unfair interpretation of symptoms which are difficult to understand, and we might consider the possibility of deranged function at a lower level—a sympathetic level—being responsible for a threshold of pain so depressed as to make many stimuli, innocuous under healthy conditions, assume noxious habits and qualities.

There is some support for such a view to be obtained on the clinical side. The patient who develops chronic abdominal pain after an abdominal operation, and who is prepared to undergo a relay of further operations for its relief, is often not of the neurotic type in the first instance. She does not respond to, or rather does not recover under, psychotherapy. Time, uninterrupted by much medical or any surgical treatment, may bring about complete recovery, unless fear and impatience combine to produce a psychoneurosis. After all, we are familiar with localized areas of hyperaesthesia in the distribution of the peripheral nerves after injury or sepsis, areas which may be exquisitely sensitive to all stimulation for months and years, and which may yet gradually approach a more normal condition if left well alone.

On the other hand, painful intestinal spasm of a paroxysmal type is a somewhat rare but very definite symptom in anxiety states, and may give rise to much difficulty in diagnosis, as well as to needless laparotomy. Such instances as I have met with have occurred in profoundly neuropathic patients, and have only yielded to psychotherapeutic measures. They are interesting from a physiological standpoint because they are examples of severe colic without any discernible local lesion. The intense pain and resulting tenderness, sometimes accompanied by a palpable knuckle of contracted gut resembling what can be felt in a cramp of a skeletal muscle, strongly suggest that splanchnic pain is chiefly due to muscular spasm, and may be independent of any morbid process either in the intestinal wall or in the bowel contents. Whether the pain is the result of pressure exerted on the intrinsic nervous structures, or due to interference with their blood supply, is a question which has yet to be answered. The early intermittent colicky pains associated with mesenteric thrombosis and embolism bring the circulatory condition into prominence as a causative factor.

There is perhaps no more important and difficult problem confronting a surgeon than the interpretation of pain in acute abdominal conditions; but, having referred to some of the features which characterize the splanchnic and peritoneal types, I shall leave their further elaboration to those who are much better qualified to deal with them than I am. In particular, we may hope to hear Mr. Morley giving us the result of his carefully considered research into the nature and meaning of the pain associated with appendicitis. Meanwhile, may I recall to your memory some of the heteronomous types of abdominal pain which are liable to entrap surgeon and physician alike?

First of all, there is the immobile painful abdomen met with in acute pulmonary disease—in pneumonia and infarct of the lower lobes, for instance. This is sufficiently common, and has been so long recognized that it deserves no more than a passing reference. Less common is the acute epigastric pain associated with vomiting and profound collapse observed in cases of coronary thrombosis, and generally found in persons suffering from arterial degeneration. It may simulate the picture of a perforated gastric ulcer so faithfully as to be a real diagnostic pitfall. Still more rare are the attacks of severe abdominal pain, colicky in type, which result from atheroma of the abdominal aorta and which have earned the title of "angina abdominalis." They resemble their cardiac namesakes in being paroxysmal, often at long intervals, and do not present associated physical signs such as should justify an exploratory laparotomy.

The gastric crises of tabes have an unpleasant notoriety for creating an atmosphere of diagnostic doubt, and the problem is generally dismissed in textbooks with the statement that the presence or absence of knee-jerks will suffice for its solution. As a matter of fact, the presence or absence of knee-jerks may guide us to the presence or absence of the tabetic process at the lumbar level of the cord, but has no bearing on the presence or absence of a lesion of the upper dorsal roots. On the other hand, the

pains of tabes, although pathogenetically obscure, are clinically characteristic, and those which infest the epigastrium present much the same features as those which inflict torture on the patients' limbs. They are lancinating, rapidly repeated, and chronologically paroxysmal. They are associated with intense superficial hyperaesthesia, but with no such muscular immobility or rigidity as is met with in peritonitis. The accompanying nausea and vomiting may be regarded as secondary and reflex in origin. In fact, careful attention to descriptive details and to the abdominal physical signs should serve to distinguish gastric crises from any morbid process originating in the abdominal organs without the necessity of worrying the patellar tendons.

Those who follow me will discuss the views held and supported by various authorities during the last forty or fifty years in regard to the origin and nature of abdominal pain. Since Ross advocated the distinction between splanchnic and somatic pain there have been many workers in this field of research. Mackenzie, Head, Hurst, Ryle, Payne and Poulton, Maylard and Bolton have all contributed much of value towards the solution of a very important and difficult problem from the clinical standpoint, and we may hope that its ventilation in a physiological atmosphere to-day may lead to its further elucidation.

Whether or not this hope is fulfilled, the discussion may be welcomed as evidence that the profession is taking an increasing interest in the meaning of pain, is more inclined to subject pain to careful analysis, and is more appreciative of the position of pain in the proper execution of its daily work. We may look forward with confidence to a time when the loose employment of such terms as rheumatic, neuritic, neuralgic, etc., for descriptive purposes is abandoned, and when due regard is paid, not only to the site and quantity, but also to the precise quality of pain as an essential factor in the diagnosis of disease.

As Hilton wrote sixty-five years ago: "Every pain has its distinct and pregnant signification, if we will but carefully search for it."

CONDITIONS SIMULATING PULMONARY TUBERCULOSIS;

WITH SPECIAL REFERENCE TO PNEUMONOCOCCUS
AND BRONCHIECTASIS.*

BY

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THE substance of what I am about to say might be crystallized in one sentence: It is by bacteriological examinations of the sputum and faeces, and by these examinations almost alone, that we can establish a diagnosis of pulmonary tuberculosis or exclude it. I know that there are exceptions, such as the very early, sometimes the milinary and the latent case, and even occasionally, but very rarely, in other instances, but in the vast majority of cases the dogma will be found true. It is because of the neglect of this simple test that a great many cases of pulmonary tuberculosis escape recognition in the early stages. It is because of this neglect that many non-tuberculous conditions are diagnosed as tuberculous, and their true nature is unrecognized. If tubercle bacilli are not found after repeated examinations of the sputum, and a few examinations of the faeces, and there are definite signs and symptoms, it is certainly the time to begin to consider that the condition may be a non-tuberculous one.

Some may ask why I insist so strongly on the examination of the faeces, and an explanation still appears to be necessary. In many cases of active pulmonary tuberculosis either no expectoration is produced or what is produced is merely mucus or muco-pus from the nasopharynx. This is specially apt to occur in children and

* A paper read in opening a discussion in the Section of Tuberculosis at the Annual Meeting of the British Medical Association, Manchester, 1929.

women. All the pulmonary secretion is swallowed. Millions of tubercle bacilli may be swallowed in this way, and may easily be found in the stools by the antiformin method of examination. Such bacilli, when tuberculous food or milk is excluded, imply pulmonary tuberculosis, for a primary tuberculous enteritis is a pathological curiosity, and there is no other origin of the bacilli. The existence of these bacilli has been known for some time, but the test was not a routine one in most clinics. In 1924 I made, with my house-physician Dr. Stobo, an investigation into the ques-syphilitic infection, and the presence of tubercle bacilli, was of real practical clinical value. We found it of the greatest value, and since then we have made it a routine test at Victoria Park, when the sputum fails us. The controls had no tubercle bacilli in the faeces. Subsequent experience has fully confirmed the importance of this examination.

There are a great many conditions which may simulate pulmonary tuberculosis, sometimes only when they are fairly advanced as with pneumoconiosis, sometimes only at the beginning when symptoms may be similar or identical—for instance, cough, sputum, haemorrhage—later they begin to diverge and the picture in each case becomes distinctive—as, for example, bronchiectasis. Occasionally tuberculosis may coexist with one of the diseases by which it is simulated, which may cause great difficulty for a time. The knowledge that such conditions may be present concurrently will help us to arrive at the correct dual diagnosis. I will enumerate the more common of these apparently fortuitously associated affections. I say apparently fortuitous, for so it seems to be, except in the case of certain forms of pneumoconiosis, where silica increases the liability to tuberculous infection.

1. Pneumoconiosis and pulmonary tuberculosis.
2. Lymphadenoma and pulmonary tuberculosis.
3. Syphilis and pulmonary tuberculosis.
4. New growth and pulmonary tuberculosis.
5. Bronchiectasis and pulmonary tuberculosis.
6. Mitral stenosis and pulmonary tuberculosis.

The following points will help in the diagnosis. In each case it is assumed that tubercle bacilli have been found.

1. *Pneumoconiosis and Pulmonary Tuberculosis.*—Here the history of the patient's employment would help. The other distinctive points will be discussed later.

2. *Lymphadenoma and Pulmonary Tuberculosis.*—This may cause great difficulty, but a gland may be available for examination, though this unfortunately is not always histologically distinctive. Curious intermittent exacerbations of temperature may suggest lymphadenoma. The x-ray picture may show enlarged glands or masses in the mediastinum. If there is a large pleural effusion obscuring the x-ray picture, replacement of the fluid by oxygen or air and subsequent radiography may reveal the presence in the mediastinum or lung of large masses characteristic of lymphadenoma or growth, and unlike tuberculosis. The response to arsenic and x-ray treatment may help.

3. *Syphilis and Pulmonary Tuberculosis.*—Here a strongly positive Wassermann reaction or a history of previous syphilitic infection, and the presence of tubercle bacilli, will help to establish the dual diagnosis.

4. *New Growth and Pulmonary Tuberculosis.*—This combination may cause great difficulty, but the deterioration of the patient's condition, more rapid than one would expect with the amount of disease present if it were tuberculous, and the presence of radiographic shadows unlike those produced by tubercle, will give rise to a suspicion, which will be confirmed as the case progresses. Sometimes, however, a very dense fibrosis may simulate neoplasm, both in cases of tuberculosis and pneumoconiosis, but the progress of the case usually gives a clue to the true nature of the affection. We have had many cases at Victoria Park Chest Hospital where tuberculosis and primary malignant growth have coexisted in the lung. In certain cases the x-ray picture, in the early stages, is by no means pathognomonic. In such a case under my care certain symptoms were suggestive of either tuberculosis or malignant growth, and at first the diagnosis was not easy. But the absence of tubercle bacilli, the steady deterioration of the patient, and yet so small an area of disease aroused a suspicion of malignant disease,

which was confirmed later. If tubercle bacilli had been present at that stage a correct diagnosis would have been problematical. I have known a tuberculous mass to be removed from the lung in the belief that it was a growth. On the contrary, growths in an apparently operable stage may be left, because they are thought to be inflammatory. When the diseases coexist the mistake does not matter so much, as drastic treatment could scarcely be undertaken. But with malignant disease by itself a correct and early diagnosis is important if we hope to employ effective treatment. Although some cases may present great difficulty, the absence of tubercle bacilli, the symptoms, and the x-ray picture will usually provide sufficient evidence on which to diagnose malignant neoplasm of the lung. Paralysis of one of the phrenic nerves or of one of the recurrent laryngeal nerves is strong evidence in favour of growth and against tuberculosis.

5. *Bronchiectasis and Pulmonary Tuberculosis.*—The diagnosis of bronchiectasis will be discussed in more detail later. With modern methods of diagnosis the coexistence of these two conditions can be demonstrated almost with certainty.

6. *Mitral Stenosis and Pulmonary Tuberculosis.*—The haemorrhages which may occur in mitral stenosis may simulate tuberculosis, but the characteristic signs of a mitral lesion with tubercle bacilli in the sputum usually make the diagnosis easy. The physical signs may be of no help. The x-ray picture may be characteristic of tuberculosis, but the presence of infarction, and the denser striations of pulmonary engorgement, may make the picture equivocal, and therefore reliance must not be placed on the x-ray findings alone.

The conditions which may simulate pulmonary tuberculosis may resemble it because of symptoms such as continuous fever, or wasting, or haemorrhage, or because of signs such as those of fibrosis, or because of the appearance of a serous pleural effusion. It would be far too long a task to discuss in detail the differential diagnosis of every one. I must content myself, therefore, with enumerating the various diseases, giving here and there an impressionist hint at the most important points in their differentiation, and dealing in fuller detail with those with which we are most likely to be confronted, with especial reference to pneumoconiosis and bronchiectasis.

Serous Effusion.—The causes of a serous effusion, other than tuberculosis, are lymphadenoma, malignant growth (secondary or primary), dermoid, cardiac, or renal disease, subphrenic abscess, polyserositis.

Lymphadenoma I shall refer to again later. A *malignant serous effusion* may be diagnosed by replacement and thoracoscopy in cases where nothing else will help. Occasionally the x-ray picture is distinctive after replacement. In spite of what is often written, the effusion is seldom bloody, and the cytology of the fluid seldom of help. A *dermoid* is usually obvious in the skiagram. With an effusion secondary to *cardiac* or *renal disease* careful examination will, as a rule, show the underlying cause. Sometimes, however, a recurrent unilateral effusion may be cardiac in origin, and it may be difficult to say whether it is tuberculous, malignant, cardiac, or due to polyserositis. I have seen a case of serous effusion above a subphrenic abscess called tuberculous. But there was no excuse for the mistake, for there was a high leucocytosis and daily rigors, and the latter never occur in tuberculosis.

In *polyserositis* there may be associated changes, mediastino-pericarditis, ascites, perihepatitis, and perisplenitis, with enlargement of the liver and spleen. The effusion, however, may be an isolated phenomenon, and the diagnosis will only be made after many aspirations, the fluid tending ever to recur while the patient's general condition deteriorates surprisingly little.

Many cases of long-continued fever without physical signs may arouse a suspicion of tuberculosis. If the patient is seriously ill miliary tuberculosis may be suspected. Repeated x-ray examinations of the chest may reveal quite definitely the evolution of miliary tuberculosis in the lungs. This may be illustrated by three slides. The first shows widely distributed small opaque dots. The second shows an increase in size of these shadows. The third shows a spontaneous pneumothorax to have occurred, while the

lungs are becoming more and more grossly involved. The diagnosis was confirmed at post-mortem examination.

In every case of obscure fever it is important first to exclude a *Bacillus coli* infection of the urinary tract and those diseases that may be diagnosed by blood examinations and agglutination reactions—as, for example, the typhoids, malaria, and even rat-bite fever, Malta fever, and *Bacillus abortus* fever. With the last-mentioned rigors are a feature, and these, as I have already remarked, never occur in tuberculosis.

Subacute bacterial endocarditis may be extremely difficult to diagnose. Blood cultures, which should always be performed, are frequently disappointing. The most important diagnostic point, in my opinion, is the occurrence of embolic phenomena in the skin, the kidneys, the spleen, and elsewhere.

Cryptic collections of pus, as in a subphrenic or perinephritic abscess, will almost invariably give a high leucocytosis, but rare cases of pyonephrosis and carbuncle of the kidney with a blocked ureter may fail to give rise to this leucocytic response and may yield few or no urinary signs, thus making the diagnosis more difficult. Rigors, however, should at once make the diagnosis of pus, somewhere, almost certain. With generalized streptococcal and staphylococcal infections great difficulty may be experienced. Blood cultures and urine cultures may help, but I would utter a warning against accepting, as proof positive, the finding of streptococci and staphylococci in the urine by culture only.

Hyperthyroidism of the type that begins with great wasting and tachycardia, with no other signs or symptoms, may suggest tuberculosis; but the absence of fever, and of tubercle bacilli and physical signs, would almost exclude tuberculosis, for such severe and dramatic symptoms could hardly be due to incipient phthisis. The determination of the basal metabolism would probably suggest hyperthyroidism.

Chronic conditions which may simulate pulmonary tuberculosis owing to signs and symptoms may be tabulated as follows:

1. Unresolved pneumonia, or broncho-pneumonia.	9. Fungus Infections:
2. Obscure empyema or simple unrecognized empyema.	Actinomycosis.
3. Non-tuberculous fibrosis.	Aspergillosis.
4. Apical catarrh.	Blastomycosis.
5. Pneumonocystosis.	10. Spirillitis (or broncho-pulmonary spirochaetosis).
6. Bronchiectasis.	11. Pulmonary distomiasis.
7. Lung abscess.	12. Hydatid.
8. Neoplasm (malignant or innocent).	13. Mitral stenosis.
	14. Lymphadenoma.
	15. Syphilis.
	16. Foreign body in bronchus.
	17. Bronchial stenosis.

Of these the spirillum, fungus, and fluke infections will be diagnosed by bacteriological examination. Before accepting a spirillum or spirochaete as the cause of the symptoms the following conditions must be satisfied. The spirillum must be constantly present, present in large numbers, and the sputum must contain the organism in numbers far in excess of those found in the saliva. Such a differential examination should therefore always be made. A massive basal tuberculosis may simulate unresolved pneumonia, especially when the condition has apparently followed an acute influenzal attack. The condition of the patient, the absence of leucocytosis, the presence of tubercle bacilli, are the diagnostic points.

Certain cases of *obscure empyema* may cause difficulty, but the history, the leucocyte count, the x-ray examination, skilful exploration, and even exploratory thoracotomy, will usually give the clue. From time to time cases of chronic empyema that have been treated as tuberculosis are admitted to hospital. This mistake is usually unardonable, and is mostly due to ignoring the history of onset.

The only method that I know of distinguishing a *non-tuberculous* from a *tuberculous fibrosis* is by the presence or absence of the tubercle bacillus. If tubercle bacilli are present it is a tuberculous fibrosis, if absent it is a non-tuberculous fibrosis, except of course in completely healed extensive tuberculosis or after the cessation of artificial pneumothorax treatment, when tubercle bacilli had previously been found.

Apical Catarrh.

There is no doubt, I think, that after certain catarrhal conditions of the lung, especially after influenza, constant crepitations may be heard at the apices which may persist for some time. They naturally suggest the possibility of tuberculosis. I have investigated many such cases, and have come to the conclusion that a simple catarrh, possibly associated with adhesions, explained the signs. The only way to be certain is to follow the usual routine of evening temperature record, weight record, sputum examinations, and possibly an x-ray examination. In some epidemics, however, this phenomenon will be found so frequently that we are probably compelled to use our judgement as to which cases require the complete investigation.

Pneumonocystosis.

This condition may arise from many occupations. Thus we get anthracosis, silicosis, or chalicosis, siderosis, asbestosis, byssinosis (workers in cotton, felt, and shoddy mills). In most cases, however, the trouble is due to silica.

Symptoms.—Of 36 cases of silicosis analysed by Drs. Sutherland and Bryson,¹ seventeen had no symptoms. In other cases the symptoms were almost entirely shortness of breath, cough, and expectoration. There was no instance of haemoptysis. Wasting was not usual. In some of the cases of asbestosis that we have investigated at Victoria Park Hospital, however, there has been extreme wasting and easily produced cyanosis on exertion, or on contracting a cold, etc.; but perhaps the most remarkable symptom is the dyspnoea. Many cases of asbestosis have been collected recently by Dr. W. Burton Wood, who has made a valuable contribution to the subject in the May issue of *Tubercle*.

The diagnostic points are the history, the symptoms, the physical signs, the absence of tubercle bacilli, the x-ray picture, and, in some cases of asbestosis, the finding of asbestos fibres in the sputum, described by Dr. S. Roodhouse Gloyne in the June issue of *Tubercle*, or, as pointed out by Dr. M. J. Stewart, the discovery of yellow club-shaped "curious bodies" which had previously been demonstrated in lung sections. Lung puncture has also been tried, but I personally do not consider this justifiable.

The physical signs may be simply those of a delicate fibrosis or perhaps emphysema. Later there may be contraction of the chest wall and dullness to percussion. Gross fibrosis may occur. The adventitious sounds are like those of any other type of fibrosis.

The x-ray picture differs according to the stage of the disease and the type of dust—for example, gold miners' lungs, potters' lungs, and asbestos lungs may all give a different appearance. Help has been claimed from the localization of the abnormal shadows, but these cannot be regarded as pathognomonic; the appearance may simulate tuberculosis, any sort of fibrosis, or even new growth of the lung. On the other hand, we occasionally get skiagrams which reveal an unexpected widespread fibrosis throughout the lungs in cases in which there is no history of exposure to dust.

Where the condition is established, however, there will be little difficulty in diagnosing the condition from the x-ray picture, the history of the case, and the absence of tubercle bacilli. The distribution of the fibrosis is suggestive—for example, its rough symmetry, its selection of the mid zone. In the early stages diagnosis may be most difficult, and it is only the history of the patient's occupation, the dyspnoea, and perhaps the x-ray picture, on which a diagnosis may be based.

Bronchiectasis.

A few years ago the differential diagnosis between bronchiectasis, pulmonary tuberculosis, and other diseases was by no means easy. In the advanced stage, of course, there was little difficulty, though even here serious mistakes could be made—for example, abscess, chronic empyema, or foreign body in the bronchus might be called bronchiectasis—but in the early stages of the evolution of the

¹ Report on the Occurrence of Silicosis among Sandstone Workers. H.M. Stationery Office.

disease, stages which can extend over very many years, a diagnosis was often difficult or impossible. At such a time I should have been compelled to talk at length about symptoms and signs, and at the end have left matters as they were—uncertain. We might hazard a shrewd guess, but it would have been an opinion, not an established fact. Now, owing to the introduction of iodized oil by Sicard and Forestier, we can claim to prove or exclude bronchiectasis and to localize it. The symptoms at first may be identical with those of tuberculosis—cough, sputum, haemorrhage; the physical signs do not really help, for tuberculosis may be basal, and bronchiectasis apical. The x-ray picture of both diseases may be similar, or in both cases may show nothing distinctive. If, therefore, with such symptoms there is an absence of tubercle bacilli in the sputum and faeces, a non-tuberculous condition should be thought of, iodized oil should be given, and the diagnosis established. I must confess, however, that in one or two cases I have been a little puzzled to determine whether the cavities revealed were tuberculous or bronchiectatic. In the example shown, however, I think it will be agreed that the appearance is that of lung cavitation rather than dilatation of bronchi.

In chronic *lung abscess* there are no tubercle bacilli. The x-ray appearance is suggestive. Lipiodol, as a rule, does not enter the affected area, thus differentiating it from bronchiectasis.

Malignant growth has already been discussed. *Innocent tumours*, as a rule, have a distinctive appearance on the skiagram. One great pitfall is to mistake lymphadenoma for an innocent tumour, but to discuss this would be to digress too far beyond the limits of my subject.

SOME PHARMACOLOGICAL ASPECTS OF AVERTIN.

BY

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ANY recent advances which have been made in anaesthesia have been rather those of improved methods of administration and technique than the introduction of new anaesthetics. This is due partly to the fact that anaesthetics have come to be regarded as substances to be administered by inhalation, a method which has attained popularity because it permits accurate control and regulation of the duration and depth of anaesthesia; and partly to the reliability of three volatile drugs—ether, chloroform, and nitrous oxide—introduced in the very earliest days.

The rectal administration of anaesthetics does not permit of such fine control of anaesthesia as can be obtained by inhalation. Gwathmey⁹ has shown that anaesthesia results when ether is absorbed at a constant rate from a solution in olive oil given by the rectum, and that the drug is excreted by the lungs. Obviously such considerations do not apply to non-volatile anaesthetics. Rectal anaesthesia has certain practical advantages: it is more comfortable for the patient, it does not necessarily require the presence of an anaesthetist, for the surgeon himself can administer the anaesthetic, and it is more convenient for operations about the head and neck. The main disadvantages are the risk of irritation to the intestinal mucosa and the difficulty of maintaining a constant concentration of the anaesthetic in the tissues. This second problem is intimately connected with the absorption and excretion of the drug in question, for the administration of a rectal anaesthetic is not continuous, and, once given, it is impossible to control its action further.

Some three years ago it was discovered that tribromomethylalcohol, a simple organic compound of bromine, possessed the property of producing anaesthesia. Moreover, it has been found¹⁴ that the therapeutic margin of safety is greatest with this substance when it is administered by the rectum. A quantity of experimental and clinical data has been accumulated (1-7 10 12 13 14 17-23, and others) regarding

Lymphadenoma may simulate pulmonary tuberculosis in many ways—by long-continued fever, by lung symptoms and signs, by the production of a pleural effusion. It is one of the most difficult of all causes of obscure fever, but the pattern of the temperature chart, the curious intermittent regular bouts of fever, will frequently help. If primary in the chest, the bulk of the disease is probably in the mediastinum, and will so appear in the skiagram. If an effusion is present and there is doubt of the diagnosis, replacement by gas and subsequent radiography will probably reveal masses in the mediastinum, perhaps extending into the lung. The response to treatment by arsenic and x rays may be a further help in diagnosis.

Syphilis.—Although many different syphilitic affections of the lung are described, such as massive consolidation, chronic bronchitis, bronchiectasis, etc., the only certain syphilitic conditions that I have seen are an apparent *gumma* in the lung, a gummatous mass in the mediastinum causing venous obstruction, bronchial stenosis, and the changes following pressure by an aneurysm. The history, the Wassermann reaction, the x-ray picture, and the response to treatment may give the diagnosis.

With *foreign body* in the bronchus, the history and bronchoscopic examinations are the most important points. In *bronchial stenosis* lipiodol injections and bronchoscopic examinations are essential.

To sum up, in difficult cases the routine to pursue will be as follows: A careful history, an exhaustive bacteriological investigation, lipiodol injections, x-ray examinations, and bronchoscopic examinations. In certain cases such procedures as replacement and thoracoscopy will be needed, in others even an exploratory thoracotomy. Of all these I would put first the bacteriological investigation.

this substance, which is now issued under the trade name of "avertin." The opinion has been formed that the drug represents a definite advance towards the ideal anaesthetic, largely because of the simplicity of its administration. All that is necessary is to give a definite proportion of the drug per unit of body weight in aqueous solution by the rectum. Administration is not continuous, for, once the requisite quantity of the drug has been injected, the patient becomes anaesthetized and remains so for a period depending upon the amount of avertin given. The drug is absorbed with extreme rapidity,²¹ so that, when the operation is over, it is useless to attempt to remove any which may remain in the rectum, as is the case with other rectal anaesthetics.

The pharmacology of avertin has been studied mainly in Germany, and no experimental investigations have been recorded in this country as yet. The present paper includes the main results of an investigation on these lines to confirm and extend previous observations.

Some Physical and Chemical Considerations.

Avertin is an odourless, white, crystalline powder. It has a melting point of 80° C., and is soluble in water to the extent of 3 per cent. at 37° C. Solution occurs very slowly; it takes upwards of forty-five minutes, aided by vigorous shaking, to produce a clear solution. As the solution must be freshly prepared for each anaesthesia this is a material disadvantage, and the makers have to some extent solved the difficulty by issuing the substance dissolved in amylene hydrate, itself a rectal anaesthetic, in the proportion of 1 gram of avertin in 1 c.cm. of the solvent. This solution is readily soluble in water, and avertin can thus be prepared conveniently for rectal injection.

The chloroform/water distribution coefficient of avertin is comparable with that of ether, and indicates that the two drugs have a similar anaesthetizing power. Avertin contains a high proportion, approximately five-sixths, of bromine. In aqueous solutions it is decomposed by heating above 50° C. into hydrobromic acid and dibrom-acetaldehyde:

Many of the earlier difficulties in the use of avertin by rectal administration were due to the presence of these decomposition products. It has been found that they are