sputum negative for tubercle bacilli.

	Artificial Pneumothorax.	Phrenic Paralysis.
Better	50 per cent.	58 per cent.
Stationary	27 ,,	32 ,,
Worse	19 ,,	10 ,,

Rist (9) treated 200 patients with phrenic paralysis between 1923 and 1931. Six to eight months after the operation he found the results so good that 50 per cent. appeared cured. In November 1933, however, only 18 per cent. were cured, 8 per cent. were improved and 43 per cent. were dead. 23 out of the 26 who were cured had a long course of sanatorium treatment in addition to the operation.

Sauerbruck (10) advocates phrenic evulsion before thoracoplasty but thinks that in itself it is practically valueless. Purce and Clarke (11), however, state that it may bring about arrest in pulmonary tuberculosis and closure of cavities.

In my opinion phrenic evulsion or crushing should not be performed instead of artificial pneumothorax in early cases. The necessity of attending regularly for refills is an advantage as it ensures that the patient is kept under supervision.

Indications :—

1. To relieve symptoms such as dry cough due to diaphragmatic irritation, vomiting after meals or persistent hiccup. I have seen several cases where indigestion has ceased after phrenic evulsion.

2. When there are unilateral fibrosis and displacement of the mediastinum. In these cases the increased capacity of the thoracic cavity during inspiration produces a suction which throws a strain on the healthy lung and heart because the fibrotic part of the lung cannot expand. This strain is lessened if the diaphragm on the affected side can rise during inspiration. After completion of pneumothorax treatment phrenic evulsion is indicated if there is much displacement of the heart and mediastinum.

3. To give additional rest to the lung when artificial pneumothorax has failed. It is more valuable in basal than in apical cases.

4. As a preliminary to thoracoplasty. If, however, only an upper stage thoracoplasty is contemplated, phrenic evulsion is contra-indicated as it interferes with the movement of the healthy basal part of the lung.

5. As an aid to artificial pneumothorax if the lung is adherent to the apex and diaphragm. The rise in the diaphragm relieves the tension on the lung and will frequently lead to the closure of a cavity which was being held open. If there are adhesions which cannot be cauterised, phrenic evulsion should be performed to relieve the pull of the lung on them and so it is definitely indicated if there is a so-called suspended cavity, that is, one which is held by adhesions, since it tends to prevent rupture of the lung.

In my experience phrenic evulsion does not delay re-expansion of the lung in cases of pneumothorax and enable the interval between refills to be lengthened.

Purce and Clarke state that in cases of artificial pneumothorax there is a tendency for the pleural cavity to become obliterated after phrenic evulsion. They found

that in most cases there was contraction of the diseased part of the lung (selective contraction) without any diminution in the extent of the healthy lung tissue. Contra-selective contraction, however, occurred in 17 per cent. of their cases after phrenic evulsion.

Success depends on the amount of rise of the diaphragm. Purce and Clarke obtained satisfactory diaphragmatic paralysis in 99 of their 816 cases and had no serious complication or ill effect in this series. Complications and accidents have, however, been reported. The subclavian vein has been ruptured during phrenic evulsion. Bleeding may occur as the nerve is being twisted but it usually stops when the traction ceases. Injury to the thoracic duct with flow of milky fluid has been recorded; the flow was stopped by packing with gauze. Fatal cases have occurred as a result of mistaking the vagus nerve for the phrenic. The operation may increase symptoms in certain cases when a flabby diaphragm is a danger. For example, if the mediastinum is over mobile and displaced: in certain cases of chronic effusion: sometimes in bronchiectasis by weakening the force of cough and so increasing the accumulation of sputum in the tubes.

Bilateral Phrenic Evulsion. O'Shaughnessy (12) has described four cases in which evulsion of both phrenic nerves was performed. The intervals between the operation varied from one to six months. In one case the patient became worse as a result of the operation but in the other three there appeared to be improvement.

Thoracoplasty. Morriston Davies (13) considers this operation a serious one which is usually advised to prolong a patient's life when other methods have failed. Some are cured, others receive no benefit and others are made worse by the operation even in carefully selected cases. He wrote, "For those who win the gain is enormous, whilst those who lose have gambled a year or two of an invalid's life against health and freedom."

The one-stage ten-rib resection of Sauerbruch is now rarely performed and the operation is done in two or more often three stages. Morriston Davies (14) at the first stage excises the second and then the first rib and 10 to 12 cm. of the third and fourth. He also removes the transverse processes of the second, third and fourth vertebræ and the necks of the ribs. Three weeks later he performs the second stage of the operation when he removes 10 to 12 cm. of the fifth to eighth ribs, and at the third stage three weeks after this similar portions of the ninth and tenth ribs are removed.

Mason (15) states that whether the upper ribs should be removed before the lower or *vice versâ* is a matter for individual preference. He prefers starting above because thus he can better regulate the degree and rate of the consequent collapse. It is the usual practice in the United States to do the same, but many surgeons in this country prefer to start with the lower ribs in order to lessen the risk of spreading the disease from above downwards. If, however, there is gross disease in the upper zone and the lower one is not badly affected it is usual to start with the upper ribs and conduct the further stages according to the results. Plate XLVI. shows the chest after complete thoracoplasty.

There is an increasing tendency to avoid a complete thoracoplasty where possible and to confine the operation to an upper one. Roberts (16) has successfully performed upper thoracoplasty where a pneumothorax collapsing the lower part of the lung has been present. He states that the risk of aspiration into the lower lobe is obviated and advocates maintaining the pneumothorax whether or not the lower part of the lung is healthy in such cases before undertaking apicolysis and upper stage thoracoplasty. If the pleura is perforated and air escapes it does not matter, but he regards an effusion in the pneumothorax cavity as a contra-indication and if one is present thinks the pneumothorax should be abandoned and the lung expanded before operation.

It is often sufficient to perform partial thoracoplasty and

it is usually done to close an apical cavity. (Plate XLVII.) The first two ribs and portions of the third, fourth and fifth are removed. This procedure causes collapse from the side but not from above downwards, so that in addition to upper thoracoplasty apicolysis is performed by some surgeons.

Holst, Semb and Frimann-Dahl (17) state that the success of thoracoplasty depends on the closure of cavities and that if there are no cavities surgical collapse treatment is rarely indicated. Bad results are due to deficient relaxation of the diseased part and this applies especially to cavities in the upper zone which is the most common site for the tuberculous cavity. Of 136 cases of isolated cavity they found 128 were in the upper and only 8 in the middle and lower lobes.

Semb performs extra-fascial pneumolysis in association with thoracoplasty (Plate XLVIII.). The first stage of this operation consists of an upper stage thoracoplasty which is very thorough and is done in one or more stages. At a later stage apicolysis is performed and the apex of the lung completely freed. By this operation closure of the cavities was obtained in 90 per cent. of the cases and the mortality was just under 3 per cent. They think the mortality is largely due to impairment of the ability to expectorate resulting in retained secretions and atelectasis or pneumonia. To prevent this it is necessary to avoid removing too many ribs at any one stage and not to perform phrenic paralysis before the operation. If necessary the phrenic nerve can be paralysed afterwards.

Hedblom and Van Hazel (18) found that most deaths occur within the first eight weeks of the operation and are due to shock, cardiac or pulmonary complications and can to a certain extent be prevented by the preliminary preparation of the patient. After the first eight weeks deaths are due to spread of the tuberculosis in the lungs. In 1934 they recorded the results of 8,762 cases of complete and partial thoracoplasties from the literature since 1926. Of these 85·3 per cent. were free from symptoms, 22·1 per cent. were

improved and 33·6 per cent. were dead at the time of their report. Semb performs the operation under local anæsthesia and it is claimed that the cough reflex is thereby retained and the risk of secretion accumulating in the tubes lessened.

Magill (19) advocates nitrous oxide and oxygen after a preliminary narcotic consisting of omnopon gr. $\frac{1}{3}$ and scopolamine gr. $\frac{1}{150}$. He thinks that paraldehyde or avertin per rectum are contra-indicated in these cases, as it is important for the patient to clear his lungs of sputum as soon as possible after the operation. It is better if the surgeon gives an injection of a 1 per cent. solution of novocain in the region of each intercostal nerve as soon as the ribs are exposed at the beginning of the operation, as this reduces the liability of shock when the ribs are divided. With this anæsthesia the best results are obtained and Magill thinks nitrous oxide is the best anæsthetic. He refers to 202 thoracoplastic operations without an anæsthetic death, the five deaths in this series being one on the second day from pulmonary embolism, one on the sixth day from tuberculosis in the other lung, one on the fourth and another on the twentieth day from tuberculous pneumonia, and the other on the eighth day from syncope.

Edwards (20) has described sympathetic shock in thoracoplasty when dealing with the neck of the second rib and transverse process. Sudden extreme collapse occurs due to irritation of the inferior cervical sympathetic ganglion. Infiltration with novocain of the ganglion and of the upper dorsal sympathetic chain will prevent this complication.

Indications. Morrison Davies (18) gave the following indications for thoracoplasty :—

1. Every case of chronic unilateral tuberculosis in patients up to the age of forty-five which cannot be arrested by simpler means. The term unilateral does not exclude some fibrosis in the contralateral lung, but does exclude active processes.

2. Every unilateral case with single or multiple cavities with rigid walls.

3. Cases with hæmoptysis which cannot be controlled. When the hæmoptysis is a danger to the patient's life some latitude is permissible in respect of the disease in the other lung. Such disease must not, however, be progressive and cavities are a contra-indication.

4. In exceptional cases, thoracoplasty is done when the disease is exudative in character.

Artificial pneumothorax is a better method of collapsing the lung than thoracoplasty which should very rarely be performed unless pneumothorax has been tried and has failed. If, however, there is so much adherent pleura that the pneumothorax is inefficient it is better to abandon the pneumothorax and consider thoracoplasty. Sometimes, however, when the upper zone is adherent and the lower collapsed by pneumothorax it is better to maintain the pneumothorax and perform an upper thoracoplasty. Roberts (16) thinks that by this means there is less risk of activity occurring in the lower zone than if the pneumothorax is abandoned and the lower part of the lung allowed to re-expand before the operation.

I think thoracoplasty is also indicated in certain cases of tuberculous empyema with thickened pleura when the pleural cavity will not close. In my opinion, however, the chief value of thoracoplasty is to close an upper zone cavity which cannot be closed by other means.

In bilateral disease it is sometimes possible to perform thoracoplasty on one side and maintain a selective collapse on the other. In a case of bilateral pneumothorax if the collapse is insufficient on one side it may be substituted by apicolysis or a partial thoracoplasty. Tobé (21) has described three cases in which upper thoracoplasty on one side was combined with pneumothorax on the other with good results.

Livingstone (22) has described methods of assessing the clinical condition of the patient before advising thoracoplasty. He emphasises the importance of a thorough clinical examination including the mental make-up of the patient.

Serial radiographs are important to show the progress of the disease. In addition he referred to the value of the sedimentation test, Houghton's blood index which has been described on page 50 and the vital capacity.

In my opinion vital capacity is of the greatest value because patients with a very low one are not likely to do well with thoracoplasty. Jacobæus (23) has described a method of taking the vital capacity separately from the right and left lung. In the following example it will be seen that his method showed that the patient was suitable for a left thoracoplasty.

	Right lung.	Left lung.
Vital Capacity	1,145	40

Contra-Indications. At the present time when a partial upper thoracoplasty can be done instead of the old complete operation bilateral disease is not of necessity a contra-indication. Active disease in the better lung is, however, a definite contra-indication, except in the form of a localised lesion in the upper zone for which localised pneumothorax may be performed. Thoracoplasty is an operation for those who have some resistance as indicated by fibrous formation and so acute exudative disease contra-indicates the operation. The physique of the patient is of more importance than his actual age, but it may be said that patients over fifty are not usually suitable although the operation has been successful in older patients whose general condition is good.

Complications must be taken into consideration. Chronic bronchitis, emphysema or myocardial weakness increase the risk and, if severe, contra-indicate the operation altogether. When the operation is proposed for a tuberculous pyopneumothorax the mediastinum should be rigid or the patient is likely to die from mediastinal flutter and in any case a mobile mediastinum increases the risk of thoracoplasty.

A patient who is toxic, wasted and quite unsuitable for the

operation may become fit for it after a course of medical treatment and in any case the patient's general condition should be brought up to the highest possible standard by rest, diet and if necessary medicine to correct anæmia, constipation or other complications. Transfusion may be required if the hæmoglobin is deficient. If possible, the operation should be performed in the summer or spring as pulmonary complications are more common in the winter.

After-treatment is important. Morriston Davies (14) advised placing a sorbo-pad in the axilla on the side of the operation and over it two elastic straps are stretched and kept in position by strapping pinned at either end of the elastic and fixed to the skin. This elastic tension is maintained continuously until the wound from the final operation is healed, when he fits a special belt which exerts constant tension by means of elastic webbing. This belt should be worn until the cut ribs have become reunited to the spine by new bone formation. He also states that scoliosis is apt to occur if the transverse processes are excised and to prevent it the patient should be encouraged to lie on the side of the operation with two firm pillows under the chest and only a thin one under the head. A tendency to kyphosis can be prevented by avoiding pushing forward the head and neck with pillows. Sandbags are used after the operation to bring the cut ends of the ribs together. Roberts (21) prefers using light weights acting continuously for several hours to heavy ones applied for a few minutes only. With the partial upper thoracoplasty weights are often unnecessary.

In conclusion it may be stated that surgical opinion is more and more becoming in favour of a partial as opposed to a full thoracoplasty. Mason truly remarks that the principal requirement of a fibrotic lesion is facility for its scar tissue to retract, but an exudative lesion requires above all things, rest, at any rate until fibrosis develops.

Thoracoplasty has undoubtedly saved lives and prolonged

many more but it cannot be regarded as a good operation, like lobectomy, where the disease is actually removed. At present, it is the best method of treatment known for certain stages of the disease, but one should always bear in mind that the function of the physician is to prevent the patient from reaching that stage in which such serious surgical procedures become necessary.

Scalenectomy. The object of this operation is to shorten the longitudinal axis of the thorax. The scaleni anchor the upper ribs for the action of the three upper intercostal groups. It is necessary also to paralyse the phrenic nerve in order to prevent the descent of the diaphragm with inspiration. The results of this procedure are disappointing and it is now rarely performed.

Multiple Intercostal Neurectomy. Alexander (24) found that unilateral multiple intercostal neurectomy in dogs and rabbits greatly decreased the mobility of the hemithorax and caused some decrease in its size. He performed the operation on six patients, and the results were good in three. One had the operation too recently for any conclusions to be formed. In one case there was no result either good or bad, and the other patient died, the operation being a contributory cause. Alexander advised phrenicectomy some two or three weeks before the operation in order to reduce the size of the pleural cavity.

In order to perform the operation, longitudinal incisions are made over the angles of the ribs under local anæsthesia. Two or more centimetres of the second to the eleventh intercostal nerves are removed. The operation is safe and painless. The dangers and shock of thoracoplasty are avoided, so that neurectomy can be used for a larger group of patients. Those who are not bad enough to expose to the risk, as well as those who are too ill to stand the shock of thoracoplasty, might well benefit from multiple intercostal neurectomy. Alexander points out that the operation cannot be regarded as a substitute for thoracoplasty, although

it may be a preliminary. If a patient is too ill for thoracoplasty, he may improve sufficiently to have it after intercostal neurectomy. Thoracoplasty closes the chest, which neurectomy cannot do to any appreciable extent, and thus is not so satisfactory in the case of a cavity or lesion which requires closing the lung as well as resting it. A theoretical objection to intercostal neurectomy is that by preventing or hindering expectoration spread of disease and toxæmia may occur from stasis. This did not occur in Alexander's cases, nor did he find any undue dyspnœa.

Roberts (16) thinks there is a future for this operation in its limited field, but up to the present time no striking results have been recorded.

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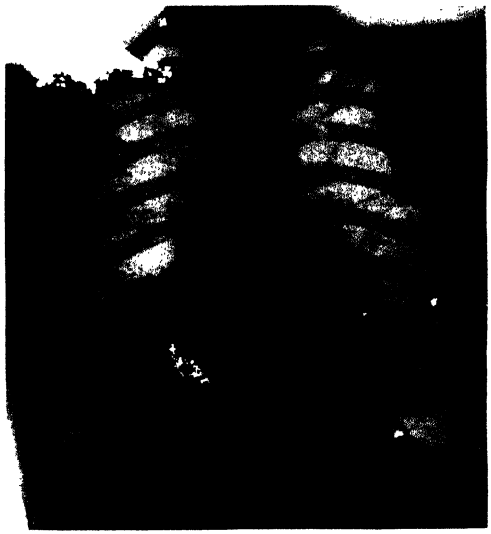
PLATE II

NORMAL LUNG. THE HEAVY ROOT SHADOW [illegible] PATHOLOGICAL SIGNIFICANCE

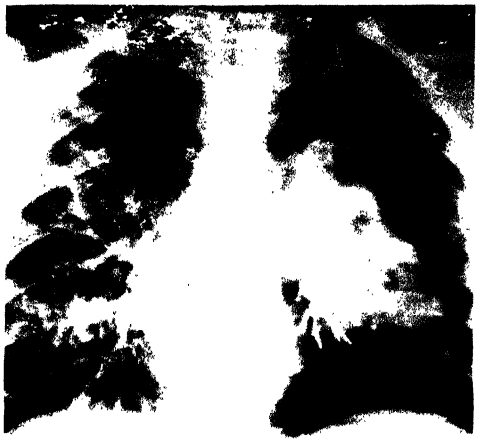
PLATE III



THE NIPPLE SHADOW IS SEEN ON THE LEFT. THERE IS LIPIODOL IN THE RIGHT LOWER LOBE BRONCHI WHICH [illegible] ORMAL. (See p. 55.)

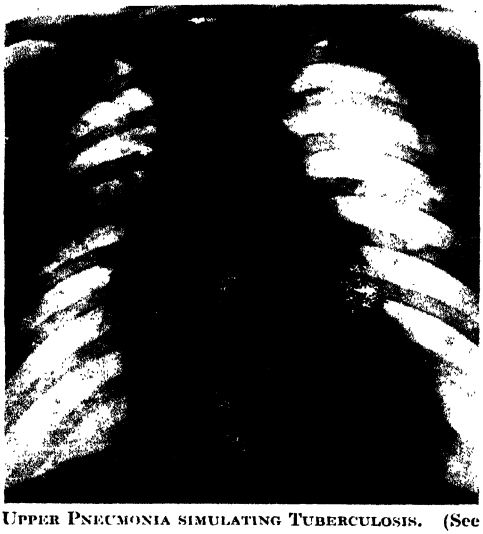
PLATE

THE ACCESSORY LOBE OF THE AZYGOS VEIN. THE COMMA-SHAPED SHADOW CAN BE SEEN NEAR THE STERNUM JUST ABOVE THE THIRD RIGHT RIB. (See pp. 41 and 55.)



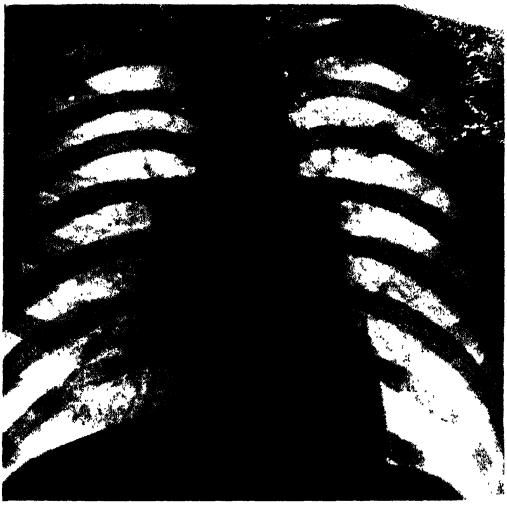
LEFT ARTIFICIAL PNEUMOTHORAX WITH LIPIODOL IN THE BRONCHI. THE PATIENT HAS HAD A SEIDLITZ POWDER, SO THAT THE STOMACH IS DISTENDED WITH GAS, AND BRONCHI CAN BE SEEN THROUGH THE CUPOLA OF THE DIAPHRAGM BELOW WHAT APPEARS TO BE THE EXTREME BASE OF THE LEFT LUNG. (See p. 55.)

PLATE VI



RIGHT UPPER PNEUMONIA SIMULATING TUBERCULOSIS. (See p. 57.

PLATE VII

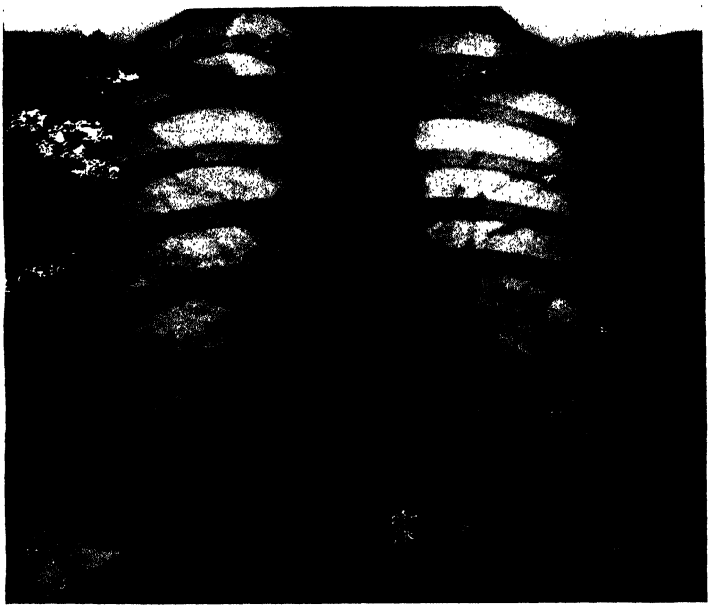


ASSMANN'S FOCUS, SEEN OVER THE FOURTH LEFT RIB IN THE MID-CLAVICULAR LINE. THERE ARE ENLARGED MEDIASTINAL GLANDS IN CONNECTION WITH IT. (See p. 58.)

PLATE VIII

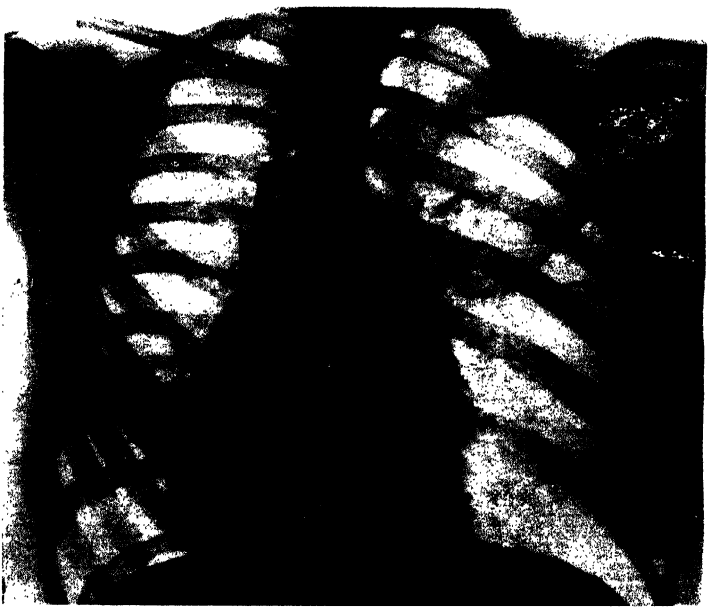
GHON'S PRIMARY FOCUS, OR PARROT'S NODE, IS SEEN IN THE LOWER ZONE OF THE RIGHT LUNG, WITH CALCAREOUS GLANDS AT THE HILUM. (See p. 58.)

PLATE XII



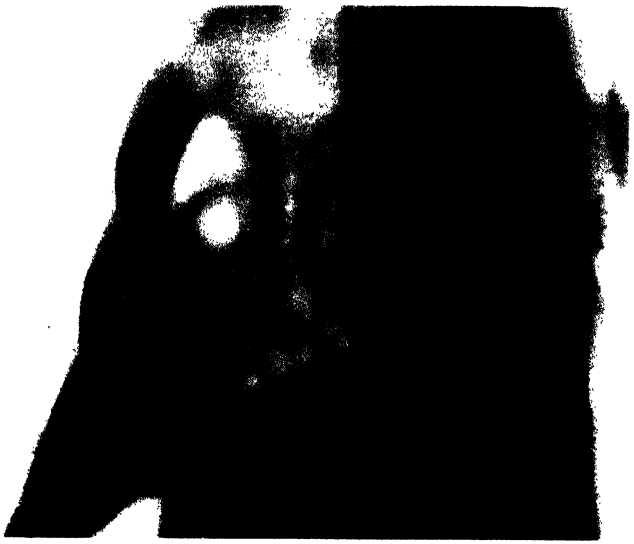
ANTERO-POSTERIOR VIEW. CAVITY IN THE RIGHT LUNG IS OBSCURED BY THE SHADOW OF THE VERTEBRAL COLUMN AND MEDIASTINUM. (See p. 59.)

PLATE XIII



OBLIQUE VIEW SHOWING CAVITY WHICH IS NOT SEEN IN PLATE XII. NOTE THE FLUID LEVEL. (See p. 59.)

PLATE XVI



BRONCHIAL TREE AND CAVITY SHOWN BY THE TOMOGRAPH. (See p. 60.)

PLATE XVII

EXTENSIVE BILATERAL DISEASE. (See p. 65.)

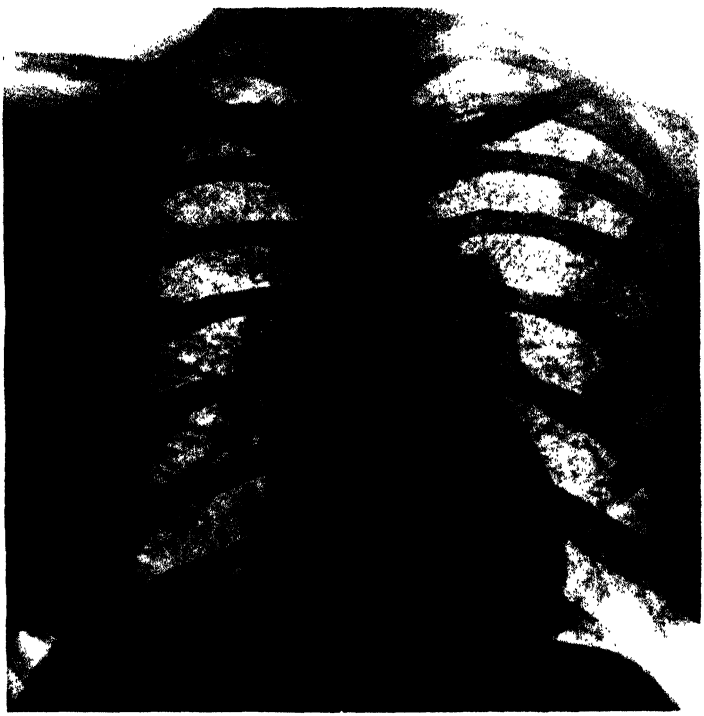
PLATE XVIII

THE SAME CASE AS SHOWN IN PLATE XVII AFTER SIMPLE MEDICAL TREATMENT. (See p. 65.)

PLATE XIX

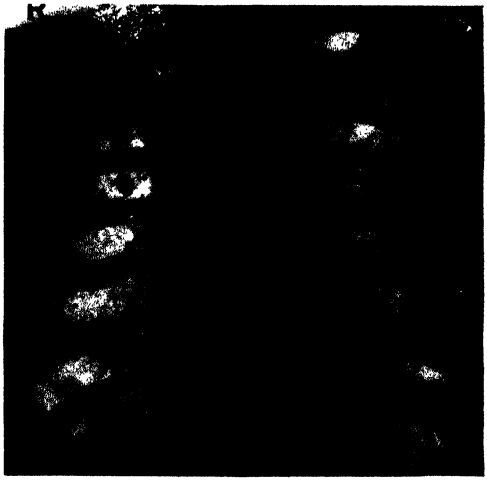
EXTENSIVE DISEASE WITH CAVITATION. (See p. 66.)

PLATE XXIII



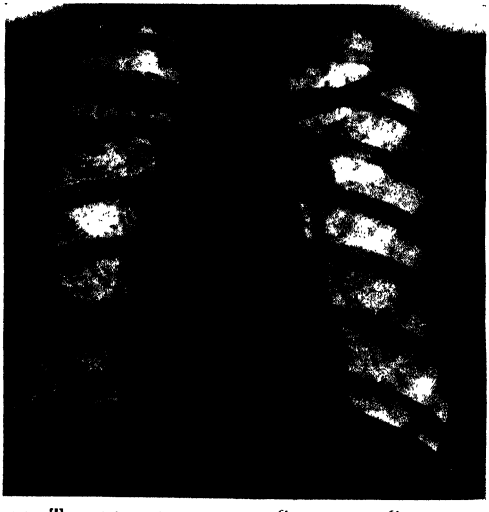
CHRONIC MILIARY TUBERCULOSIS, JANUARY, 1935. (See pp. 67 and 145.)

PLATE XXV



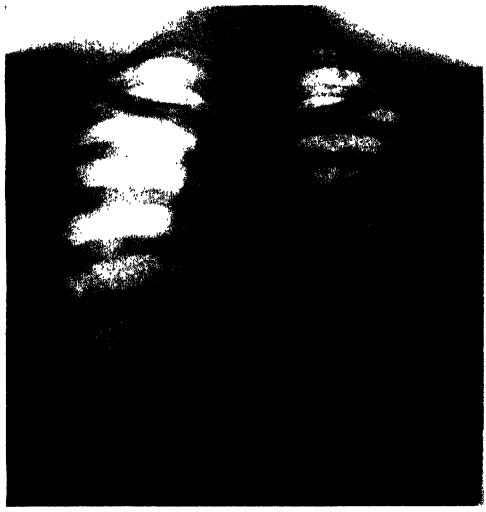
SCATTERED CALCAREOUS DEPOSITS IN A CASE OF HEALED TUBERCULOSIS
(See pp. 67 and 144.)

PLATE XXVI



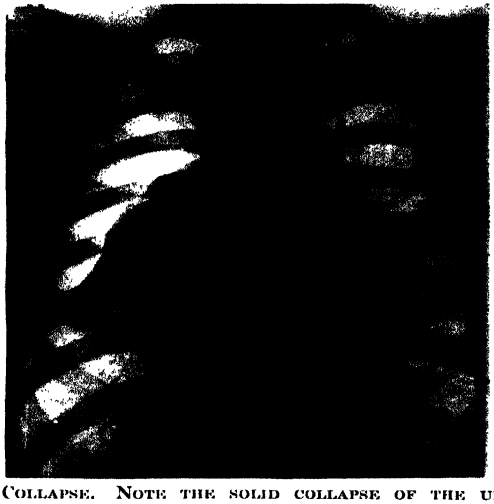
HEALED MILIARY TUBERCULOSIS SHOWING SCATTERED CALCAREOUS DEPOSITS.
(See pp. 67 and 144.)

PLATE XXXV



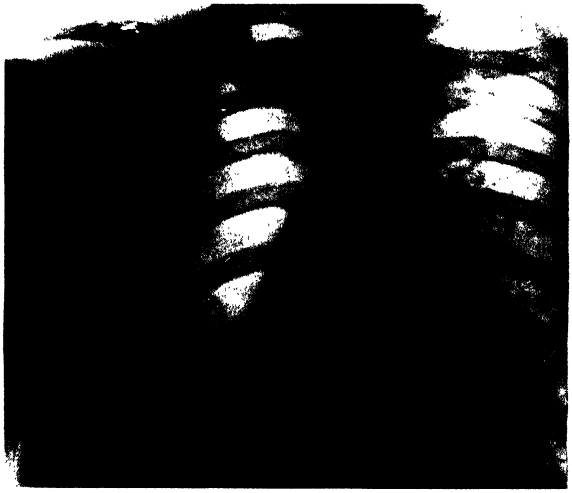
THE SAME CASE AS SHOWN IN PLATE XXXIV TAKEN TWO YEARS LATER. THE LEFT LUNG HAS RE-EXPANDED AND A PNEUMOTHORAX IS BEING KEPT UP ON THE RIGHT SIDE. (See p. 224.)

PLATE XXXVI



SELECTIVE COLLAPSE. NOTE THE SOLID COLLAPSE OF THE UPPER LOBE, WHICH IS COMPLETELY AIRLESS. (See p. 244.)

PLATE XXXVII



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EXTRA-FASCIAL PNEUMOLYSIS AND UPPER THORACOPLASTY.
(See p. 309.)

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