

Blood & Blood-Pressure, in relation
to the Etiology, Prophylaxis,
Treatment, and cure of Pulmonary

Tuberculosis: also suggested coincident or
alternative Therapeutic Measures

Henry Arthur Murray

M B

MD 1928

CONTENTS

Preamble.

Hypotheses.

Evidence adduced in Support of Hypotheses.

A. Etiological Considerations.

1. Normal Blood Supply of the Lung Apices.
2. Normal Slowing of Blood Stream in Lung Capillaries.
3. Anaemia: Its Relationship to Pulmonary Tuberculosis.
4. Low Blood Pressure: Its relationship to Pulmonary Tuberculosis.
5. Lack of Exercise: _ Pulmonary and Physical.
6. Pulmonary Tuberculosis: - A Disease of the Poor.

B. Diseases which influence the Onset and Progress of Pulmonary Tuberculosis.

1. Those which predispose to the Disease.
2. Those which confer Some Degree of Protection against the Disease.

C. Treatment which is known to have produced Good Results in Pulmonary Tuberculosis.

D. Miscellaneous Considerations.

1. Study of a Tuberculous Family.
2. Decline in Death Rate of Pulmonary Tuberculosis.
3. Relative Immunity of the Human Race to Demonstrable Pulmonary Tuberculosis.
4. Rarity of Pulmonary Tuberculosis among the Native Inhabitants of the Mountainous Districts of Basutoland.
5. A Possible Function of the Spleen.
6. Bactericidal Action of the Blood.
7. Probable Action of Some Recently Suggested Therapeutic Measures.

Prophylaxis.

Treatment.



Coincident or Alternative Therapeutic Measures.

A. Intrathoracic Injections of Iodine.

B. Artificial Pneumothorax.

C. Removal of the More Involved Lung.

D. Production of Artificial Mitral Stenosis.

Summary.

References.

PREFACE.

The line of thought followed in this thesis was suggested by the observation and study of a tuberculous family, details of which will be considered under the appropriate section.

Although I do see occasional cases of pneumoconiosis (miner's phthisis) here, true pulmonary tuberculosis is rare, and I have the greatest difficulty in persuading the natives to come into hospital unless they are acutely ill, as they wish to get back to their homes to attend to their cattle or lands.

In consequence, I have not the opportunity for clinical trial of the method herein advocated, and must therefore confine myself mostly to theoretical discussions and the drawing of logical conclusions.

Tuberculosis is generally regarded as a general systemic disease, and, by many authorities, as primarily involving the lymphatic system, from whence it may spread to bones, brain, or to any organ, but it is when the lungs become the chief seat of the disease, that the real danger to life arises, in that it gradually deprives the patient of vital organs, namely the lungs, so that he would be bound ultimately to die from want of them, if his life were not brought to an untimely end by one of the natural terminations of the disease, such as hæmorrhage or toxicæmia intercurrent.

BLOOD AND BLOOD-PRESSURE IN RELATION TO THE
ETIOLOGY, PROPHYLAXIS, TREATMENT AND CURE OF
PULMONARY TUBERCULOSIS; ALSO SUGGESTED COINCIDENT
OR ALTERNATIVE THERAPEUTIC MEASURES.

PREAMBLE.

The line of thought followed in this thesis was suggested by the observation and study of a tuberculous family, details of which will be considered under the appropriate section.

Although I do see occasional cases of pneumokoniosis (miner's phthisis) here, true pulmonary tuberculosis is rare, and I have the greatest difficulty in persuading the natives to come into hospital unless they are acutely ill, as they wish to get back to their homes to attend to their cattle or lands.

In consequence, I have not the opportunity for clinical trial of the method herein advocated, and must therefore confine myself mostly to theoretical discussions and the drawing of logical conclusions. Tuberculosis is generally regarded as a general systemic disease, and, by many authorities, as primarily involving the lymphatic system, from whence it may spread to bones, lungs, or to any organ, but it is when the lungs become the chief seat of the disease, that the real danger to life arises, in that it gradually deprives the patient of vital organs, namely the lungs, so that he would be bound ultimately to die from want of them, if his life were not brought to an untimely end by one of the natural terminations of the disease, such as haemorrhage or toxæmic intoxication.

Up to a certain point, the disease as it affects the lungs, appears, as it progresses, to run a more and more chronic course, presumably owing to Nature's protective and reparative efforts; and, but for the inevitable spread of the disease throughout the lungs, this reparative process would undoubtedly effect a cure; no less than it does when the disease manifests itself in such structures as bone or lymphatic glands, aided merely by more or less conservative treatment. The pulmonary form of the disease is much the commonest clinically, and the cause of, by far the greatest amount of morbidity — and highest mortality — amongst the human race. If tuberculosis of the lungs could be regarded as of no more serious moment than that of other structures, much would have been attained. This, at least, I venture to believe, is within the realms of possibility.

HYPOTHESES.

The development of pulmonary tuberculosis in man, is made possible solely in consequence of "anaemia" (in the sense of a deficient supply, either of whole blood, or blood elements) — natural or acquired — of the lungs. If this "pulmonary anaemia" be prevented, or, if already existing, cured, tuberculosis of the organ will not ensue.

An established tuberculosis of the lungs — at least in the early stages — can be cured; and greatly improved in practically any stage, by the efficient treatment of the pre-existing "pulmonary anaemia."

EVIDENCE ADDUCED IN SUPPORT OF HYPOTHESES.

(A) Etiological Considerations.

1. Normal Blood Supply of the Lung Apices.— The apices of the lungs, especially the right apex, have normally a relatively poor blood supply, and it is common knowledge that pulmonary tuberculosis generally begins in one of the apices of the lungs, and in a majority of cases, in the right apex. This fact is very suggestive, and is best explained by the aforementioned deficient blood supply. It is well recognised that any injured or devitalised tissue is more liable to be attacked by the tubercle bacillus: and any organ or tissue with a deficient blood supply is, potentially, devitalised, and therefore more likely to offer a favourable nidus for the bacillus.

2. Normal Slowing of Blood Stream in Lung Capillaries.— All the lymph in the body is ultimately poured into the venous blood, with which it goes to the lungs. When in the small capillaries of the lungs, the blood stream undergoes a normal slowing, which naturally favours the deposition of any tubercle bacilli which happen to be in the blood, which may have been carried from any lymph gland in the body,— cervical, bronchial, peritoneal etc.— and therefore from any organ or tissue in the body. It can therefore be easily understood that an exaggerated retardation of the blood stream, such as occurs in cases of lowered vascular tone, will make the deposition of the bacilli more probable; whereas an acceleration will make it less likely, and so allow the blood elements sufficient time to deal effectively with the offending organism.

3. Anaemia: Its Relationship to Pulmonary Tuberculosis.— Anaemia occurs in practically all cases of pulmonary tuberculosis, and is generally said to be secondary in type, with a low colour index. Granted,

secondary anaemia does occur in consequence of the toxæmia, but there is also a primary anaemia of the aforementioned description — deficient quality or quantity of blood — in the normal apex, prior to the onset of tuberculosis, so that the secondary anaemia completes a vicious circle: and, incidentally, should for that reason, be all the more energetically treated.

4. Low Blood Pressure: Its Relationship to Pulmonary Tuberculosis.— The great majority of patients suffering from pulmonary tuberculosis, have a low blood pressure, which is also generally supposed to be secondary to the disease. While there may be some lowering of blood pressure in consequence of the tuberculosis, I believe that in most, if not all cases, the hypotension, like the anaemia, exists before the lung disease, and that the presence of the former has much to do with the development of the latter.

Barach,⁽¹⁾ basing his observations on the examination of over four thousand young adults, says that low blood pressure is associated with a poor respiratory apparatus, and occurs in patients of a small chest capacity.

Now pulmonary tuberculosis is commonest in those with undeveloped chests of small capacity, therefore my belief is borne out by Barach's findings.

5. Lack of Exercise: — Pulmonary and Physical.— A considerable proportion of people do not expand their lungs sufficiently, which is comparable to the lack of exercise in, say, a patient having to lie in bed for any length of time. In this latter case, to take the lower limbs as an example, they will get less blood than they would normally, and will, from want of nourishment and exercise, become abnormal, in that their muscles will atrophy. This appears to prove that lack of exercise means lack of blood, in

the lungs, just as in the muscles or elsewhere. In the case of the lungs, one has to consider a two-fold deficiency: firstly, that of the whole of the venous blood of the body, which must pass through the lungs to be aerated, and, secondly, that of the arterial blood which goes to supply the lung tissue. In both cases, from want of exercise of the lungs, there is retardation and insufficiency of the blood supply. Now it is known that people who lead a sedentary life, and consequently do not breathe deeply enough, or exercise their bodies so as to improve their general circulation, including that of the lungs — other things being equal — are more prone to be attacked by the tubercle bacillus.

6. Pulmonary Tuberculosis: A Disease of the Poor.—

Pulmonary tuberculosis is essentially a disease of the poor, and they, in consequence of insufficient nourishment, unsuitable environment etc., will naturally have, on the whole, a poorer quality of blood, a poorer general vascular tone, and lower blood pressure than the well-to-do, who are well fed and live under much better conditions. Now if, as stated by Sir Robert Philip and others, (2) practically all members of the working class are tuberculized by they reach the age of fifteen years, why do only a relatively small proportion of them shew clinical evidence of the disease? This question can be satisfactorily answered, only if the hypothesis which I have formulated, be accepted. It is in consequence of this small proportion having suffered from neglected devitalising diseases, such as measles, whooping cough and anaemia, together with the abovementioned general unhygienic and normally unfavourable conditions, these factors favouring a state of lowered vascular tone, and consequent anaemia of the apices of the lungs, which allows the latent disease to develop, or makes the

subject more vulnerable to fresh and repeated infections.

An obvious criticism of this statement would be that many more of the working classes suffer from these conditions than ultimately develop tuberculosis. Admittedly so, but, in those who escape the disease there will have been some other factor at work, such as efficient treatment of illness, outdoor life, or manual labour together with the ability to obtain sufficient food, which will have counteracted the tendency to lowered vascular tone, thus permitting Nature to produce an efficient and beneficial hyperaemia in response to the attack of the tubercle bacillus. The part which Nature plays in the prevention of demonstrable pulmonary tuberculosis, will be discussed under "Miscellaneous Considerations".

(B) Diseases which influence the Onset and Progress of Pulmonary Tuberculosis.

1. Those which predispose to the Disease.

(a) Congenital Pulmonary Stenosis.— In this condition the blood is greatly hindered in its passage through the stenosed valve, so that the lungs receive very little blood. These patients are extremely liable to contract pulmonary tuberculosis.⁽³⁾

(b) Anaemia.— In this disease the lungs, as the rest of the body, are supplied by blood of an inferior quality. The predisposition to pulmonary tuberculosis is well known.

(c) Syphilis.— In the acute stages of syphilis there are anaemia⁽⁴⁾ and low blood pressure,⁽⁵⁾ dependent upon toxæmia, and the disease is said to favour the development of pulmonary tuberculosis.⁽⁶⁾ In the later stages of syphilis the patient is less liable to contract tuberculosis of the lungs, and, if he does, it is usually fibroid in character and slowly

progressing, (7) most probably in consequence of some syphilitic endarteritis raising the blood pressure — and maybe aortic stenosis — and thus producing a more vascular condition of the lungs.

(d) Measles and Whooping Cough.— These are debilitating and devitalising diseases, in which convalescence is often incomplete, leaving the patients with a lowered vascular tone, in consequence of which they are very apt to succumb to pulmonary tuberculosis. (8)

(e) Diabetes Mellitus.— Diabetes mellitus influences the prognosis of pulmonary tuberculosis gravely. (9) This does not suggest that all diabetic patients are necessarily more prone to be attacked by the tubercle bacillus, but that the combination, when it does occur, is serious.

"An inverse ratio exists between the degree of glycosuria and blood pressure. This might be looked for, as the milder cases of diabetes are apt to occur in elderly persons with some arteriosclerosis and consequent high pressure. As regards acid intoxication, it would seem to be attended with hypotension, owing probably to the generally more asthenic conditions in these patients, rather than to acid toxins acting directly. In no case was the excessive hypertension declared by Potain and Teisser to be characteristic of diabetes observed. All the evidence points to the fact that it is to the complications, especially arteriosclerosis and chronic nephritis, that we must look for the cause of the cardio-vascular secondary conditions." (10)

According to the foregoing extract diabetes is apt to be more severe in younger people, and to be attended with a low blood pressure. If investigations were made, I think that this would be found to be the type of diabetic patient who develops pulmonary tuberculosis, the reason being the sluggishness and less

than normal amount of the blood in the lungs, in consequence of the low blood pressure. Certainly this type of diabetic is of the age more liable to be attacked by the tubercle bacillus.

(f) Insanity.— Most forms of insanity are accompanied by a very low blood pressure, (11) and the insane exhibit a distinct susceptibility to pulmonary tuberculosis. (12)

(g) Gastro-intestinal Diseases.— These diseases very commonly have a low blood pressure, (13) and pulmonary tuberculosis frequently begins with gastric symptoms. (14)

(h) Alcoholism.— Publicans, innkeepers, bar attendants and the like, according to statistics, shew a higher death rate from pulmonary tuberculosis, than do the general population. This is presumably due to the excessive amount of alcohol which they, as a very class, consume. Similarly Osler states that chronic drinkers are much more liable to pulmonary tuberculosis, the reason being a lowering of the vitality by the alcohol. (15) How does alcohol produce this susceptibility? If taken in moderation alcohol has no effect on the blood pressure. If taken in excessive amount it produces a lowered blood pressure, but if this excess be long continued, sclerotic changes occur in the blood vessels, and a consequent high blood pressure follows. (16) Now the age at which pulmonary tuberculosis is most likely to ensue, is long before the age when sclerotic changes will have had time to develop; that is, at the time when the alcohol is causing a low blood pressure. This is further aggravated by the fact that alcohol causes dilatation of the vessels of the skin, thus depriving the lungs, along with the other organs, of much blood which would otherwise go to them.

(i) Any disease in which there is anaemia, or low blood pressure, will favour the development of

pulmonary tuberculosis.

2. Those which confer Some Degree of Protection against the Disease.

(a) Acquired Valvular Disease of the Heart, especially Mitral Stenosis. (17)— In the presence of these lesions, which cause various degrees of backward pressure of the blood in the lungs, and therefore an increased vascularity of them, pulmonary tuberculosis is very rare. The more direct the backward pressure, (as in mitral stenosis) and therefore the greater the vascularity of the lungs, the greater is the protection against the tubercle bacillus. This fact is very significant.

Further, a comparison between mitral stenosis, and the exactly opposite condition congenital pulmonary stenosis, — the possessors of which lesion are very prone to suffer, and die from, pulmonary tuberculosis — is extremely suggestive. Each lesion is analogous to a closed floodgate; the former will not let the blood out of the lungs, and the latter will not let it into them.

These two facts alone, are very strong and convincing evidence that a sufficient amount of blood in the lungs can prevent the onset of pulmonary tuberculosis, and, conversely, that a deficient blood supply favours the development of the disease.

(b) Asthma and Emphysema.— These conditions are believed to afford some protection against pulmonary tuberculosis. (18) I am aware that the emphysematous portion of a lung is atrophied and anaemic, but it is not the emphysema, as such, but its cause (chronic cough, wind instrument blowing etc.) which affords the protection, by producing a negative pressure in the thorax, and a consequent increase of blood from the right side of the heart into the lungs, during the age period when pulmonary tuberculosis is most

likely to develop. Furthermore, if one accepts the theory that tubercle bacilli are carried into the blood stream by means of the lymph, and ultimately deposited in the capillaries of the lung, then there is protection even when emphysema is an accomplished fact, because these lung capillaries have to a great extent disappeared. It is significant that emphysema occurs mostly in the area in which tuberculosis most commonly begins, namely the apex of the lung.

Similarly, in asthma the difficult respiration and chronic cough cause a negative pressure in the thorax, and consequent increased vascularity of the lungs.

(c) Gout.— Sufferers from this malady have a high blood pressure both during, and between the acute attacks.⁽¹⁹⁾ They are said to be less liable to pulmonary tuberculosis in consequence of having gout,⁽²⁰⁾ which would presumably be on account of the increased blood pressure, improving the vascularity of the lungs.

(d) Pneumonia.— The first stage of pneumonia is hyperaemia of the lungs, and one attack predisposes to another, so that it would appear that those subject to pneumonia, are inclined to have hyperaemic lungs. Now pulmonary tuberculosis only rarely supervenes on an ordinary pneumonia: if this appears to have occurred, it will, in all probability, have been tubercular from the beginning.⁽²¹⁾

(e) Any disease which increases the amount of blood circulating in the lungs will prevent the onset of pulmonary tuberculosis.

(C) Treatment which is known to have produced Good Results in Pulmonary Tuberculosis.

The most important therapeutic measures which come under this category are the following:—

1. An abundance of good nourishing food.
2. Cod liver oil.

3. Fresh air, by day and night.
4. Residence at a high altitude.
5. Graduated exercises, or labour.
6. Breathing exercises.
7. Regular habits, including a sufficiency of sleep.
8. Heliotherapy or light therapy, including residence where there is much sunshine.
9. Hydrotherapy.
10. Artificial pneumothorax.
11. Iodine in various forms.
12. Iron.
13. Strychnine.

I do not think the statement that all the above remedies have benefited tuberculous patients, can possibly be challenged. Now why should such a variety of "treatments" produce good results in one disease? To answer that question, one must ask another; what is common to all these therapeutic measures? One thing only, that they act favourable on the blood-vascular system: they all, either directly or indirectly, improve the quality or increase the quantity, of blood going to the lungs, and that is why they improve or cure patients suffering from pulmonary tuberculosis.

I shall now explain how I believe these various treatments to act, so as to produce the same result.

1. Food improves the quality of the blood, and raises the blood pressure, and consequently improves the vascularity of the lungs.
2. Cod liver oil simply acts as a food.
3. Fresh air supplies nourishment in the form of oxygen. Also, as stated by Pugh,⁽²²⁾ breathing cool air with low vapour tension promotes evaporation from the respiratory membrane, and so enhances the outflow of secretion from it, and the flow of lymph and arterial blood through it.
4. High altitude. At a high altitude the air is

rarefied, therefore the patient must breathe more deeply, thus exercising his lungs and increasing the blood to them. Another effect of the rarefaction of the air is to increase the red blood corpuscles.

Also, the blood pressure is increased.

5. Graduated exercises or labour, necessitate deeper breathing, and in this respect act in the same manner as the preceding example.

Barcroft⁽²³⁾ has found experimentally that after a cat had taken exercise, its spleen was reduced to about half its previous volume, it having expelled blood into the general circulation, which was considerably richer in red blood corpuscles and haemoglobin than was the circulating blood. This observation would appear to indicate that exercise improves the condition, and increases the volume of circulating blood, and consequently raises the blood pressure. In virtue of these factors exercise improves the quality and increases the quantity of the blood going to the lungs.

6. Breathing exercises. Simple deep breathing, exercises the lungs and therefore improves their blood circulation. This is augmented by the rise in blood pressure which occurs during forced expiration.

Impeding of inspiration, by inhaling through a small aperture made by the lips, causes a negative pressure in the thorax and consequent aspiration of blood from the right side of the heart into the lungs, producing a passive hyperaemia and improved lymph circulation. Also, the consequent lower oxygen tension acting upon the blood forming apparatus, produces an increase of red and white blood corpuscles and haemoglobin.

7. Sleep. While a person sleeps, his brain, limbs etc. are resting, and consequently receive less blood, and, although much goes to the splanchnic area, there is more available for the lungs than

during waking hours. Again, since people breathe more deeply during sleep, the lungs are exercised, and each deep respiration produces a negative pressure in the thorax, with consequent aspiration of blood from the right side of the heart into the lungs.

8. Heliotherapy or light therapy. By this method radiant energy is transformed into other forms of energy in the patient's body. The most important effect is the production of heat in the tissues, including the lungs, and a consequent hyperaemia, and improved circulation of blood, produced by the infra-red radiations, which penetrate the most deeply. LoGrasso and Balderry⁽²⁴⁾ state that heliotherapy improves the blood picture, as shewn by an increase in haemoglobin and lymphocytes. Bronfin⁽²⁵⁾ also has observed a lymphocytosis. The possible influence of this lymphocytosis will be considered in the section on "Iodine".

In the light-treatment of tuberculosis, most attention has been given to ultra-violet radiation, and, contrary to the general opinion, Crocket⁽²⁶⁾ has not found it to be necessarily contra-indicated in the pulmonary form of the disease. He says that, avoiding fatigue of the patient, improvement has been effected in 85% of cases as compared with 67% by ordinary methods. This belief is also held by LoGrasso and Balderry, who claim to have observed a large number of cases.⁽²⁷⁾ Furthermore, Johnstone⁽²⁸⁾ considers that it is the over-heating and lack of ventilation which generally obtains in lamp rooms, which is responsible for the recorded cases of haemoptysis following light treatment.

Now ultra-violet radiations are generally administered by means of the arc light, or the sun, the emanations from which are rich in infra-red as well as ultra-violet radiations. I believe it is the infra-red radiations which are the most beneficial in the

treatment of tuberculosis, because they penetrate more deeply — in fact right to the lesion, if the direct method be employed — producing hyperaemia and consequent improved blood and lymph circulation. Rollier, the pioneer of heliotherapy, believed that the deeper the pigmentation of a tuberculous patient's skin, the better was the prognosis, but this theory appears to have been disproved by the following observation made by Pugh. (29) He classified 232 cases of surgical tuberculosis, $3\frac{1}{2}$ years after collection, according to result. The mortality among the "very good" pigmenters had been 16%; the "good" pigmenters 15%; the "slight" pigmenters 10%; and the non-pigmenters 9%. To carry this example a step further, if a Britisher suffering from pulmonary tuberculosis goes to reside in a country such as Africa, where there is an abundance of sunshine, he undoubtedly improves; whereas, if a coloured native of the same country should contract the disease, he does not improve to the same extent, if at all. The reason for this difference is that the pigmented layers of the native's skin greatly absorb the ultra-radiations, thus depriving him of the beneficial hyperaemia of the lungs, which the white man derives from the deep penetration of the rays. It would therefore appear that the less deeply pigmented, or liable to pigmentation, a tuberculous patient's skin, the more benefit will he derive from heliotherapy, because apparently the infra-red radiations are permitted a degree of penetration, inversely to the amount of pigmentation. Certainly Pugh's observation is in accordance with the facts as observed between European and coloured races. This fact also lends support to my belief that the infra-red rays are responsible for the benefit presumed to be derived from ultra-violet radiation, and also affords explanation how this benefit is pro-

duced. Since ultra-violet rays are absorbed in the superficial layers of the skin, it is difficult to explain how they can produce improvement in a deep-seated lesion, for example in the lungs; whereas, if infra-red rays be given the credit, their deep penetration, (stated by Kellogg to be fully six inches) and the fact that they produce heat, and therefore hyperaemia and improved circulation of blood and lymph, is a feasible explanation.

To push this argument to a logical conclusion one must advocate in the treatment of pulmonary tuberculosis, the use of the carbon-filament incandescent lamp, since its radiations are rich in all the frequencies except ultra-violet, these last named being filtered out by the glass wall of the bulb. In the absence of the ultra-violet rays there will be no pigmentation, and the infra-red rays will act unhindered.

Further evidence of the utility of infra-red rays is found in the observation of LoGrasso and Balderry,⁽³⁰⁾ who report very gratifying results from exposures to the sun, both as to general improvement and decreased activity of the pulmonary lesion in forty-nine moderately advanced cases, with an unfavourable prognosis after a thorough trial of the much-vaunted rest cure. These results could only have been due to the hyperaemia of the lungs, produced by the infra-red radiations.

9. Hydrotherapy, in the form of cold baths, deepens the inspirations, and also improves the general blood circulation, including that of the lungs. This is enhanced by the friction which is generally employed, during and after the use of the bath. The appetite is also improved.

10. Artificial pneumothorax. The benefit derived from this form of treatment is usually attributed to the fact that the lung is at rest, but I am doubtful

whether this is really the beneficial factor.

Artificial pneumothorax is said to be indicated in moderately advanced unilateral disease, and contra-indicated in the presence of extensive adhesions. Now in moderately advanced cases there will generally have been some pleurisy, resulting in adhesion formation over the diseased area, which will prevent this particular portion of lung from collapsing: therefore the remaining relatively healthy part of the lung will collapse, in consequence of which there will be a great deal more blood circulating through the uncollapsed diseased area. It is this factor which I believe produces any good results from artificial pneumothorax.

Matson⁽³¹⁾ reports 235 satisfactory collapse cases of which 48% are clinically well, 18% arrested, and 22% dead. Now if rest, in consequence of lung collapse, were the important point in artificial pneumothorax, one cannot satisfactorily explain why it should succeed in approximately only half of the cases. It is to be expected that in some of Matson's cases the lesions were superficial, while in others they were situated more deeply in the substance of the lung. I believe that those with the superficial lesions, and consequent pleurisy, resulting in adhesions over the lesions, will have numbered greatly amongst the 48% clinically well. On this assumption — and on no other — can one understand the many failures which occur after artificial pneumothorax. Furthermore, since rest of the lung was obtained in all Matson's 235 cases, if rest were the curative factor, one would expect a more uniform result than actually occurred: whereas, if the increased amount of blood were the important point, one would anticipate good results in only that proportion in which there were adhesions over the lesion, which is, numerically, apparently what happened.

More recently, Matson and his co-workers, ⁽³²⁾ having experience of over 800 cases of artificial pneumothorax, claim that the importance of disease in the contralateral lung has been greatly exaggerated, because improvement in this lung is often coincident with improvement on the collapsed side. Now if the benefit accruing from artificial pneumothorax were due to the collapse, how could the same degree of improvement possibly be obtained in the other lung in consequence of the exactly opposite condition? It would appear that the improvement must be due to some other factor than rest, and the only other factor, so far as can be ascertained, is the possibility of increased vascularity in certain parts of the collapsed lung, and the certainty of this condition in the contralateral lung.

11. Iodine, administered internally, whether in the form of the tincture, the iodides, or iodoform, is absorbed as iodides, and iodine is liberated in the tissues. Iodine produces hyperaemia of the lungs, and also ⁽³³⁾ acts as a lymphagogue. In the first place there occurs a transudation of fluid from the blood vessels, in consequence of which the lymph becomes richer in salts, and therefore extracts water from the surrounding tissues. This fluid then re-enters the blood stream, causing a rise in blood pressure, and also improving the lymphatic drainage. The merits of iodine in pulmonary tuberculosis will be more fully considered under "Treatment".

12 and 13. Iron and strychnine. The action of these drugs on the blood and vascular system respectively, and consequently their value in the treatment of pulmonary tuberculosis, are so well known as to require no discussion. Jagic ⁽³⁴⁾ says strychnine is a good vasotonic. It should therefore raise the blood pressure and increase the vascularity of the lungs.

Consideration of the evidence which I have put forward, leaves one with the impression that these various therapeutic measures, when they have produced beneficial results in the treatment of pulmonary tuberculosis, have done so in virtue of their action in producing an increased vascularity of the lungs, or an improved condition of the circulating blood.

As a further argument in favour of hyperaemia being the beneficial factor in the treatment of pulmonary tuberculosis, may be cited its curative effect in surgical tuberculosis. If the blood is capable of inhibiting, or rendering innocuous, the tubercular process in one situation, it is reasonable to suppose that it would have the same power in any situation.

(D) Miscellaneous Considerations.

1. Study of a Tuberculous Family.— This study illustrates the type of person likely to fall a victim to pulmonary tuberculosis, also the type that will probably escape the disease.

The family in question may be divided into two well defined groups:-

Group 1. The mother and two sons, who were all of the pale, delicate, ex-sanguious type, and of poor physique.

Group 2. A daughter and two sons, of the plethoric, more or less florid, full-blooded type, and of good physique.

(a) The first member of the family to shew clinical evidence of pulmonary tuberculosis, was a son in the first group. The diagnosis was made in 1915 at the age of twenty-five years. The disease ran a chronic course, and the patient died in 1924 at the age of thirty-four years. In the meantime the other

members of the family were exposed to the infection, especially since the termination of the European War. (b) The next case to be diagnosed was a son, aged twenty-three years, in the year 1920. The disease ran an acute course, and the patient died six months later. This boy was one of the second group, but, only a few months previous to contracting the disease, had returned from Salonica, where he had done $3\frac{1}{2}$ years war service, and had suffered somewhat severely from malaria. After his return home he frequently had severe malarial attacks, which were inadequately treated, since he said he could not, and would not, take quinine. In consequence, malarial cachexia and anaemia supervened, converting him into the type of the first group as above described. It is to this fact that I attribute his contracting, and dying from, pulmonary tuberculosis.

(c) The next member of the family to fall a victim to the disease, was the mother, who had been weak and ailing for a year or so, when, in the year 1923, at the age of fifty-three years, the case was diagnosed. The patient has had frequent haemoptyses, more or less slight, and has been bed-ridden ever since.

(d) Next, in the year 1924, the remaining son of the first group, who had, from his school days, been considered "anaemic", was, at the age of twenty-three years, found to be suffering from pulmonary tuberculosis. The disease is running a more or less chronic course.

(e) The daughter and remaining son in the second group, their ages now being respectively twenty and thirty-four years, have entirely escaped the disease, as far as can be ascertained by clinical signs and symptoms. This remarkable fact can be attributed only to their possessing the type, condition and amount of blood circulating in their lungs, which I have asserted and believe to be necessary to prevent the onset

of pulmonary tuberculosis. Conversely, the absence of these qualities in the blood of the first group, and also in that of the converted member of the second group, appears to be the only rational reason why they should have succumbed to the disease, while the other differently constituted members of the family escaped.

The particularly virulent attack which befell the son in the second group, I attribute to the very low vascular tone, and excessive degree of anaemia from which he suffered as a result of untreated malaria. The father of this family enjoys good health.

Pearl⁽³⁵⁾ has studied fifty-seven persons, the family histories of whom, covering 5000 blood relatives, had been traced. He found that virtually three-fourths of the non-tuberculous offspring of tuberculous parents, had been in just as close contact with active open cases as their brothers and sisters who developed the disease.

This fact bears out my observations, and, although Pearl does not offer any explanation, I believe he would find the tuberculous and non-tuberculous offspring to be differently constituted in the same manner as the members of the family which I have studied.

2. Decline in Death Rate of Pulmonary Tuberculosis.— According to the Registrar-General's figures — quoted by Sir Robert Philip⁽³⁶⁾ there has occurred a progressive decline in the death rate of pulmonary tuberculosis. The same authority states that this is from "some cause or other", which is tantamount to admitting that the reason is unknown.

It is quite conceivable that the decline in the death rate may be due to the community as a whole breathing more deeply, and having higher blood pressures, and therefore a better blood supply to their lungs, in consequence of the more strenuous existence, more

active life and development of outdoor sports, which have undoubtedly become more and more popular. In support of this possibility, is the fact that this progressive increased activity of the people is the only thing which has gone hand in hand with the progressive fall in the mortality from tuberculosis. Sanitation and hygiene also, have greatly improved; but when practically all possible had been accomplished in that direction, the mortality from tuberculosis continued to fall, as the activity of the people, as above described, continued to increase. Sanitation and hygiene of course, include many things which would tend to improve the general vascular tone of the people, and therefore would act to some extent in the same manner as the other factors.

3. Relative Immunity of the Human Race to Demonstrable Pulmonary Tuberculosis.— Since more than 90% of the population contract pulmonary tuberculosis, why does such a small proportion — 10% as a maximum — shew clinical evidence of the disease? Nobody seems to have given a satisfactory answer to this question, and most people appear to be satisfied to accept such like semi-mysterious, or even mythical, explanations as:— "the human organism does not provide a fertile soil for the growth of the tubercle bacillus": or, "those who succumb to the disease, do so in consequence of a lowered resistance, or an inherited tendency". But, as with all things in life — and especially in the realm of medicine — the simplest explanation is most often the correct one. What is the simplest explanation? Bier, in the year 1892, described his hyperaemic treatment, and taught that the signs of acute inflammation (excepting the pain) were curative and should not be checked, in fact, on the contrary, should be artificially increased. This is the generally accepted view to-day.

Similarly, the rise of temperature which occurs in infectious diseases, was at one time universally thought to be detrimental, and endeavours were made to reduce it; whereas nowadays it is believed to be Nature's protective effort, and no attempt is made to bring it down unless it reaches dangerous heights. I would suggest that the chronic inflammation of pulmonary tuberculosis should come under the same category as these two examples; and this appears to be proved by fact.

There occurs in the first place, Nature's effort in the form of hyperaemia, which is wrongly regarded as the first stage in the pathology of the disease. Far from being pathological, it is curative, and does actually cure some 70 to 80% of cases of pulmonary tuberculosis: i.e. that percentage of tuberculized people who do not shew clinical evidence of the disease. This is very powerful evidence in favour of increasing the hyperaemia, as we do in the treatment of acute inflammation. What therapeutic measure that produced at least 70% of cures in any disease, would not be hailed as practically a specific? In acute inflammation, the "inflammation", in the great majority of cases, would ultimately cure the "infection": and, just as we have a minority progressing to lymphangitis, cellulitis, septicaemia or pyaemia; so, in the case of pulmonary tuberculosis, the minority progress to demonstrable pulmonary tuberculosis, i.e. "lymphangitis", "cellulitis", "septicaemia" or "pyaemia". This then, is the simplest, and apparently the correct explanation of the fact that so relatively few people shew clinical evidence of pulmonary tuberculosis: that although infection is practically inevitable, Nature's production of hyperaemia cures the great majority of cases.

4. Rarity of Pulmonary Tuberculosis among the Native Inhabitants of the Mountainous Districts of Basuto-

land.— The population of the district of Qacha's Nek,⁽³⁷⁾ of which I am Medical Officer is 34,931, and the altitude ranges between 5,000 and 10,000 feet.

During the year 1925, individual patients to the number of 3167 attended at the out-patient department; of which only twenty-six were suffering from pulmonary tuberculosis.⁽³⁸⁾ Of the latter, twenty-two had a history of having worked in the gold or diamond mines, and of having suffered from "miner's phthisis"; so that there were only four cases of true pulmonary tuberculosis. That is to say, presuming that the whole population had as great a proportion of tuberculous patients, as the 3167 who attended at the dispensary, (which is improbable) the incidence of the disease in this district is less than 1% if we include the cases of pneumokoniosis, and just over 0.1% if we exclude them. What can be the reason for this very low incidence of the disease? The uninitiated may imagine that it is due to an abundance of sunshine and fresh air, and an absence of over-crowding; but, although obtainable, the natives do not willingly avail themselves of these conditions. Their houses consist of window-less mud huts, the interiors of which rarely see the light of day. A fire is lighted in the centre of the hut, and there is no outlet for the smoke, except the doorway: and one hut generally provides living and sleeping accommodation for a whole family. Therefore their conditions would appear to be unhygienic in the extreme. I believe the explanation is that the natives' lungs are well supplied with blood, in consequence of the following factors:-

(1) They must take exercise, firstly, since their work is manual, and secondly, because the only two methods of getting about the country are on foot or horseback. The exercise which they are obliged to take, improves their general blood circulation, in-

cluding that of the lungs. This will no doubt be augmented by the plentiful sunshine, to which the natives are exposed, during the time when they are necessarily out of doors.

(2) They live at a high altitude, which increases their blood pressures and deepens their respirations, and therefore the vascularity of their lungs is increased. Also, their red blood corpuscles will be increased in number.

In fifty "healthy" native men, between the ages of twenty-eight and thirty-four years, I found the systolic blood pressure, estimated in the sitting position by the auscultatory method, to range between 130 and 153mm. Hg; the average being 139.8 for an average age of thirty-one years. This is approximately 9mm. higher than the usual normal, for that age, at sea level. For the purpose of this thesis, I consider the systolic pressure to be the more important, because it represents mainly heart strength, and therefore indicates increased vascularity of the lungs. The average diastolic blood pressure was 92.3mm. Hg. Relative to the systolic pressure this is rather low, furnishing further evidence that the high systolic pressure was due to cardiac strength, and not to increased peripheral resistance.

5. A possible Function of the Spleen.— Barcroft's experiments⁽³⁹⁾ shew that increasing the fluid circulating in the blood vessels, by injecting large quantities of normal saline, causes the spleen to become at least twice its previous size, thus reducing the amount of fluid in the general circulation.

According to this observation, it would appear that one function of the spleen is to keep the blood pressure low, by lessening the amount of circulating blood; therefore it is quite conceivable that the increased resistance to tuberculosis, which is stated to accrue after splenectomy,⁽⁴⁰⁾ is due to the re-

removal of this inhibitory influence, allowing the blood pressure to become higher, and therefore to improve the blood circulation. The least that can be claimed, is that these facts strongly suggest that the development, or otherwise, of pulmonary tuberculosis, is dependent upon the condition or amount of the circulating blood.

6. Bactericidal Action of the Blood.— In a person suffering from pulmonary tuberculosis, many tubercle bacilli are bound to get into the blood: in some cases carried by the lymphatics which drain the diseased area, and poured with the lymph into the venous blood; and in those who have haemoptysis, (generally said to be more than 50% of the cases) the bacilli may enter directly into the arterial blood. Despite this inevitable occurrence, only a very small proportion of the cases shew clinical evidence of the disease elsewhere, therefore it appears to be obvious that the blood can, in virtue of its own anti-tubercular powers, destroy the organism. How much more then, can it do this if both its quality and quantity, especially in the lungs, be brought to the highest possible standard.

7. Probable Action of Some Recently Suggested Therapeutic Measures.—

(a) Calcium chloride intravenously, according to Cheinisse, (41) has been found valuable in 200 cases of tuberculosis. The course of treatment consisted of thirty injections, each of ten ounces of a 1% solution, and the typical result was an increase of weight, with improved heart action, and early improvement of symptoms and physical signs.

Now while there may be some truth in the theory, that calcium therapy expedites calcification in the tubercular lesion, what is known regarding the action of the drug, is that it has a general tonic effect, and strengthens the heart beat. Furthermore, the large

amount of fluid used for each injection, would tend to increase the blood pressure, and this, together with the abovementioned actions of the calcium, would increase the vascularity of the lungs. It is to this occurrence that I am inclined to give most credit for any benefit that may have been derived from the treatment.

(b) Sulpharsenol is considered by Guy and Page, (42) to be valuable in some cases, amongst which they include anaemic young adults. I believe that much of the improvement would be due to the well known favourable action of arsenic on the blood.

(c) Sanocrysin was, in the year 1924, introduced by Mollgaard of Copenhagen, (43) who admits that from its use there is a danger, amongst other things, of inflammation of the lungs. This being so, it is, I think reasonable to suppose that it always produces hyperaemia of the lungs, in the cases in which there has been efficient dosage. If this reasoning be correct, accurate dosage would appear to be a very important consideration, and one can well imagine three possibilities.

(1) Inefficient dosage, and consequent non-production of hyperaemia, to have no effect.

(2) Over-dosage, to produce inflammation of the lungs, and possibly death.

(3) Optimum dosage, to produce hyperaemia (short of "inflammation") of the lungs, and consequent improvement in the condition of the patient. These results might vary according to the susceptibility of the patient.

Bang (44) severely criticises the claim that sanocrysin has a bactericidal action, his remarks being based on experimental evidence. He concludes that the gold salts investigated, have no specific action upon tubercle bacilli, and that any curative result found in human tuberculosis must depend on other factors.

De Witt⁽⁴⁵⁾ states that when reactions occur from the use of gold salts, they seem to be associated with marked hyperaemia in the neighbourhood of the tubercles. In accordance with my belief, this hyperaemia would appear to be the curative factor referred to by Bang.

(d) Controlled Diaphragmatic Breathing is described by Knopf,⁽⁴⁶⁾ as a substitute for artificial pneumothorax, and he states that few cases are not benefited by it. "The patient voluntarily reduces the number of breaths to ten a minute, and makes them of the diaphragmatic type. When not using this procedure he should practise shallow breathing."

Now I maintain that it is a physiological impossibility, for a person to voluntarily control either the length, or number of his respirations for any length of time: and that if he tries to shorten them, or reduce their number, he will produce the opposite effect to that at which he is aiming, because he will be compelled to take a very deep breath about every minute, so that, if he keeps this up all day, his lungs will be very well exercised, and in consequence their vascularity increased. This effect will be enhanced by the fact that, the impediment of inspiration will produce a negative pressure in the thorax, and consequent suction of blood from the right side of the heart into the lungs.

By experimenting upon myself and others, I have proved the truth of these statements regarding this type of breathing; therefore, this mode of treatment, as well as "true" artificial pneumothorax, (previously discussed) would appear to act by increasing, rather than decreasing, the vascularity of the lungs.

more especially during the night when these apartments are occupied. The remaining rooms of the house should be well ventilated, and all windows and doors opened for a short period every day. Similarly,

PROPHYLAXIS.

Anything which will ensure an efficient amount of healthy blood circulating through the lungs, and especially the apices, will prevent the onset of pulmonary tuberculosis.

The most important prophylactic measures are the following:-

1. Physical exercises, including breathing exercises.
2. Fresh air.
3. Sunshine, failing which, plenty of daylight.
4. Personal cleanliness, including cold baths.
5. Sufficient nourishing food, including an abundance of water.
6. Regular habits, including sufficient sleep.
7. Efficient treatment of all slight ailments.
8. Complete convalescence after all illnesses.
9. Periodical medical examination of all school children, including blood pressure estimation, and correction of any cause of lowered vascular tone.
10. Management of those already suffering from pulmonary tuberculosis.

(1) Exercises.— Everybody should be encouraged to indulge in outdoor exercises, games and sports; and these, as well as physical drill, should be compulsory at all schools. Breathing exercises and singing should be included in the curriculum of every school.

(2) Fresh Air.— All houses should have verandahs on which people can sleep, and everybody, as far as possible, should sleep out of doors. Where this is impossible or impracticable, bedroom and dormitory windows should be kept wide open during all weathers, more especially during the night when these apartments are occupied. The remaining rooms of the house should be well ventilated, and all windows and doors opened for a short period every day. Similarly,

schoolrooms and workshops should be well ventilated, and all windows constantly wide open.

(3) Sunshine and Daylight.— All buildings, especially dwellings, should be entirely above ground, and admit the maximum amount of daylight. Advantage should be taken of every scrap of sunshine; the infrared rays from which, will improve the person's blood circulation, and the ultra-violet rays (if the sun shines through open windows) will kill any micro-organisms which may be lurking in the room.

(4) Personal Cleanliness, including Cold Baths.— Attention to personal cleanliness will remove any bacilli which may be harbouring about the body or clothes. Cold baths, or if these cannot be tolerated, cold douches will tone up the system generally, and improve the blood circulation.

(5) Food and Water.— It should be made possible for everybody to get sufficient nourishing food. Most people drink far too little water, and suffer in consequence. The tissues become more or less dehydrated; there is constipation, with its toxæmia undermining the general health, as a result of insufficient water in the intestine; and the volume of blood tends to be less than it should be, thus lowering the blood pressure, and lessening the blood supply to the various organs including the lungs. Therefore everybody should drink much water or other bland fluids, which would remove all the above undesirable and devitalising factors.

(6) Regular Habits, including Sufficient Sleep.— A sufficiency of sleep is the most important, and will greatly increase the resistance to tuberculosis, as well as to other diseases.

(7) Efficient Treatment of All Slight Ailments.—

This is especially important in children and adolescents, particularly in regard to "colds", sore throats, digestive disturbances, anaemia, whooping cough, measles and in fact, anything debilitating.

(8) Complete Convalescence after All Illnesses.—

This also, is especially important up to the age of adolescence. Often, after an illness, the patient is left devitalised, and with lowered vascular tone, therefore the doctor should remain in attendance, until the patient's health reaches the highest possible standard.

(9) Periodical Medical Examination of All School

Children.— I am aware that school children are at present medically examined, but I doubt whether sufficient attention is paid to the condition of their blood, the state of their vascular tone, nutrition and physical development, and the nature of their breathing. By these signs incipient pulmonary tuberculosis could be detected, at which stage I believe the disease to be absolutely curable by the simple procedures outlined in this and the next section.

Any child found with the slightest degree of anaemia, or enfeeblement of circulation; or with poor physical development, especially with regard to the thorax, such as "flat chest", "round shoulders" or "deficient expansion", should be seriously taken in hand at once, and have these defects energetically and thoroughly treated. Children suffering from mal-nutrition should be dealt with similarly.

(10) Management of Those already suffering from Pulmonary Tuberculosis.— The precautions which should be taken are obvious, but very difficult of attainment. However, if the foregoing prophylactic

measures be utilised, there will be little to fear from this source; but a method of treatment will later be described, which would, incidentally, render sputum of those suffering from pulmonary tuberculosis relatively harmless. *realising at what they are aiming. Again, Osler⁽⁴⁵⁾ says, "as a rule, make a patient grow fat and strong, and the local disease may be left to take care of itself".*

The important point **TREATMENT.** *the general health*
 Specific and "laboratory" treatment of pulmonary tuberculosis has had a fair trial, and I am of the opinion that as much could have been attained without it, since the fall in the incidence and mortality of the disease, was apparent before any of the methods came into being. *no vascularity of the lungs will im-*
 The use of such substances as tuberculins, mixed infection vaccines, pneumosan, Dreyer's vaccine, *be* Spahlinger treatment, aurocyanide and sanocrysin certainly may, and often have, effected improvement; but this is very uncertain, and many believe that much the same results could be obtained by the use of any foreign protein. Danysz⁽⁴⁷⁾ states that in tuberculosis, the hypodermic or intradermal injection of cow's milk may produce the same pathologic and therapeutic reactions as tuberculin. *they would obtain bet-*
 Linnell⁽⁴⁸⁾ states that he agrees with an eminent bacteriologist, who recently said that nothing further could be expected from vaccine therapy. I incline to this view; therefore I think the time is ripe for the energetic trial of some totally different method. The treatment now to be advocated is based on the assumption that my hypotheses are proved, and resolves itself into the use of any substance or method that will improve the quality, or increase the quantity, of blood circulating through the lungs, more especially in the "threatened" or diseased part. The treatment of pulmonary tuberculosis is often

summed up in the following phrase:- "Bring the general health up to the highest possible standard." With this end in view physicians prescribe "tonics", and attend to the patient's digestion, but entirely miss the mark through not realising at what they are aiming. Again, Osler⁽⁴⁹⁾ says, "as a rule, make a patient grow fat and strong, and the local disease may be left to take care of itself".

The important point is that when the general health is better, the blood, vascular tone and blood pressure are also better, therefore there is more blood, of a better quality, circulating through the lungs. These are indisputable facts, therefore it is difficult to account for the belief, held by many to-day, that lessening the vascularity of the lungs will improve the pulmonary condition. The greater and better blood circulation in the lungs, would appear to be the beneficial factor; therefore the above phrase should be altered to the following:- "Bring the blood supply of the lungs, in quality and quantity, up to the highest possible standard"; then the result would be nearer to perfection, since it would be at what had been aimed, and not merely an incidental occurrence. This phrase should be the slogan of all medical men, then, I am confident, they would obtain better results, even if for no other reason than that they would have something definite at which to aim. Compare this picture with what obtains at present. The tuberculous patient is really very little better off than he was fifty years ago. He still gets his tonic and cough mixture, and perhaps in addition, an injection of something or other, the action or result of which, nobody professes to foretell.

My own opinion is that the fall in the tuberculosis mortality, is due to a more or less parallel fall in the incidence of the disease, rather than to its treatment.

Medical men must keep constantly before their minds the effects, as herein indicated, which they expect from the treatment which they prescribe, and if these effects are not being produced, the treatment should be altered or augmented at once. Every medical man knows of the means available to improve the quality, or to increase the quantity, of blood circulating through the lungs: it remains for me only to emphasise a few of the more important, or debatable points. I will not discuss in detail any particular form of treatment, which has been fully considered in a previous section.

Among the "treatments" which will produce, or tend to produce, the desired effect, may be mentioned the following:-

(A) Hygienic.

1. Physical exercises, and outdoor games and sports.
2. Breathing exercises, especially impeding of inspiration, as previously described.
3. Living in the fresh air, day and night.
4. Sunshine and daylight.
5. Personal cleanliness.
6. Cold baths, or cold douches.
7. Regular habits, including sufficient sleep.

(B) Dietetic.

1. Sufficient nourishing food.
2. Abundance of water, or other bland fluids.

(C) Medical.

1. Light therapy. (Fully discussed already.)
2. Poultices, or hot fomentations.
3. Drugs.
 - (a) Strychnine and digitalis, to tone up the heart and vascular system.
 - (b) Iron and arsenic, for the anaemia.
 - (c) Pituitrin, adrenalin and ergot, may be used

to raise the blood pressure.

- (d) Iodine, in some form, to produce hyperaemia of the lungs; to improve the lymph circulation; for its germicidal properties etc.

Water.— The importance of an abundance of water, or other bland fluids, cannot be over-estimated. It will prevent the tissues from becoming dehydrated and shrivelled: it will lessen constipation and its toxæmia, by allowing sufficient water for the intestinal contents; and also purify the blood, by ridding it of the toxins of the tubercle bacillus. These benefits will greatly improve the patient's general condition, and considerably increase his resistance to the disease. Furthermore, the total volume of circulating blood will be greater than otherwise, and will therefore tend to make the blood pressure higher, and in consequence there will be more blood going to the lungs, and the lymphatic drainage will be improved.

Poultices or Hot Fomentations.— If a large poultice or fomentation be applied over, say, a diseased lung apex, it will certainly produce hyperaemia of the skin, and will tend also to increase the vascularity of the underlying lung.

Pituitrin, Adrenalin and Ergot.— While the effects of these drugs in raising the blood pressure are somewhat transient, and their value perhaps debatable, a method of administration may be discovered by which they would be found to produce the desired effect in the lung. For example, ergot has long been prescribed, more or less empirically, for hæmoptysis; but Cushny⁽⁵⁰⁾ believed it to be useless for this purpose, therefore it is possible, since the drug is known to cause constriction of blood vessels in other regions, that it may thereby actually increase the vascularity of the lungs.

Iodine.— Although contrary to many authoritative opinions, I believe that iodine, in some form, will be found to be more useful, and to produce better results in the treatment of pulmonary tuberculosis, than any other drug.

Many authorities deprecate the use of iodine or the iodides in the treatment of this disease, because they may apparently increase the cough and expectoration, and also the crepitations. Even if these effects are produced, I cannot see that they indicate that the tubercular process is being made worse: they are merely signs of the physiological action of the drug; signs that hyperaemia of the lung, which will ultimately be beneficial, is being produced.

There is also said to be a danger of oedema of the lungs, but I am very sceptical regarding this. I think the cause of this belief, is merely that the thoughts of those who believe it, are centred on the lung disease, and they, in consequence, magnify the danger. One does not hear anything of oedema of the lungs as a result of the treatment of syphilis, in which the maximum dose of iodide is given without any thought of danger, or any untoward results except in very occasional cases: and before the discovery of salvarsan, enormous doses of iodide were given — often as much as one dram three times daily — therefore I consider that this risk can be discounted.

Referring to the treatment of pulmonary tuberculosis, Sajous⁽⁵¹⁾ says, "in the early stages, local application of iodine over the threatened or diseased area is of great service". How it acts, when thus applied, is probably a matter of doubt. It does produce hyperaemia of the skin, and may also have the same effect on the underlying lung. Again, since iodine is absorbed through the skin, it may produce hyperaemia of the lungs just as it would if taken orally: similarly there may be some antiseptic action after

absorption. Suberous children, may be markedly Joseph⁽⁵²⁾ endorses Bier's statement in regard to the great advantage of giving internally sodium iodide, to supplement the local hyperaemia in the treatment of surgical tuberculosis. mononuclear Salomon⁽⁵³⁾ similarly found potassium iodide to enhance the treatment. suggest most strongly, that There would appear to be no doubt that, given internally, iodine will produce hyperaemia of the lungs, and this is presumably the cause of the fear of pulmonary oedema. of pulmonary tuberculosis, a gradual As previously stated, iodine acts as a lymphagogue, thereby improving the lymphatic drainage and circulation. The lymph, as well as the blood, will contain iodine, as also will the lymph glands, which⁽⁵⁴⁾ take up and store iodine abundantly. Now pulmonary tuberculosis is spread in the lungs greatly by the lymphatics, the bacilli being temporarily held up in the lymph glands: therefore if the lymph conveys the bacilli, the iodine in the lymph and lymphatic glands, owing to its germicidal action, will inhibit the activity of the bacilli, and thus tend to prevent the spread of the disease, and to arrest its progress. Another effect of iodine, according to Lortat-Jacob,⁽⁵⁵⁾ is that of markedly stimulating lymphoid tissue in general, as well as serous membranes, the result being a pronounced increase in the production of lymphocytes. even it is not preponderating too much to Further, referring to the treatment of surgical tuberculosis with iodoform, Thomson and Miles⁽⁵⁶⁾ say, "its curative effect would appear to depend upon the liberation of iodine, which restrains the activity of the bacilli, and upon its capacity for irritating the tissues and so inducing a protective leucocytosis, (meaning lymphocytosis presumably) and also of stimulating the formation of scar tissue". Again, Barbier⁽⁵⁷⁾ states that the general hygienic

treatment of tuberculous children, may be markedly reinforced by the use of 10% tincture of iodine, of which he gives ten drops daily per year of the child's age: and further, that the drug is well borne, and always increases the large mononuclear cells.

The above observations suggest most strongly, that iodine is a specific excitant of defensive lymphocytic activity.

Great stress has been laid on the lymphocyte index⁽⁵⁸⁾ in the prognosis of pulmonary tuberculosis, a gradual rise of which is favourable, while it becomes lower and lower as the disease advances. Now it is apparently presumed that the fall in the lymphocyte index, is due to the tubercular process becoming worse, but, consideration of the above evidence for the production of lymphocytes by iodine, and also, as previously stated, by heliotherapy, would appear to prove that the betterment or otherwise in the patient's condition, to some extent at least, is dependent upon the presence or absence of the said lymphocytosis: therefore it is quite conceivable that a gradual failure to produce lymphocytes, is a causative factor in the progress of the disease.

In ordinary lobar pneumonia, nucleic acid will cause a leucocytosis and so bring a hopeless case into the category of a moderately severe one; therefore, as an analogy, I believe it is not propounding too much to say that the production of a lymphocytosis by the exhibition of iodine, would similarly improve the prognosis in pulmonary tuberculosis.

To summarise the advantages of iodine in the treatment of pulmonary tuberculosis, according to the evidence stated herein, they are:-

1. Production of a beneficial hyperaemia of the lungs.
2. Improved lymphatic drainage and circulation.

3. Production of a beneficial lymphocytosis.
4. Restraint of the action of the bacilli, in virtue of the germicidal action of iodine.
5. Stimulation of scar tissue formation.

Details of Treatment of a Suspected, Incipient, or Early Case of Pulmonary Tuberculosis.— It is essential to enlist the intelligent co-operation of the patient, as so much depends upon himself. His interest should be aroused, and he should be told why he must follow the instructions implicitly.

Instructions to Patient, and Routine Treatment.—

1. Eat as much nourishing food, and drink as much water or other bland fluids as possible.
2. Spend as much time as possible in the open air, and always sleep out of doors.
3. Do not wear anything, such as braces, which would hamper or restrict apical respiration.
4. Respirations to be made thoracic in type, and as deep as possible.
5. Take breathing exercises at least twice daily; ordinary deep breathing, and also the type which impedes inspiration.
6. Take physical exercises daily; especially those which will strengthen the muscles of the neck, back, and abdomen, to enable the head to be held erect, the shoulders back, and the abdominal wall flat. These exercises will increase the capacity of the thorax.
7. Indulge in outdoor games, sports and labour, which will be graduated by the medical attendant.
8. Wear a waist-belt, rather tightly if necessary. This will remind the patient to breathe thoracically, and also help him to hold himself erect.
9. Acquire regular habits, and take meals regularly.
10. Obtain sufficient sleep. Sleep either on the back with a low pillow, or on the sound side.

11. Take a cold bath or cold douche every morning, followed by a brisk rubbing.
12. The cleansing bath to be taken warm, and at night.
13. Apply a poultice over the affected area of the lung just after going to bed. The poultice may be removed later if it keeps the patient awake.
14. Paint the chest, over the affected area of the lung, with tincture of iodine every morning, treating the back and front on alternate days.
15. Heliotherapy, or preferably, infra-red radiations.
16. Iodine, in some form, internally.
17. Iron, and maybe arsenic, for the anaemia.
18. Strychnine, digitalis etc. if necessary, to stimulate the cardio-vascular system.
19. Treat constipation, but avoid making the motions fluid.
20. Symptomatic treatment according to general principles.

The instructions to the patient will be subject to variation according to age, intelligence etc.

The treatment of moderately advanced, advanced, and acute cases may need some modification, according to the condition of the patient. Also, it may be necessary to augment, or partly replace, this treatment, by one or more of the therapeutic measures to be described in the next section.

COINCIDENT OR ALTERNATIVE THERAPEUTIC MEASURES.

(A) Intrathoracic Injections of Iodine.

This method of treatment occurred to me as being practicable, and as having great possibilities of success. I was therefore greatly surprised to learn that it had been previously employed. Referring to this mode of treatment Sajous⁽⁵⁹⁾ says,

"although the cases were more or less advanced, and other methods had afforded but little encouragement, very distinct amelioration of their condition was obtained. The injections were well borne: no haemorrhage was caused, even where haemoptysis had previously occurred. Two of the patients were children, aged four and ten years, respectively. The injections caused disappearance of the tubercle bacilli from the sputum, gradual cessation of the fever and cough, all other physical signs being also gradually eliminated. Open air exercise and the patients' daily vocations being in nowise interfered with, the cases progressed steadily toward recovery while earning their livelihood." Sajous injected the material into the centre of the lesion.

In view of this apparent success, it is a matter of surprise to me that the method is not more widely practised. I presume the unwarranted prejudice against iodine in the treatment of pulmonary tuberculosis, has much to do with this reluctance.

Rankin and Weigel⁽⁶⁰⁾ report successful chemical sterilisation of tuberculous cavities by means of gentian violet and methylene blue, introduced through surgically established fistulae. I think the establishment of fistulae is unnecessary, and that iodine is preferable to these dyes, in fact preferable to anything, in consequence of its beneficial actions in addition to that of a bactericide.

Since iodoform emulsion has been so successful in the treatment of surgical tuberculosis, I think it would be the best means of administering the iodine.

d'Amico⁽⁶¹⁾ preferred an iodoform emulsion containing guaiacol, but since it is the action of the iodine we anticipate, there seems no reason to add anything to it.

Technique.— A knowledge of the anatomy of the lung will enable the operator to avoid the larger blood

vessels. If a needle of very fine calibre be used, there will be practically no danger of producing pneumothorax, and in any case, the visceral pleura will probably be adherent to the chest wall.

The main lesion, such as an area of consolidation, or a cavity, should be accurately located by clinical methods, including the study of X-ray photographs taken at various angles.

The "healthy" lung around the lesion should be injected first, so as to iodize it, and thus tend to prevent the spread of the disease. As the emulsion is being slowly expelled from the syringe, the needle should be slowly withdrawn, to prevent the possibility of the whole of the medicament being deposited into an air vesicle or bronchiole: also so that the emulsion will be deposited all along the needle track, and thus some of it will enter directly into the interstitial tissue and lymph vessels, and also into any stray tubercles which may have been penetrated by the needle. This procedure will also ensure getting some iodoform into the superficial lymph vessels of the lung, which, according to Miller, (62) (although his assertion would appear to have been disproved) only very rarely anastomose with the deep lymph vessels.

An attempt should next be made to inject the walls of cavities, and the growing exterior of consolidated areas. The chance of success will be enhanced by withdrawing the needle as above described.

Finally, the principal lesion, a consolidated area or a cavity, should be injected. When a cavity is being injected, the patient should, as far as possible, lie on the affected side; and for an hour or so after the injection, he should lie on that side to allow some time for absorption of the iodine, and to prevent the possibility of the emulsion immediately entering a bronchus.

If a little bismuth be added to the emulsion, an X-ray photograph would shew whether or not the emulsion was in the right place.

The skin puncture may be sealed with collodion.

The injections should be repeated as frequently as possible, and continued for some considerable time after the patient is apparently well.

Advantages of the Method.— In addition to the beneficial effects of iodine as described in the preceding section, this method will also tend to sterilise the sputum, and will bring the iodine into contact with certain tubercle bacilli, which might possibly escape the bactericidal action of iodine otherwise administered.

According to Kinnaman, (63) a 1-100 solution of iodine kills the tubercle bacillus in seven minutes, and the pyogenic cocci, (which take part in the mixed infection of pulmonary tuberculosis) in greater dilution and less time.

As the lymph is drained away from the tuberculous area, and the adjacent areas which have been injected, it will contain iodine, which will overtake and destroy any commencing tubercle formation. The iodine will also be taken up and stored by the lymphatic glands, thus forming a defensive barrier. Thus it will be seen that apart from any curative properties, the iodine will prevent, or tend to prevent, contiguous and lymphatic spread of the disease within the affected lung.

Again, since the sputum will be sterile, or at least less infective than otherwise, there will be considerably less risk, if any, of aspiration spread of the disease to the other lung, or to other parts of the same lung. Similarly, there will be the same protection against laryngeal, oral and intestinal tuberculosis. Lastly, there will be little or no danger of spread of infection to other people by the medium of

dried sputum. It should be noted, that even if the sputum be expectorated before the iodine has had time to sterilise it, it will contain iodoform, which will liberate iodine and thus greatly reduce its infectivity.

(B) Artificial Pneumothorax.

There would appear to be plenty of evidence that this procedure often greatly improves matters, and, believing it to act in the manner which I have described, I would advocate its use if more conservative measures fail.

(C) Removal of the More Involved Lung.

I am aware that this operation is a very serious one, and that it has been abandoned. Nevertheless, I believe there are cases in which it may be indicated. For instance, a case in which all treatment, including artificial pneumothorax, had failed to check its progress, and in which an early death was obviously to be the termination. I think there would be much more chance of recovery as a result of this operation, than from such operations as phrenicotomy and thoracoplasty, after artificial pneumothorax had failed. The operation would entirely remove the greater part of the disease, and also therefore, a great proportion of the bacilli and their toxins, and thus greatly improve the patient's general condition. In addition to this, and, I believe of great importance, the remaining lung would receive much more blood than previously, which might even cure the disease if not too far advanced in that lung. The case would also then be more amenable to other forms of treatment.

(D) Production of Artificial Mitral Stenosis.

The effects of a partial mitral stenosis, could be obtained by an operation on the pulmonary vein or

veins, whose tributaries arose in the involved lobe or lobes. The operation should be of such a nature as to make the lumen of the vein smaller; and the consequent backward pressure of blood would involve only, or mainly, the lobes whose veins had been dealt with. This of course would be a difficult operation, and the mortality would no doubt be high; but, if my assumption be correct, the results, regarding the lung disease, would be good: and, as with the previously suggested operation, there may be some cases in which the risk would be worth while.

consequence of anæmia, low blood pressure, lack of exercise, (physical or pulmonary) insufficient food or fresh air, inefficient treatment of some illness, or some other factor which has, directly or indirectly, further decreased the vascularity of the lungs. This is borne out by my study of a tuberculous family in which I found that those who had escaped the disease were of the plethoric, full-blooded type and of good physique; while those who had succumbed were pale, delicate, straggling and of poor physique. Similarly, it has been observed, after a study of five thousand blood relatives, that three-fourths of the non-tuberculous offspring of tuberculous parents, had been in just as close contact with active open cases, as their brothers and sisters who developed the disease. I believe that those who escaped the disease, and those who did not, will be differently constituted in the same manner as the members of the family which I studied.

The anæmia and low blood pressure which obtain in pulmonary tuberculosis, although no doubt aggravated by the disease, I believe to be primary in nature, and of great etiological importance.

There is evidence to show that the type of person liable to contract pulmonary tuberculosis, is likely

SUMMARY.

Etiological and General.

Important points in the etiology of pulmonary tuberculosis are the normal slowing of the blood stream in the lung capillaries, and the normal poor blood supply of the lung apices. But for these two factors, I believe pulmonary tuberculosis would be a rare disease. In the person who succumbs to the disease, instead of this poor normal having been maintained at its best, it has been allowed to become worse, in consequence of anaemia, low blood pressure, lack of exercise, (physical or pulmonary) insufficient food or fresh air, inefficient treatment of some illness, or some other factor which has, directly or indirectly, further decreased the vascularity of the lungs. This is borne out by my study of a tuberculous family, in which I found that those who had escaped the disease were of the plethoric, full-blooded type and of good physique; while those who had succumbed were pale, delicate, exsanguious and of poor physique. Similarly, it has been observed, after a study of five thousand blood relatives, that three-fourths of the non-tuberculous offspring of tuberculous parents, had been in just as close contact with active open cases, as their brothers and sisters who developed the disease. I believe that those who escaped the disease, and those who did not, will be differently constituted in the same manner as the members of the family which I studied.

The anaemia and low blood pressure which obtain in pulmonary tuberculosis, although no doubt aggravated by the disease, I believe to be primary in nature, and of great etiological importance.

There is evidence to shew that the type of person liable to contract pulmonary tuberculosis, is likely

to possess a low blood pressure, dependent upon a deficient thoracic capacity, and independent of any disease therein.

Even though anaemia, in the sense of a deficiency of blood elements, may not have existed prior to the lung disease, there certainly will have been a "pulmonary anaemia" where the lesion has developed.

Further, there are certain diseases which, to a great or lesser extent, predispose to pulmonary tuberculosis, and these without exception, directly or indirectly, according to evidence adduced, reduce the vascularity of the lungs. The outstanding example of these diseases is congenital pulmonary stenosis, the sufferers from which are extremely liable to contract, and die from, pulmonary tuberculosis. Other diseases, predisposing in various degrees, are anaemia, syphilis, measles, whooping cough, diabetes mellitus, insanity, gastro-intestinal diseases and alcoholism.

Conversely, some diseases appear to confer varying degrees of protection against the tubercle bacillus. In this series, mitral stenosis is the outstanding example. In so far as the vascularity of the lungs is concerned, this is the antithesis of congenital pulmonary stenosis: and, as might be expected, the degree of protection which it affords, is as great as the predisposition to the disease in the other case. This fact alone is very significant. Other diseases affording protection against pulmonary tuberculosis are, other varieties of acquired valvular disease of the heart, asthma, emphysema, gout and pneumonia. I have adduced fairly convincing evidence that these diseases do afford protection, in each case by increasing the vascularity of the lungs.

Regarding mitral stenosis there can be no doubt. Emphysema and asthma produce the desired effect by causing a negative pressure in the thorax, and consequent suction of blood from the right side of the heart into the lungs. It is not the emphysema as such, but its causes, which produce this effect, during the age period when the patient is most liable to contract pulmonary tuberculosis.

Gout is beneficial in consequence of the accompanying high blood pressure.

In pneumonia, since one attack predisposes to another, the patient would appear to have normally hyperaemic lungs; and it is said that pulmonary tuberculosis rarely supervenes on an ordinary pneumonia. If this sequence apparently occurs, the pneumonia will probably have been tuberculous in origin.

Evidence as to the favourable influence of increased vascularity of the lungs when pulmonary tuberculosis is manifest, may be obtained from a consideration of the therapeutic measures which are known to have produced good results in the treatment of the disease. Of these may be mentioned sufficient food, fresh air, residence at a high altitude, graduated exercises or labour, a sufficiency of sleep, light-therapy, hydrotherapy, artificial pneumothorax, iodine, iron and strychnine. I have given evidence to shew that all of these measures increase the vascularity of the lungs. With regard to some of them, their action may probably be debatable, but I have already discussed them in detail, and the more important points will be touched upon in a more appropriate section of this summary.

Again, some recently suggested therapeutic measures, when beneficial in the treatment of pulmonary tuberculosis, would appear to act differently from that

which is generally believed to be the case; namely, by improving the condition of the blood, or inducing hyperaemia of the lungs. Those which I consider to do this are calcium, sulpharsenol, sanocrysin and controlled diaphragmatic, or shallow, breathing. Calcium has a general tonic effect, and strengthens the heart beat.

Sulpharsenol contains arsenic, which is tonic, and acts favourably on the blood.

The avowed bactericidal action of sanocrysin has been denied on experimental evidence, and any benefit accruing from it attributed to some other factor. It causes hyperaemia of the lungs, and this I believe to be the beneficial factor.

Regarding "controlled breathing", I have proved experimentally, that it is a physiological impossibility for anybody to voluntarily take shallow inspirations, or a fewer number than usual, for any length of time. If a patient attempts to take fewer, or shorter, breaths than usual, he will be compelled to take a deep breath about every minute, thus well exercising his lungs, and increasing their vascularity. This effect will be augmented, by the impediment of inspiration causing a negative pressure in the thorax, and consequent suction of blood into the lungs. Good results are claimed for this method, on the assumption that it lessens the blood circulation in the lungs. I do not doubt the good results, in fact I would expect them, but they are in consequence of the exactly opposite condition from that which is claimed, namely, increased vascularity of the lungs.

There has occurred a progressive decline in the death rate of pulmonary tuberculosis, hand in hand with a change in conditions which will have, on the whole, improved the vascularity of the lungs of the

general population. These changes are, increased wages and consequent better feeding and housing; the appreciation of the value of fresh air; improved hygiene and sanitation; a more strenuous existence and more active outdoor life; a growing popularity of outdoor sports and games; and in short, a progressive increased activity of the people.

The incidence of pulmonary tuberculosis in the mountainous district of Qacha's Nek, the altitude of which ranges between 5,000 and 10,000 feet, is less than 1%; and, excluding cases of pneumokoniosis, only 0.1%. This low incidence, I attribute to the fact that the inhabitants have, as a class, high blood pressures and increased vascularity of their lungs, in consequence of the high altitude, and the great amount of exercise which they necessarily must take. In fifty "healthy" native men, I found the average systolic blood pressure to be 139.8mm. Hg, for an average age of 31 years.

There would appear to be abundant evidence, that pulmonary tuberculosis ensues in consequence of an avascular condition of the lungs; also that if the pulmonary blood supply be maintained at its best, tuberculosis of the organ will not develop; and that improving the vascularity, or inducing hyperaemia, of the lungs, (even incidentally and by haphazard methods) greatly improves, and often cures, pulmonary tuberculosis.

Prophylactic and Therapeutic.

Anything which will ensure an efficient amount of healthy blood circulating through all parts of the lungs, will prevent the onset of pulmonary tuberculosis. Consideration of the adduced evidence causes one to conclude that the beneficial increased

vascularity, or hyperaemia, may be either active or passive.

The most important prophylactic measures are:-

1. Physical exercise, including breathing exercises.
2. Fresh air.
3. Sunshine, failing which, plenty of daylight.
4. Personal cleanliness, including cold baths.
5. Sufficient nourishing food, including an abundance of water.
6. Regular habits, including sufficient sleep.
7. Efficient treatment of all minor ailments.
8. Complete convalescence after all illnesses.
9. Periodical examination of all school children.
10. Management of those already suffering from pulmonary tuberculosis.

Fresh air, sunshine, daylight, cold baths, sufficient sleep, food and water, all, directly or indirectly, improve the pulmonary circulation, and also the general condition of the patient.

Outdoor sports, games, and physical and pulmonary drill, should be compulsory at all schools. If these be indulged in from early school-days; and children be periodically examined for evidence of anaemia, lowered vascular tone, or such conditions as would cause "pulmonary anaemia", and when found, remedied; and if a thorough convalescence be obtained after all illnesses, no matter how trivial they apparently may be, then the incidence of pulmonary tuberculosis would be very materially diminished: in fact, I venture to suggest that it would soon be relegated into the category of rare diseases. Furthermore, if this regime obtained, there would be little to fear from the danger of dried tuberculous sputum.

proves that these natural cures must amount to

The real danger to life from tuberculosis arises when the disease involves the lungs, owing to its inevitable spread throughout these organs in a great

majority of clinical cases. I believe this spread could be prevented, by placing the treatment of the disease upon a sound and rational basis.

Specific and "laboratory" treatment of pulmonary tuberculosis has had a fair trial, and I am of the opinion that as much could have been attained without it, since the fall in the incidence and mortality of the disease, was apparent before any of the methods came into being. The use of these various specific and chemical substances may, and undoubtedly has, effected improvement, but their action is very uncertain: and it is stated that the same results have been obtained with injections of cow's milk. I believe that, in common with some of the more recently suggested therapeutic measures, any benefit derived, may have been from the occasional incidental production of increased vascularity of the lungs, often maybe, by means of some coincident treatment.

By the production of hyperaemia, Nature cures all those people, who are attacked by the tubercle bacillus but do not shew clinical evidence of the disease. In a smaller number, Nature similarly cures them by the time their disease is beginning to become clinically manifest. In the remaining relatively small proportion who develop clinical pulmonary tuberculosis, Nature has presumably failed to produce a sufficient degree of hyperaemia, owing to the poor supply of blood available in the attacked parts of their lungs; therefore, if Her efforts were augmented, I believe pulmonary tuberculosis would almost always be cured. Clinical and post-mortem evidence proves that these natural cures must amount to at least 70% of those infected with the tubercle bacillus. Now if any treatment cures 70% of cases of any disease, it is surely worthy of a thorough trial



in the remaining 30%, and I think it could be used with a degree of confidence amounting almost to certainty, regarding the result.

The treatment which I would advocate is based on the same principles as the suggested prophylactic measures; and resolves itself into the use of any substance or method which will increase the vascularity of the lungs, and especially in the diseased area. Consideration of the various treatments which are known to have benefited tuberculous patients, (such as sufficient food, fresh air, residence at a high altitude, sunshine and graduated exercises) proves beyond doubt that this method has really long been in vogue, but the fact has apparently not been appreciated by those administering the treatment. The results that have accrued, also prove the efficacy of this hyperaemic treatment, even when it has been only an occasional and accidental occurrence; therefore, if the principle were applied thoroughly and with intent, the benefit would be proportionately increased. I would emphasise some therapeutic measures, the action or utility of which, might appear to be debatable.

The importance of an abundance of water cannot be overestimated. It will prevent the tissues from becoming dehydrated and shrivelled: it will lessen constipation and its toxæmia, and also rid the blood of tubercle toxins. These factors will greatly improve the patient's general condition, and considerably increase his resistance to the disease. Furthermore, the circulating blood will be greater in amount than it would be otherwise, and will therefore tend to make the blood pressure higher, and thus further increase the vascularity of the lungs, and improve the lymphatic drainage.

Of the efficacy of light-therapy there would appear to be no doubt, but, as in previously cited instances, the benefit resulting is attributed to the wrong factor. It is generally believed that the ultra-violet rays are responsible, in some mysterious way, for the betterment in the patient's condition, but I believe it is really due to the infra-red radiations, which emanate from the source which is utilised for the application of ultra-violet radiations. There is considerable evidence in support of my contention. The infra-red rays penetrate the most deeply, and induce hyperaemia in the underlying tissues. Since the blood is heated, the lungs are made more vascular, whether they are treated directly or indirectly, but the direct method would appear to be the more rational. It has been stated, on the strength of clinical observation, that the less liable a tuberculous patient's skin is to pigmentation, the more benefit will he derive from heliotherapy. Although this is contrary to Rollier's original theory, it is in accordance with the fact that "white" people are more amenable to heliotherapy than are "black" people. Therefore, since the ultra-violet rays are responsible for the production of pigmentation, it is hardly reasonable to give them credit for the therapeutic effects: these would appear to be due rather to the infra-red rays, the action of which is produced in inverse ratio to the presence of pigmentation. In view of these facts, one would advocate the use of the carbon-filament incandescent lamp in the treatment of pulmonary tuberculosis, the glass bulb of which will filter out the ultra-violet rays, and thus ensure the full benefit of the infra-red rays.

There has been much reluctance to use light-therapy in the treatment of this disease, in consequence of the belief that it may cause haemoptysis, but

experience appears to have proved this belief to be erroneous, and to be founded on bad technique.

Despite many authoritative opinions to the contrary, I believe that iodine, in some form, will be found the most useful drug, and to be very beneficial, in the treatment of pulmonary tuberculosis.

I have given good reasons for stating, that the belief that the drug may cause oedema of the lungs is fallacious.

In addition to claims of successful treatment by the internal administration of iodine, its external application would also appear to be of great service. The beneficial action of iodine would appear to be essentially due to the production of hyperaemia of the lungs. Other advantages are, improved lymphatic drainage and circulation, production of a beneficial lymphocytosis, restraint of the action of the tubercle bacilli, and stimulation of scar tissue formation. When administering the drug, the patient should be examined to see if these effects are being produced, and if they are not, the dose should be increased in frequency or amount, until the desired effect is produced.

Iodine, like heliotherapy, induces a lymphocytosis, and since a fall in the lymphocyte count indicates a bad prognosis, it is possible, in fact probable, that the lymphocytes in some manner, and to some extent, control the development of the disease. Iodine, by inducing a lymphocytosis in pulmonary tuberculosis, may quite conceivably act in the same beneficial manner, as nucleic acid does by producing a leucocytosis in pneumonia.

There is evidence to shew that intrathoracic injections of iodoform emulsion are very efficacious in the treatment of this disease. This is not surprising, since it is so successful in the treatment of

surgical tuberculosis. In addition to the aforementioned advantages of iodine, this method has the additional advantage of tending to sterilise the sputum, thus protecting the patient from tuberculosis elsewhere, including aspiration spread, and also eliminating the danger to others from dried sputum.

The good results claimed for artificial pneumothorax, are generally attributed to the fact that the lung is at rest, but I am doubtful whether this is the beneficial factor. In the type of case in which it is generally said to be indicated, there will probably be some pleuritic adhesions over the lesion, especially if the lesion be superficial. In consequence, this portion of the lung will not collapse, but, on the contrary, will become more vascular. A large number of cases is reported, in all of which, collapse of the lung was obtained, but in about only 50% was it therapeutically successful. These would presumably be the cases with adhesions over, and consequent hyperaemia of, the lesion. If rest of the lung were the curative factor, one would expect a more uniform result; whereas, if hyperaemia were the beneficial agent, one would expect good results in only that proportion with moderate adhesions, that is, approximately, the numerical result which was obtained. This contention is supported by the statement, based on experience of over 800 cases, that disease in the contralateral lung, contrary to general belief, is not a contraindication to the production of artificial pneumothorax; and that its production causes a coincident improvement in the condition of the uncollapsed lung, which would certainly be hyperaemic. This latter statement is very strong evidence, in favour of the hyperaemic treatment of pulmonary tuberculosis. Believing artificial pneumothorax to act in this manner, I would advocate its

use when more conservative measures fail.

Removal of the more involved lung, may occasionally be indicated when other treatment has failed. It would have the advantage of entirely removing a greater part of the disease, and of producing hyperaemia of the remaining lung, which may possibly cure the disease.

Similarly, it may happen that production of an artificial mitral stenosis, by an operation on a pulmonary vein, may be thought worth while. This would produce hyperaemia of the affected lobe or lobes.

Of the efficacy of the production of hyperaemia in the treatment of pulmonary tuberculosis, there is apparently no doubt: and since what has been attained is in consequence of only indirect methods, and more or less accidental, there would appear to be a strong case for the energetic application of this form of treatment. The obvious conclusion is that results would be infinitely better, both as regards prophylaxis and treatment, if the preventive and curative factor were recognised, and produced in all cases with certainty.

The words of my hypotheses, which I believe to be proved, most effectually summarise my conclusions:— The development of pulmonary tuberculosis in man, is made possible solely in consequence of "anaemia" (in the sense of a deficient supply, either of whole blood, or blood elements) — natural or acquired — of the lungs.

If this "pulmonary anaemia" be prevented, or, if already existing, cured, tuberculosis of the organ will not ensue.

An established tuberculosis of the lungs — at least in the early stages — can be cured; and greatly

improved in practically any stage, by the efficient treatment of the pre-existing "pulmonary anaemia".

2. Philip, R. W.: *Dist. of Pract. Med.*, Vol. 3, p. 380.
3. Osler and McGrae: *Princip. and Pract. of Med.*, 9th. Ed., p. 210.
4. Harrison, L. W.: *Dist. of Pract. Med.*, Vol. 3, p. 510.
5. Nicholson, P.: *Sajous's Analyt. of Pract. Med.*, 9th. Rev. Ed., Vol. 2, p. 510.
6. Young, R. A.: *Dist. of Pract. Med.*, Vol. 3, p. 38.
7. Young, R. A.: *Dist. of Pract. Med.*, Vol. 3, p. 38.
8. Osler and McGrae: *Princip. and Pract. of Med.*, 9th. Ed., p. 210.
9. Osler and McGrae: *Princip. and Pract. of Med.*, 9th. Ed., p. 210.
10. Elliott, A. B.: *Jour. Amer. Med. Assoc.*, July 25th, 1907.
11. Nicholson, P.: *Sajous's Analyt. Cyclop. of Pract. Med.*, 9th. Rev. Ed., Vol. 2, p. 511.
12. Young, R. A.: *Dist. of Pract. Med.*, Vol. 3, p. 38.
13. Mackenzie, L. W.: *N. Y. Med. Jour.*, Sept. 24th, 1914.
14. Osler and McGrae: *Princip. and Pract. of Med.*, 9th. Ed., p. 191.
15. Osler and McGrae: *Princip. and Pract. of Med.*, 9th. Ed., p. 302.
16. Nicholson, P.: *Sajous's Analyt. Cyclop. of Pract. Med.*, 9th. Rev. Ed., Vol. 2, p. 512.
17. Osler and McGrae: *Princip. and Pract. of Med.*, 9th. Ed., p. 210.
18. Young, R. A.: *Dist. of Pract. Med.*, Vol. 3, p. 38.

REFERENCES.

1. Barach, J. H.: *Ibid.*, Feb., 1925, p. 151.
2. Philip, R. W.: *Dict. of Pract. Med.*, Vol. 3, p. 380.
3. Osler and McCrae: *Princip. and Pract. of Med.*, 9th. Ed., p. 210.
4. Harrison, L. W.: *Dict. of Pract. Med.*, Vol. 3, p. 310.
5. Nicholson, P.: *Sajous's Analyt. of Pract. Med.*, 9th. Rev. Ed., Vol. 2, p. 510.
6. Young, R. A.: *Dict. of Pract. Med.*, Vol. 3, p. 38.
7. Young, R. A.: *Dict. of Pract. Med.*, Vol. 3, p. 38.
8. Osler and McCrae: *Princip. and Pract. of Med.*, 9th. Ed., p. 209.
9. Osler and McCrae: *Princip. and Pract. of Med.*, 9th. Ed., p. 210.
10. Elliott, A. R.: *Jour. Amer. Med. Assoc.*, July 6th, 1907.
11. Nicholson, P.: *Sajous's Analyt. Cyclop. of Pract. Med.*, 9th. Rev. Ed., Vol. 2, p. 511.
12. Young, R. A.: *Dict. of Pract. Med.*, Vol. 3, p. 39.
13. Mackenzie, L. F.: *N. Y. Med. Jour.*, Sept. 26th, 1914.
14. Osler and McCrae: *Princip. and Pract. of Med.*, 9th. Ed., p. 191.
15. Osler and McCrae: *Princip. and Pract. of Med.*, 9th. Ed., p. 389.
16. Nicholson, P.: *Sajous's Analyt. Cyclop. of Pract. Med.*, 9th. Rev. Ed., Vol. 2, p. 512.
17. Osler and McCrae: *Princip. and Pract. of Med.*, 9th. Ed., p. 210.
18. Young, R. A.: *Dict. of Pract. Med.*, Vol. 3, p. 39.

19. Nicholson, P.: Sajous's Analyt. Cyclop. of Pract. Med., 9th. Rev. Ed., Vol. 2, p. 503.
20. Young, R. A.: Dict. of Pract. Med., Vol. 3, p. 39.
21. Young, R. A.: Dict. of Pract. Med., Vol. 3, p. 38.
22. Pugh, G.: Harveian Soc. Meeting, Reported Lancet, April 3rd, 1926, p. 741.
23. Barcroft, J.: Lancet, March 13th, 1926, p. 544.
24. LoGrasso, H., and Balderry, F. C.: Amer. Rev. of Tub., Oct., 1924, p. 117.
25. Bronfin, I. D.: Colorado Med., Nov. 1924, p. 334.
26. Crocket, J.: Tub. Confer., Reported Lancet, April 3rd, 1926, p. 731.
27. LoGrasso, H., and Balderry, F. C.: Amer. Rev. of Tub., Oct., 1924, p. 117.
28. Johnstone, J. G.: Tub. Confer., Reported Lancet, April 3rd, 1926, p. 731.
29. Pugh, G.: Harveian Soc. Meeting, Reported Lancet, April 3rd, 1926, p. 741.
30. LoGrasso, H., and Balderry, F. C.: Amer. Rev. of Tub., Oct., 1924.
31. Matson, R. W., and Matson, R. C.: Amer. Rev. of Tub., June, 1924.
32. Matson, R. W., Matson, R. C., and Bisailon, M.: Tubercle, Oct., 1925, p. 12.
33. Martinet: Quoted by Sajous, G. E. de M., Sajous's Analyt. Cyclop. of Pract. Med., 9th. Rev. Ed., Vol. 6, p. 11.
34. Jagic: Wein. klin. Woch., Jan., 1925.
35. Pearl, R.: Quoted by Wynn, W. H., Med. Annual, 1926, p. 485.
36. Philip, R. W.: Dict. of Pract. Med., Vol. 3, p. 379.
37. Basutoland Census: May 3rd, 1921.
38. Annual Nosolog. Returns for Qacha's Nek District, 1925: Office of P. M. O., Maseru, Basutoland.

39. Barcroft, J.: Lancet, March 13th, 1926, p. 547.
40. Editor: Lancet, March 20th, 1926, p. 608.
41. Cheinisse, L.: Presse Med., Sept. 20th, 1924.
42. Guy, J. and Page, G. B.: Lancet, April 26th, 1924.
43. Mollgaard, H.: Chemotherapy of Tuberculosis, 1924.
44. Bang, O.: Brit. Med. Jour., 1925, i., 1096.
45. De Witt: Quoted by Wynn, W. H., Med. Annual, 1926, p. 491.
46. Knopf, S. A.: Amer. Rev. of Tub., June, 1924.
47. Danysz: Presse Med., July 19th, 1924.
48. Linnell, J. W.: Tub. Super. Soc. Meeting, Reported Lancet, Oct. 31st, 1925, p. 921.
49. Osler and McCrae: Princip. and Pract. of Med., 9th. Ed., p. 224.
50. Cushny, A.: Text Book of Pharmacology and Therapeutics.
51. Sajous, C. E. de M.: Sajous's Analyt. Cyclop. of Pract. Med., 9th. Rev. Ed., Vol. 6, p. 26.
52. Joseph, E.: Therapie der Gegenwart, June, 1913.
53. Salomon: Mitteil. a. d. Grenz. d. Med. u. Chir., Bd. 27, Nu. 1, 1913.
54. Labbe: Quoted by Sajous, C. E. de M., Sajous's Analyt. Cyclop. of Pract. Med., 9th. Rev. Ed., Vol. 6, p. 13.
55. Lortat-Jacob: Quoted by Sajous, C. E. de M., Sajous's Analyt. Cyclop. of Pract. Med., 9th. Rev. Ed., Vol. 6, p. 11.
56. Thomson, A. and Miles, A.: Manual of Surgery, 6th. Ed., Vol. 1, p. 142.
57. Barbier, H.: Bull. Soc. de ped. de Paris, Feb.-Mar., 1924.
58. Webb, G. B., Gilbert, G. B., and Newman, J. A.: Amer. Rev. of Tub., Feb., 1923.
59. Sajous, C. E. de M.: Sajous's Analyt. Cyclop. of Pract. Med., 9th. Rev. Ed., Vol. 6, p. 26.

60. Rankin, H. P., and Weigel, B. J.: Jour. Amer. Med. Assoc., Feb. 9th, 1924.
61. d'Amico: Lancet, March 29th, 1913.
62. Miller: Quoted in Cunningham's Text Book of Anatomy, 4th. Rev. Ed., p. 1096.
63. Kinnaman: Quoted by Sajous, C. E. de M., Sajous's Analyt. Cyclop. of Pract. Med., 9th. Rev. Ed., Vol. 6, p. 10.