CHEST EXAMINATION

TRAIL

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THE CORRELATION OF PHYSICAL AND X-RAY FINDINGS IN DISEASES OF THE LUNG

By

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CHEST EXAMINATION

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AND

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FOREWORD

WHILE still a medical student I felt strongly that the next generation would condemn the practice of herding cases of proved pulmonary tuberculosis in out-patient departments. There they would sit in rows among the other patients, coughing over them, ultimately to be rewarded by a bottle of cod liver oil and some linctus. But we did have the opportunity of learning our physical signs from these unfortunates. To-day I can affirm from my experience as an examiner at various universities that the weakest part of the candidates' equipment is their recognition and interpretation of physical signs in the lungs. To some extent this may be due to the segregation of tuberculous cases into departments where students do not freely enter, but still more to their tendency to short-circuit their examination by flying to the X-ray film, using it as a substitute instead of a confirmation of ordinary clinical methods. The teachers, however, are not entirely free from blame, particularly in the matter of multiplying labels for the various sounds heard through the stethoscope; the degree of accuracy thereby implied is largely illusory, and merely confuses the student. It is one of the many merits of Wing Commander Trail's book that he follows the tradition of my own teachers, Dr. Gee and Dr. Samuel West, by using as few terms as possible, and correlating each with its pathological significance.

This plan is characteristic of the whole work; anatomy, physiology, pathology, symptoms, physical signs and X-ray findings are closely correlated, enabling the student or practitioner to build up a mental concept of exactly what is happening inside the chest. Many will be interested to learn how greatly modern X-ray technique has advanced interpretation in skilled hands. Even more will be surprised to find what helpful deductions from physiological principles they habitually ignore. And all will be relieved to find how a grasp of the first principles involved will enable them to arrive at a correct opinion without imposing burdensome details on their memory.

As a former teacher I know how valuable a contribution this book makes to the understanding of vital problems, while the freshness of its approach will be patent to all.

W. LANGDON-BROWN.

PREFACE TO THE SECOND EDITION

THE Second Edition has been slightly enlarged, particularly by the addition of illustrative X-rays, set in the text so that they are easy of reference. The order of Contents has been changed, and notes added on such points as the interpretation of breath sounds and the " normal abnormalities " of the bony thorax.

These alterations are due to the helpful criticism of notices in the Medical Journals, and in particular to the kindness of many readers who wrote personally to the author. Among the latter I wish to thank especially Dr. J. D. Grove-White, Dr. R. L. Midgley, and Dr. H. Courtney Gage, who sent their detailed observations as a general practitioner, a chest physician, and a radiologist respectively.

R. R. TRAIL.

PREFACE TO THE FIRST EDITION

THIS book is founded on courses of lectures which it has been the writer's good fortune to give to students and post-graduates. They owe their present form in great part to attempts made to answer the difficulties of those students who showed an interest in an approach to examination of the chest founded on a knowledge of applied anatomy, physiology and pathology, without which it is naturally useless to attempt a reasonable interpretation and correlation of physical and X-ray findings.

Correlation is not so difficult to acquire as many students seem to fear. Much confusion has arisen of late years because of the big advances made by chest physician specialists on the one hand and chest radiology specialists on the other. More recently, however, there has been a tendency to combine these branches in the " chest physician cum radiologist," or the chest physician who likes to read his own X-rays. This is all to the good of the modern student ; it tends to bring us back to fundamentals. We find it increasingly easy to go beyond the objective reading of films. We can connect physical signs with definite

abnormalities of shadow, and combine both with the changes in normal anatomy and physiology so ably expressed nowadays by the specialised lung pathologist, who is, we must remember, but the first offshoot of that type of learned physician who laid his firm foundations for practice in his earlier post-mortem room researches, when X-rays did not exist.

The first Section of the book is, therefore, devoted to reminders on those salient points of normal anatomy and physiology which explain the abnormalities of the commoner chest diseases of general practice. Like other points of equal importance, they are repeated by reference to other sections.

In the second Section on Applied Pathology we consider the main gross and microscopical changes induced by these diseases, and, in noting the physical signs and the alterations from the normal postero-anterior film that accompany them in their various stages, an attempt is made to correlate all three aspects. A certain amount of detailed description is necessary, but this is confined as much as possible to fundamentals, even if by this statement we appear to be begging the question.

There has been of late years much discussion on the reading of abnormal films, and of necessity so much individual variation in reports, that an attempt has been made in some quarters to confine them to objective wording. As already indicated, the writer feels that we can go further ; that we should attempt to arrive at criteria for interpretation, using the work of the physician, the pathologist and the radiologist as a combined whole. We shall all agree that changes of microscopical detail cannot be expected to reflect themselves in stethoscopic signs and on the usual postero-anterior film ; we know that a lobule on the lung surface is no more than about one-quarter of an inch in its longest diameter. Nevertheless, it is felt that a knowledge of pathogenesis is fundamental for the student who would correlate his physical and X-ray findings, even if these are demonstrable only when comparatively gross areas of lung are involved. Thus we know that in phthisis stethoscopic signs are late, but we can find a reason why they are late and so why we should look for other and as important physical signs, such as lack of movement and note, that will precede these stethoscopic signs, and so warn us of the presence of a specific pathological change. It may be argued that the prime correlations here detailed are built on *a priori* reasoning ; even if this be justifiable criticism it will be

admitted that they can give some elementals that may combine the still too-well defined compartments of the pathologist, the radiologist and the physician. Even a mere workable explanation would be better than no basis at all.

Section III sums up the physical findings in the diseases discussed in Section II, and gives a scheme of interpretation of stethoscopic findings that may act as a basis for diagnosis without X-ray findings. No attempt is made to deal with all normal and abnormal stethoscopic signs, but only with such adventitious sounds as can be correlated with underlying pathogenesis. It is taken for granted that the student has listened to large numbers of normal chests and a sufficient number of abnormals to understand the principles underlying the interpretation of breath sounds and voice sounds; *e.g.*, the difference between the prolonged expiration of emphysema and the high pitch, faint or loud to the ear, that connotes bronchial breathing.

It will be seen that all these sections are inter-connected. They are in essence the parts of an interesting jig-saw puzzle. True they place personal values on the individual pieces used in the game, but they are the result of the writer's conscious attempt over several years to increase the value of each piece by using experience to alter its shape so that it fits more easily with its fellow. Each new problem in living pathogenesis continues to be instructive, while it whets the appetite for still further problems.

The ultimate aim of this handbook is to bring added help to the student undergraduate and post-graduate, in the assessment of the living and changing pathogenesis of his individual patient; only by so doing can we arrive at a reasoned basis for that wider problem of individual treatment, which must be the object, and is always the only true reward, of the happy physician.

My thanks are due to Sir Walter Langdon-Brown for his kindness in writing a Foreword, to F/Lt. J. A. Kennedy for his assistance in reading proofs and in preparing the index, and to Mr. J. Rivers of Messrs. J. & A. Churchill Ltd. for his valuable help in these days of difficulty with paper and print.

R. R. TRAIL.

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CHEST EXAMINATION

SECTION I. APPLIED ANATOMY

CHAPTER I

THE BRONCHIAL TREE

THE trachea lies behind the great vessels, embedded in the elastic areolar tissue which ensheathes all the structures lying in the mediastinum. It divides at about the level of the fifth dorsal vertebra into its two main branches, the right and left bronchi. To this point it can be seen on the normal postero-anterior film of the chest as a clear area, bearing slightly to the right of the mid-line of the thorax.

The point of division is important in all conditions which cause displacement of the mediastinum, in that it lies not far below the third dorsal vertebra, opposite which is the weakest part of the mediastinum. On this point the mediastinum swings laterally, as on a hinge. Any pull on the lung structures connected with one main bronchus is easily transmitted along the lower part of the trachea to this weak point. We shall see later that the trachea responds more than any other mediastinal structure to lung changes, and that therefore if we find evidence of its disturbance we can reason backwards to find the cause in such lung changes. Fortunately its movement is quickly reflected on the sternomastoid muscle so that we have an easily demonstrable sign of its displacement.

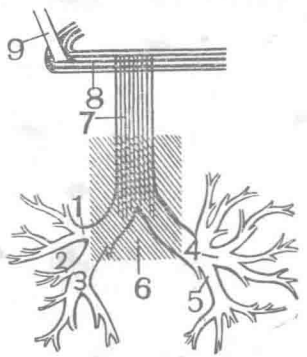

FIG. 1. The Mechanism of the Sternomastoid Sign.
1. The right upper lobe bronchus.
2. The right middle lobe bronchus.
3. The right lower lobe bronchus.
4. The left upper lobe bronchus.
5. The left lower lobe bronchus.
6. The areolar tissue of the mediastinum.
7. The pretracheal fascia.
8. The fascia of the neck.
9. The sternomastoid muscle.

The anatomical relations of the muscle explain this reaction (see Fig. 1). On the anterior surface of the trachea lies a band of tissue called the pretracheal fascia, connected below with the elastic areolar tissue of the mediastinum, and above

with the deep fascia of the neck. The latter fascia, which meets from both sides in the middle line, divides as it goes backward, to enclose the sterno-mastoid muscle, and is thus in close contact with its tendinous part which has its origin at the anterior superior border of the manubrium sterni. We see therefore how it is that tracheal displacement is reflected to an increased tension of the sternomastoid muscle in this tendinous part, on the same side as that to which the mediastinum is pulled or pushed, by traction on one side or pressure from the other.

As we proceed with our studies we shall find that a pull on the trachea is effected by all processes which interfere with the elastic tissue of the lung. This elastic tissue is continuous throughout the bronchial tree, and carried from its smallest division to the elastic surround of the air cells; indeed, with the finest, ultimate arterioles, it forms the actual alveolar wall.

All branches of the bronchial tree are also intimately surrounded by a binding of connective tissue, which acts as a supporting structure like a scaffolding. It is resilient, and moves in response to movements of the bronchi, as they alter in length and diameter by inspiration and expiration; but it is not an integral part of the bronchus like the elastic tissue, and so its pathological changes have not the same opportunity to reflect themselves on the mediastinum. Its changes appear to act only secondarily. Its commonest pathological change is in the deposition of fibroblasts, which produce peribronchial fibrosis. Increase in depth of tissue leads only to ultimate shrinkage and loss of resilience; what seems to happen is that the fibrosis obliterates lobules it was meant to support, and causes such a drag on others in its neighbourhood that it destroys their elastic tissue after distending them.

Direct and indirect destruction of elastic tissue occurs quite early in adult pulmonary tuberculosis. Material from diseased lobules enters, and then blocks, the supplying terminal bronchiole. As air cannot now reach the alveoli, empty ones collapse, and full ones organise, and both lose their elasticity. The trachea is thus pulled to the diseased side, and we find a sharp inner border to the sternomastoid muscle on the same side.

The effect of the mediastinal hinge on tracheal displacement is well shown in disease confined to the upper lobes. It is not unusual to find that the part above the third dorsal vertebra is dragged into a definite bow by localised infraclavicular tuberculosis. In the same way, a kink in this region is seen in several

cases of substernal thyroid, and in adenoma of one lobe of the gland, which pushes the trachea out of its central position.

Along with other mediastinal structures, the trachea is displaced in the common abnormality of dorsal scoliosis, which is usually convex to the right. This is really a torsion of the thoracic cage, pushing the right lower half forwards and outwards, and pulling the left lower half inwards and backwards, as viewed anteriorly. When we look at the patient from the front we see his right lower ribs forced apart to produce bulging, while the left lower half appears to be fallen in; exactly the opposite is found on looking at the back; the right lower zone is flatter, and the left lower zone more prominent than the normal. In other words, the volume of the chest cavity as a whole is not reduced. The effect of this torsion on the shadow of the heart and of the main vessels will be discussed in the chapters on the mediastinum in health and disease, and on the normal postero-anterior film.

It is interesting to note that the effect of scoliosis on rib-spaces is no longer seen when pulmonary tuberculosis supervenes. Compensatory emphysema seems to undo it.

For all practical purposes the student can consider the bronchi as dividing by dichotomy down to their smallest branches: that is, each bronchial division divides into two smaller branches, and this process continues until we reach the bronchioles. We cannot follow these divisions on the chest film, but we can see the divisions of the vascular supply of the pulmonary artery, and these are so similar, and so closely approximated to the bronchial divisions, that they form a practical guide. They can be traced on a good postero-anterior film throughout three-quarters of the lung fields from their origins in the hilar regions (Figs. 2 and 3).

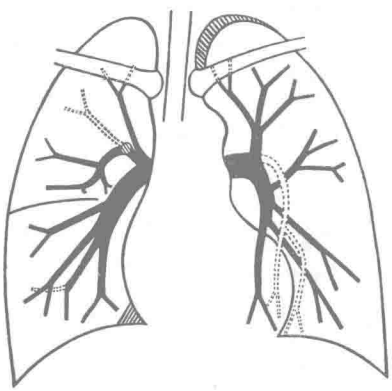

FIG. 2. The Blood Supply. The posterior branch of the right upper lobe blood supply, and the blood supply to the lingula of the left upper lobe are shown in dotted lines.

There is one point of difference that is important when reading abnormal films: blood vessels continue below the shadow

of the diaphragm; bronchial shadows do not, unless they are enlarged by disease.

The diagram of the blood supply shows the branches of the pulmonary arteries as they appear on a good postero-anterior film. The right pulmonary artery makes a T-shaped shadow at the hilum. It gives off three branches to the upper lobe, one to the middle lobe, and three to the lower lobe. The left artery gives an elbow-shaped shadow at the left hilum, about half an inch higher than the right one on a 15 × 12 inch film. It gives five branches to the upper lobe, and four to the lower lobe. It will be noted that much of the left lower lobe supply is hidden by the heart shadow, and that the lower part of the upper lobe is supplied by two branches, convex outwards, that come well down towards the

FIG. 4.

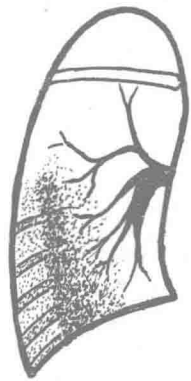

FIG. 5.

FIG. 6.

left cardiac border, supplying the thin triangular part of the lobe that lies in front of the lower lobe.

If any of these normal markings are altered in distribution, or have disappeared, we can conclude which part of the bronchial tree has been interfered with by disease, and so get a very helpful aid to diagnosis.

Thus on the film of right lower lobe pneumonia we see no shadows of the blood supply to the diseased lobe. If we can follow the markings of the rest of the blood supply to the lung we shall find them quite normal in their distribution (see Fig. 4).

If the disease is right pleural effusion we can find the shadows of congested blood vessels in the compressed lung, internal to the shadow cast by the fluid. There is no obliteration of blood supply (see Fig. 5).

On the film of pleural effusion complicating lower lobe pneumonia we shall make out the shadow of the fluid towards the periphery ; internal to it we see no markings of blood supply in the diseased lobe. The rest of the blood supply is normal in distribution (see Fig. 6).

If we are dealing with a case of collapse of the right lower lobe we shall note two effects on the shadows of the blood supply. First, there are no markings in the area of collapse, which is more or less opaque, and appears to be continuous with the heart shadow. Second, as the lower lobe has collapsed and shrunk, the space it has vacated is filled by the upper and mid lobes, which

FIG. 7.

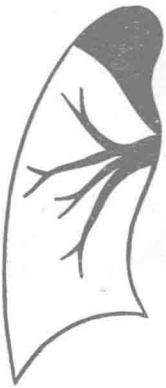

FIG. 8. Collapse of the right upper lobe.

enlarge by emphysema, so that their blood supply is splayed out as against its normal distribution (see Fig. 7). Fig. 8 shows the effects of collapse of the right upper lobe. There is no sign of the blood supply to the collapsed area ; that to the middle and lower lobes is spread laterally as against the normal distribution.

Now and again we come across subjects in whom the normal " dichotomous " division of the bronchi has gone wrong. Development has ceased at one point of bifurcation and instead of a branch we have an air-containing space, blown out by air from the bronchus and kept open by surrounding lung structures. It may be small or large, and is lined by the normal elements of the bronchus. It is known as a " congenital cyst of the bronchus." It can be found in forms which maintain their connection with the original bronchus, but many are closed off, from the first, or

later in life. Some contain a serous sterile fluid, but most are empty. The walls are thin but well enough defined, and the bigger ones may give demonstrable pressure on surrounding structures. When the connection with the original bronchus is maintained through a valvular-like flap these cysts may have a clinical significance, but the usual form has no effect on the subject. On the film these appear as single or multiple circles, sometimes superimposed on each other like heaps of thin curtain rings (Figs. 9 and 10).

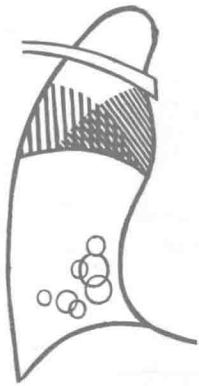

FIG. 9. Showing pneumonia of the pectoral and axillary branches of the right upper lobe, and cystic disease of the right lower zone.

We must remind ourselves of certain of the principal divisions of the bronchi, because they explain to us localised lung pathology. Each of these divisions supplies a fairly large cone-shaped area of lung tissue, the apex of the area being at the entrance of the branch. This is why disease of any one such area, which lies laterally in the lung, appears on a postero-anterior film of the chest as a triangle, and why from the position of such a triangle we can deduce the actual bronchial division involved in, *e.g.*, collapse of certain lobules by bronchial occlusion. Four such branches are of particular interest to the student, all in the right lung, three being connected with the upper lobe bronchus, and one with the lower lobe bronchus.

The right upper lobe, or epiarterial bronchus, has three main divisions: the apical, the axillary and the pectoral. The apical branch goes upwards, outwards and a little backwards. It supplies that part of the upper zone of the lung which is weak in supporting structure, and so is a common site for emphysema and for the first signs of lobar collapse from bronchial blockage. Its air-containing cells soon fall in when they are not kept fully supplied by residual air, which is their principal method of maintaining their position. They may get stretched and become emphysematous by their lack of support, and when they rupture by such stretching may cause a tearing of the visceral pleura to produce a spontaneous pneumothorax. This type of pneumothorax is known as "simple," to distinguish it from pneumothorax of tuberculous origin (see Fig. 11).