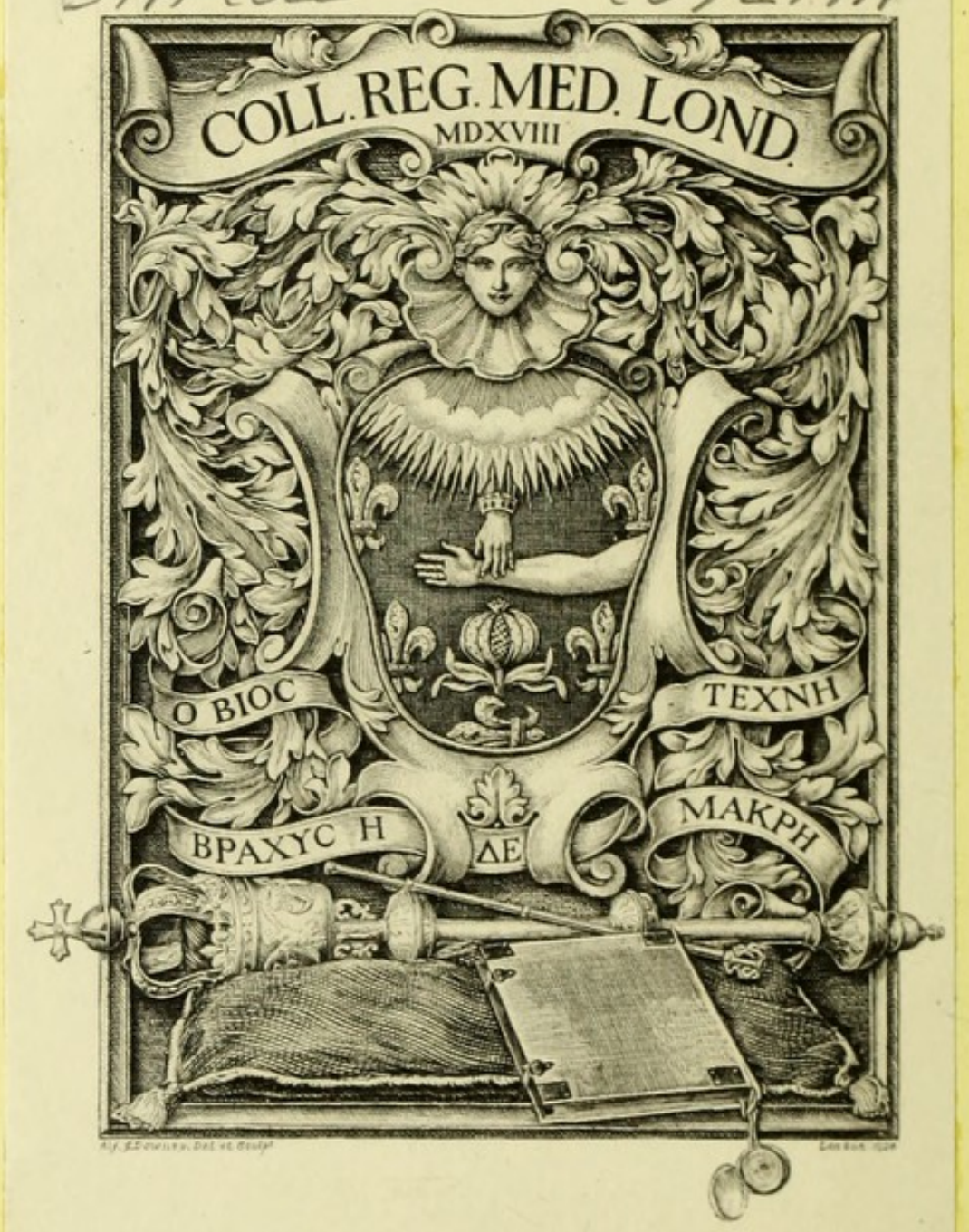
The Bradshaw lecture on intrathoracic tumours and aneurysms in their clinical aspect : delivered before the Royal College of Physicians of London on November 2, 1911 / by Graham Steell.

THE BRADSHAW LECTURE 1911.



The Bradshaw Lecture

ON

INTRATHORACIC TUMOURS AND ANEURYSMS

IN THEIR CLINICAL ASPECT

Delivered before the Royal College of Physicians of London on November 2, 1911

BY

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MR. PRESIDENT, FELLOWS OF THE COLLEGE AND GENTLEMEN,—My first duty is to express to you, Sir Thomas Barlow, my grateful thanks for the honour you conferred upon me by appointing me Bradshaw Lecturer for the present year. With the appreciation of an honour such as this there mingles, I fancy, in the minds of most recipients a sense of the greatness of the task which the appointment imposes. At any rate, it was so in my case.

My predecessor in office gave you an interesting sketch in words of Dr. William Wood Bradshaw, whose widow founded the lectureship in memory of her husband. Dr. Newton Pitt also showed his portrait, the representation of which in the *British Medical Journal* has made the profession at large acquainted with the outward personality of the man whose memory is perpetuated by this lectureship and "whose righteousness hath not been forgotten."

It so happened that the year in which I have the honour of being Bradshaw Lecturer is also the year in which I ceased, by the rules of the institution, to be a member of the acting staff of the hospital in the service of which I had been privileged to be for over 30 years. At such a time it is perhaps only natural that I should look backwards rather than forwards, and—dare I confess it!—clinical medicine seems to me at the present moment to be in danger of losing something of its old charm and, in the future, of losing much more. It was no other than Trousseau who exclaimed some 50 years ago in an address on medicine—"Pray, gentlemen, let us have a little more art, a little less science." Yet who would have welcomed with greater warmth such triumph of science as the introduction into practice of diphtheritic antitoxin? Indeed, "the individual withers, and the world is more and more." This is only evolution after all.

Introductory Remarks.

The subject I have chosen is a very practical one: "Intrathoracic Tumours and Aneurysms in their Clinical Aspect."

I regard aneurysms as tumours—only of a special kind and revealing themselves by special signs. The inclusion of aneurysms may be adversely criticised, but, I believe, I am right in regarding intrathoracic *aneurysm*, as soon as it exerts pressure on neighbouring structures—say the recurrent nerve—*as a tumour*. All who have had much acquaintance with the two conditions—neoplasm and aneurysm within the chest—will, I think, admit the difficulty occasionally experienced in distinguishing between them, even when the greatest care is taken.

Notwithstanding the great help in diagnosis afforded by recent discoveries in physical science and their utilisation in clinical medicine, for years to come, I fancy, the manifestations of intrathoracic tumours will still be investigated at the bedside in the old way—by the *symptoms* and by the *signs* they occasion, or in other words, *subjectively* and *objectively*, though the division at times proves rather arbitrary.

While including aneurysms with neoplasms as intrathoracic tumours, it is the latter (neoplasms) that I wish specially to consider, inasmuch as, paradoxically, aneurysms are common and tumours comparatively rare. The distinction between the two is, however, a matter of considerable practical importance in view of the fact that aneurysms not rarely admit of at least having their symptoms much relieved by treatment and of a useful life being considerably prolonged, while the downward progress of the patient with intrathoracic neoplasm is seldom even interrupted and our therapeutic endeavour is too often limited to the promotion of euthanasia, albeit without any shortening of life. If it be the first duty of the physician to "obviate the tendency to death" of his patient, then surely the second is to relieve his sufferings. Whether we have regard to intensity of actual pain, such as the pain of angina pectoris, or to its persistence, as in aneurysm grinding the vertebræ, or to suffering other than pain, such as suffocation and consequent air-hunger, in the whole field of clinical medicine no class of diseases can be compared with that we have under consideration in potency of production of pain and suffering. Even this is not all: the duration of such pain and other sufferings may be great, whether they be persistent, remittent, or intermittent.

No longer ago than 1889 the writer of one of the best known monographs on the subject, "Affections of the Mediastinum," Dr. Hobart Amory Hare, remarked that "for some unaccountable reason this subject has remained a field in which but few workers have toiled, and whose surface is therefore almost barren." Since these words were

written no doubt several very valuable contributions have been made to the study and literature of the subject, among which I would mention Sir Douglas Powell's and Dr. Frederick Roberts's articles in Allbutt and Rolleston's "System of Medicine," the late Dr. Lindsay Steven's contributions in the *Glasgow Medical Journal*, and most recently the contribution of Dr. Newton Pitt in his Bradshaw lecture of last year.

With these introductory remarks I will now plunge *in medias res*.

Pain.

It is customary to consider symptoms before signs, and in the clinical investigation of the former one has to consider the past as well as the present. I will begin with *pain*, the most *subjective* of all symptoms. Pain is seldom altogether absent in the course of intrathoracic tumours. One may say indeed that it is *never* absent if we include under the term such distresses as dyspnœa and insomnia. Here, however, I use the word in the conventional sense. In inquiring about this symptom in the study of a case one has to bear in mind the difficulty experienced by patients in describing their painful sensations even when present at the moment. How much greater must this difficulty be experienced when a remote pain is being described? As has been said, to recall such sensation in full intensity would be tantamount to suffering it again, and we may conclude that Nature has mercifully decreed that there shall be vague and indefinite recollection of even a severe pain. So, I fancy, clinicists till recently were apt to underrate the value of the symptom—pain—and we owe a debt of gratitude to Dr. Henry Head for the way in which he has shown how fruitful in diagnosis may be the careful investigation of this symptom, the "inwardness" of which, to use Sir William Gairdner's expression, exceeds that of all others. Speaking generally, I think it may be stated that aneurysmal tumours are the more productive of pain, and in their case not only is the frequency of pain greater, but its intensity and its persistency also. Yet the seeming caprice of pain, even with regard to its mere presence, must be borne in mind, and from time to time one is forced to marvel, in the presence of its abundant physical causes, at the absence of the symptom—such cause, for instance, as a projecting aneurysmal tumour, which by its slow terebrating action has gradually but effectively absorbed alike sternal bone and costal cartilage. In striking contrast with such painlessness is the common long-drawn-out agony of aneurysmal pressure on the spine. Actual pain is certainly in frequency, and specially in intensity, much less characteristic of neoplasm than aneurysm, though suffering other than pain—yet quite as great, if not greater—is the common accom-

paniment of neoplasm, for instance, when it compresses the trachea or superior vena cava.

Speaking of intrathoracic tumour in general, different types of pain are met with, the chief of which I will only very briefly enumerate. 1. There may be referred pain—for instance, angina pectoris, which is by no means rare in aneurysms involving the ascending aorta. My experience would lead me to believe that the coronary orifices are always involved in such cases, but I do not wish to be dogmatic, especially in view of the fact that I have had two cases of typical angina pectoris in girls with mitral stenosis as the only cardiac lesion, one of whom died and was found to have perfectly sound coronary orifices and arteries. Pain, and especially tenderness in Head's "inferior laryngeal triangle," should be remembered and inquired for, and if present, associated with probable implication of the transverse portion of the arch by aneurysm. 2. There may be pain, not referred, due to direct pressure on somatic nerves, such as is apt to occur in aneurysm of the descending thoracic aorta or secondary implication of the spine by neoplasm. Interference with other afferent functions of these nerves may be discovered, later, on objective examination. 3. There is the all-important pain, due to pressure directly upon bone, especially the spine. It is a trite saying that long-continued persistent pain in the back should make one think of aneurysm, unless there has already been neoplasm in some other part of the body. 4. Lastly, one cannot ignore the fact, I think, that severe, seemingly local, pain has been complained of in rare cases of rupture of the internal coat of the aorta, although the fact hardly concerns us in our present consideration.

While definite and persistent pain is, then, much more common in the case of aneurysm than neoplasm, indefinite pain and, still more frequently, distress, that can hardly be called pain, are seldom absent in the case of the latter. In the earlier stages of a case of aneurysm, moreover, indefinite fleeting and recurrent pains about the upper part of the chest, shoulders, upper arms, and neck are so common as, at least, to suggest a careful examination for the signs of aneurysm when they have been complained of.

I would remind you, lastly, in the consideration of pain as a symptom of intrathoracic tumour, of the occasional *occipital pain* suffered by patients the subjects of aneurysm of the arch. Twice I have known this symptom so severe as to be the most prominent of the case. The headache is not that common in heart cases and associated with irritation of the tenth dorsal spinal segment from a congested liver. Dr. Head states that when the third cervical segment of the cord is involved, the pain is found to "shoot from the back through the head to the centre of the forehead." From the description I have received from patients with aneurysm of the arch,

the pain seemed to be essentially occipital. The pain of secondary pleural and possibly of lung conditions need only be mentioned.

Dyspnœa.

Among the symptoms of intrathoracic tumour dyspnœa is entitled to an important place. Moreover, it occurs not in one form, but in various forms, and these may be met with in combination. If we consider the effects of obstruction situated, so to speak, at the root of the bronchial tree, on the one hand, and contrast them with the effects of the cutting off, and withdrawing from function, of a large portion of the vesicular field for oxygenation, on the other hand—types of dyspnœa exemplified respectively by laryngeal obstruction in croup and by pneumonia—we have the two extremes of dyspnœic types met with in intrathoracic tumour. But between these there are many intermediate varieties —such, for instance, as occur in ordinary bronchitis with much secretion in the intermediate bronchi. Those types of dyspnœa in which the obstruction is at the trunk or in the larger branches are, in common language, designated *laboured respiration*, and they occur, as might be foreseen, frequently in cases of intrathoracic tumour. On the other hand, when such obstruction has affected one main bronchus and has led to the *complete* withdrawal from function of a whole lung, while the trachea and other bronchus does not come directly under the influence of the obstruction, such dyspnœa as there may be will be expressed by simple frequency of respiration, each act being rapidly and easily performed. When it is remembered that in the first or tracheal type of dyspnœa each act of respiration becomes a laboured struggle, it is obvious that great frequency of respiration is impossible, though it is true that when the respiratory acts are failing, the element of struggle being less pronounced, their frequency may again increase at the expense of their efficiency.

Effusion into the pleura is a common complication of intrathoracic tumour and diminishes the vesicular field and *per se* should induce dyspnœa of the pneumonic type. The two types of dyspnœa contrasted as stated may, however, be present together in varying proportion, but the laboured type is the more subjective and appeals more to the patient, at any rate while he is at rest.

When present, the stridor of tracheal obstruction predominates over all other respiratory sounds and may be heard at a great distance from the patient. It is important to note whether stridor is present with both inspiration and expiration, or with inspiration only, the latter condition pointing to paralytic glottic obstruction. Adventitious sounds, other than rhonchi, are not common, and their

absence or ill-development under the circumstances may be suggestive in diagnosis.

If there be obstruction in the trunk or larger branches of the bronchial tree as the result of intrathoracic tumour, there is commonly observed the familiar "antero-lateral inspiratory retraction" of the lower part of the chest and exaggerated action of the diaphragm so well described by Dr. Sibson, who carefully distinguished between diaphragmatic "effort" and diaphragmatic "descent," as exemplified by abdominal protrusion. Sibson well remarked that "this diminution of abdominal protrusion with manifestly increased diaphragmatic effort allies in this respect emphysema and bronchitis with cases of extreme laryngeal obstruction, in which the same phenomena present themselves." [1]

One knows how in the case of simple copious pleuritic effusion any sign of interference with the function of the unaffected lung becomes a danger-signal, urgently calling for removal of the fluid, and this, though such sign may be only an occasional rhonchus. So in intrathoracic tumour, implication of the lung alone acting is of great moment. There may be only bronchitis or there may be œdema of the lung tissue itself, or there may be both conditions in varying proportion, but in any case asphyxia is threatened.

It is a remarkable fact that, even when one would think partial obstruction to the trachea or a main bronchus must be permanent and little varying, paroxysmal exacerbations are apt to recur from time to time. This occurrence is much more common in cases of aneurysm than of solid growth. Such attacks have been attributed to plugs of secretion becoming engaged at the site of obstruction, but this condition can hardly explain all cases. A most striking feature of these cases is the relief often afforded by the hypodermic injection of morphine and atropine.

Paroxysms of dyspnœa again may form the only evidence of an aneurysm situated at the posterior descending portion of the arch, in cases in which even accentuation of the aortic second sound (to be considered later) is absent, inasmuch as the aorta between the valves and the sac is not dilated. Such paroxysms are often described as "asthma." I once had the curious experience of, within a fortnight, having two patients under my care in hospital suffering from such paroxysmal dyspnœa due to the cause in question. In the first case the cause of the dyspnœa which resembled a cardiac dyspnœa, though no signs of heart disease were obtainable, was not recognised; in the second it was recognised, because the first, having proved fatal, had taught its lesson by autopsy. In both these cases an aneurysm of the posterior part of the arch had compressed

[1] Transactions of the Royal Medical and Chirurgical Societ vol. xxxi., p. 37.

the lower end of the trachea and left bronchus. These two cases presented the best examples I have met with of what Sir William Broadbent called the "aneurysm of symptoms." I take it they exemplified, too, the condition that is least incorrectly included under "asthma" and described as a symptom of intrathoracic tumour, specially aneurysm.

As a matter of clinical experience it will be found that dropsy of the lower extremities and trunk is not rarely observed towards the termination of a case of intrathoracic tumour that does not interfere directly with the inferior vena cava. In the skiagram I show there may be seen a marked projection to the right of the sternum, just below an aneurysmal similar projection, and due to dilatation of the right auricle as a part of general cardiac dilatation. In one case (neoplasm) in which a systolic murmur had long been audible over the whole of the right and unaffected side of the chest, towards the end murmur became audible over the heart itself, apparently as the result of cardiac muscle-failure, and consequent valve incompetence. It often, indeed, happens that towards the end of a case of intrathoracic tumour there is in the dyspnœa the element of cardiac muscle-failure, and the fact may possibly be utilised, in some small degree, as regards treatment. Pericardial effusion—dropsical or inflammatory—may take a share in the production of the dyspnœa of intrathoracic tumour, just as pleural effusion often does.

Pulmonary Changes Resulting from Intrathoracic Tumour.

Before considering the percussion dulness of the side, that is so apt to be developed in the course of intrathoracic tumour, it will be well to have regard to changes in the lung resonance that may precede dulness.

Changes in Lung Resonance.

My predecessor in office, Dr. G. Newton Pitt, called special attention to a very peculiar condition of lung that may arise from *partial* bronchial obstruction, whereby it becomes over-distended, as it were, with air. While respiration is active the air under the circumstances, as Dr. Pitt states, "has greater difficulty in escaping than it had in entering, and the lung becomes over distended. The pressure may be so much raised that the diaphragm may be displaced two inches and the heart pushed out of position." If the whole lung be affected, its main bronchus being compressed or otherwise obstructed, the condition may be confused with pneumothorax, though in the latter case an X ray representation indicates the collapsed lung absent in

the former. Dr. Pitt, no doubt rightly, regarded this condition as usually persisting only for a time, inasmuch as through increase of the obstruction continued ingress of air becomes impossible and the imprisoned air is absorbed, dulness finally replacing the abnormal increase of resonance in question. We are under obligation to Dr. Pitt for directing attention to the subject. On first consideration, such increase of resonance as has been referred to may seem opposed to Skoda's dictum: "That the lungs partially deprived of air should yield a tympanitic, and when the quantity of air in them is increased a non-tympanitic, sound appears opposed to the laws of physics. The fact, however, is certain."

In corroboration of Skoda's statement Dr. Walshe remarked in his classic work: "Skoda is unquestionably right as regards the matter of fact, provided the word (tympanitic) be understood to mean 'tubular' or 'amphoric.'" But these are the only varieties of tympanitic resonance commonly met with over the lungs in disease, inasmuch as the abnormal air-containing space is not large, the "tubular" variety implying a smaller such space than the "amphoric." There is, moreover, no hard-and-fast line between them, as Walshe fully admitted. The essential thing is that *tympanitic quality* should be recognised, and whether we give to the resonance the name of "tubular" or "amphoric" will depend simply on the volume of air percussed. A still fuller tympanitic resonance such as, indeed, is seldom met with over the lung is best described simply as a "full tympanitic resonance," but all three varieties possess tympanitic quality and vary only in "fulness" of resonance or volume of sound.

Let us take a healthy lung removed from the body and percuss it; it yields not the "non-tympanitic" resonance it yielded in the unopened chest, but "tympanitic" resonance, and to restore non-tympanitic resonance, such as the lung yielded in the unopened chest, what have we to do? Restore its tension by blowing it up with bellows attached to its bronchus.

There is no more familiar example of *acquisition by lung resonance of tympanitic quality* than the so-called "tubular" percussion sound above a copious pleural effusion, but the consolidating process of pneumonia offers many examples. I would urge the clinical importance of the recognition of tympanitic quality in lung resonance under abnormal circumstances, and at the bedside students, as a rule, I think, find no difficulty in distinguishing between "non-tympanitic" and "tympanitic" resonance, whether the latter be "full" or "empty," to use Skoda's most useful classification. The assumption of tympanitic quality by the lung resonance—such as it is—will often be observed in the course of intrathoracic tumour, and the principles I have referred to will generally enable its correct interpretation to be made.

That there is a very natural tendency to confuse excessive degrees of "non-tympanitic" resonance with "tympanitic" resonance is admitted, but the distinction can be made. I have often remarked upon Dr. Walshe's accuracy of statement (though he was an opponent of Skoda's teaching) displayed in his definition of simple excess of pulmonary resonance when he used the words in his description of the "quality" of resonance in emphysema—"more or less *approaching* tympanitic."

It may be permissible here briefly to refer to a remarkable occurrence associated with excess of air in the chest outside the lungs and due to a mediastinal tumour composed of tuberculous lymphatic glands. The late Dr. Joseph Coats, of Glasgow, related the following example. An infant, not known to be ill, suddenly developed dyspnœa and subcutaneous emphysema and died in 18 hours. Post mortem the anterior mediastinum was greatly blown up with air. The left lung was greatly distended with interstitial emphysema. The lymphatic glands at its root were tuberculous and there was limited tuberculosis of an adjoining portion of lung. An adherent gland had discharged its contents into the bronchus connected with this tuberculous piece of lung. "An irregular aperture ..... was found in the wall of one of the smaller primary branches of the main bronchus. This aperture led into a cavity in the gland and had allowed the softened contents of the gland to escape and infect the lung and finally the air to escape from the lung to the tissues outside." The case at least illustrates how far-reaching may be the effects of a mediastinal tumour on the respiratory system, though in the adult tuberculous lymphatic glands seldom form a "mediastinal tumour" with its usual clinical phenomena.

Excessive non-tympanitic resonance and the change from the normal non-tympanitic to tympanitic resonance of varying degrees of fulness over the chest form interesting features in the clinical manifestation of mediastinal tumour —aneurysm or neoplasm. Their explanation, of course, is the change brought about in the air-content of the lung and in the tension of its structure. Dr. Frederick Roberts in his monograph in Allbutt and Rolleston's "System," Vol. V., p. 647, goes so far as to state "that a growth may interfere with the trachea only to such a degree as to cause accumulation of air and consequent distension of both lungs." Such change, however, is, as a rule, unilateral or local.

The distinction between the percussion resonance of lung over-distended with air—that is, with a *plus* content of air—and of lung with a *minus* content of air is, I believe, of extreme importance clinically, and in conclusion of the subject I would remind you once more of the dictum of Skoda: "A healthy lung strongly inflated within the thorax so as to be made to press against its walls gives a full, clear, but non-tympanitic sound (on percussion) over every part where it comes in contact with the walls."

Causes of Dulness.

The causes of dulness over the lung area, as a result of intrathoracic tumour, are numerous, and are apt to occur in combination. The tumour itself may be the direct cause, and enormous tumours are on record, almost sufficient in themselves to account for the dulness of the whole side ; but here we are concerned chiefly with those causes that depend on secondary pleural and lung changes. Pleural effusion we have noticed to be a very common result, and Dr. Pitt has shown it to be much more common in the case of neoplasm than in that of aneurysm. The pleura itself may be the seat of new growth, and I have brought a specimen which shows the pleura covered with tumours, though not one could be said directly to be the cause of pressure symptoms. (Fig. 1.)

The patient was an engine driver, aged 58 years, who had fallen down while on his engine and injured his right side and right knee in December, 1908, and who died on April 30th, 1909. The right knee had been injured 13 years before and again later. It showed marked thickening of bone with blurring of outlines resembling an osteo-arthritis when the patient came under observation on March 8th, 1909. On the 23rd he was admitted to the medical wards and the prominent feature of his case was a copious hæmorrhagic right pleural effusion which, after tapping, rapidly reaccumulated, while there was only slight and irregular pyrexia. On April 29th there was found in the fluid a small fragment of tissue composed of large round cells and very probably a fragment of growth as stated by Dr. Loveday, superintendent of the clinical laboratory. I need not refer in detail to the remarkable appearance of the pleural surfaces on the right side, as it can be seen in the specimen I show, and is well seen in the photographs thereof. In one place the right lung below its hilus was invaded by a growth of the size of a plum. Microscopically the growth in the pleura was found to be endothelioma, though "in places it bore resemblance to a round-celled sarcoma." The growth in the joint had "a similar structure." It was "packed with large round cells and practically devoid of stroma." It may seem straining a point to bring this case under the heading of "intrathoracic tumours," though certainly such were present in abundance. Dr. Samuel West's important observations on the adhesiveness, if I may so say, of the pleural surfaces are too well known to require more than mention, but looking at this pleura, the site of almost universal malignant disease, one can hardly avoid thinking of the influence that such disease would be likely to exert upon this remarkable property of the pleural surfaces had there been no fluid to separate them. The pleura may be so thickened by new growth as itself to contribute materially to the dulness of the side present.

In the briefest possible manner I would refer to another of the specimens I have brought and have had photographed, in which the pericardium presented a pseudo-cystic tumour formed by a localised collection of purulent fluid. In a third specimen the pericardium itself is the seat of new growth.

Pulmonary Changes Associated with Dulness.

The pulmonary changes associated with dulness on percussion, as a result of intrathoracic neoplasms and aneurysms, were so thoroughly considered by Dr. Pitt last year that it will be unnecessary again to consider them in detail. I will merely briefly recall Dr. Pitt's statements that: (1) the lung may become collapsed and therefore solid; (2) there may be a so-called "retention pneumonia," the alveoli being filled with catarrhal cells while giant cells, apart from tubercle bacilli, appear (when there is first collapse and the alveolar walls remain long in contact organisation with obliteration takes place and new connective tissue is formed; further there may be a general fibrosis of the lung beyond the obstruction and a bronchiectasis); (3) dilatation of the bronchial tubes which are generally full of secretion; (4) fibroid thickening of the lung; (5) secondary infections resulting in breaking down of the lung, and (6) the still more destructive process of gangrene; and (7) from such processes as the last two, pleurisy and empyema may result.

These lung changes, however, are too often apt to be of little interest as regards auscultation, inasmuch as the bronchus of supply becomes blocked, and the glottic breath sound cannot be transmitted, while, of course, there can be no play of the lung structure. Vocal fremitus and resonance are likewise apt to be annulled. The heart sounds may, however, it would seem, be well transmitted in the neighbourhood of the heart. In the rare event of a tumour mass becoming broken down into a cavity, communicating with the glottis through an unobstructed bronchus, there may possibly be bronchial breath sound audible on auscultation, and certainly there may be cracked-pot sound on percussion. In a case which I published in THE LANCET in 1888,[2] while the latter physical sign was present, the breath sound could only be called "indeterminate."

IMPLICATION OF NERVE-SUPPLY TO THE LARYNX.

In the clinical recognition of intrathoracic tumour the interference with the innervation of the muscles of the larynx that such tumour may cause has been a matter of great

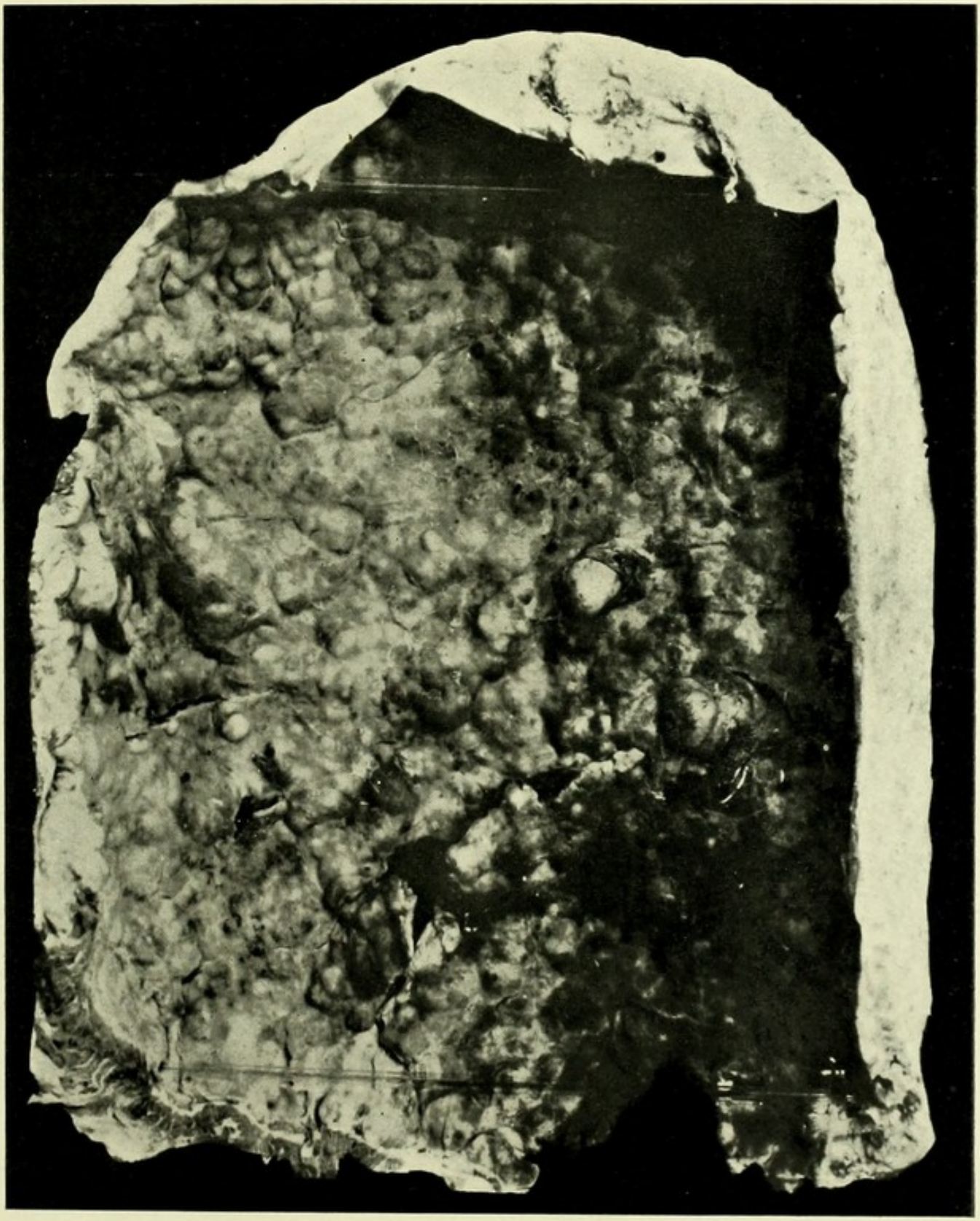
[2] THE LANCET, Oct. 27th, 1888, p. 809.

importance to the physician ever since the invention of the laryngoscope made examination of the movements of the vocal cords and associated parts feasible. Although it is in the case of aneurysmal tumours that implication of the vagus nerve, and especially ot the left recurrent branch, is most commonly met with, aneurysmal tumours have by no means a monopoly in this respect, and I am disposed to think paresis or paralysis of a vocal cord due to intrathoracic neoplasm is more common than it is generally believed to be. As regards the recurrent branch of the vagi, that of the right side hardly enters into our consideration to-day, inasmuch as it lies practically outside the mediastinum. An innominate aneurysm may, however, so extend upwards as to involve the right subclavian artery in such a way as to exert pressure upon the nerve, or, associated with aneurysm of the arch, there may be an independent aneurysm of the subclavian or of the innominate artery involving the subclavian. The frequency of the existence of more than one aneurysm in the same individual is well known. Less rarely the right recurrent nerve is caught as it were in malignant disease of the œsophagus ; but for this to happen such disease must be situated so high up as to lie in the root of the neck rather than in the thorax. Goitre may, again, cause pressure on one or both recurrents, at the same time extending behind the manubrium sterni and so becoming an intrathoracic tumour.

Then there is the interesting question, raised by the late Sir George Johnson, as to the possibility of pressure exerted upon the trunk of one vagus causing interference with the bilaterally associated nuclei of the nerves in the direction of inhibition and resulting in paralysis of both vocal cords—proof of such occurrence does not seem to have been obtained, and it would seem always more likely in a case of double recurrent nerve palsy that some accidental and independent pressure had been directly exerted upon the right nerve as well as upon the left, as, for instance, by an enlarged lymphatic gland in malignant disease, by thickening of the right pleura about the apex of the lung, or by a small independent aneurysm involving the subclavian in the case of intrathoracic aneurysm.

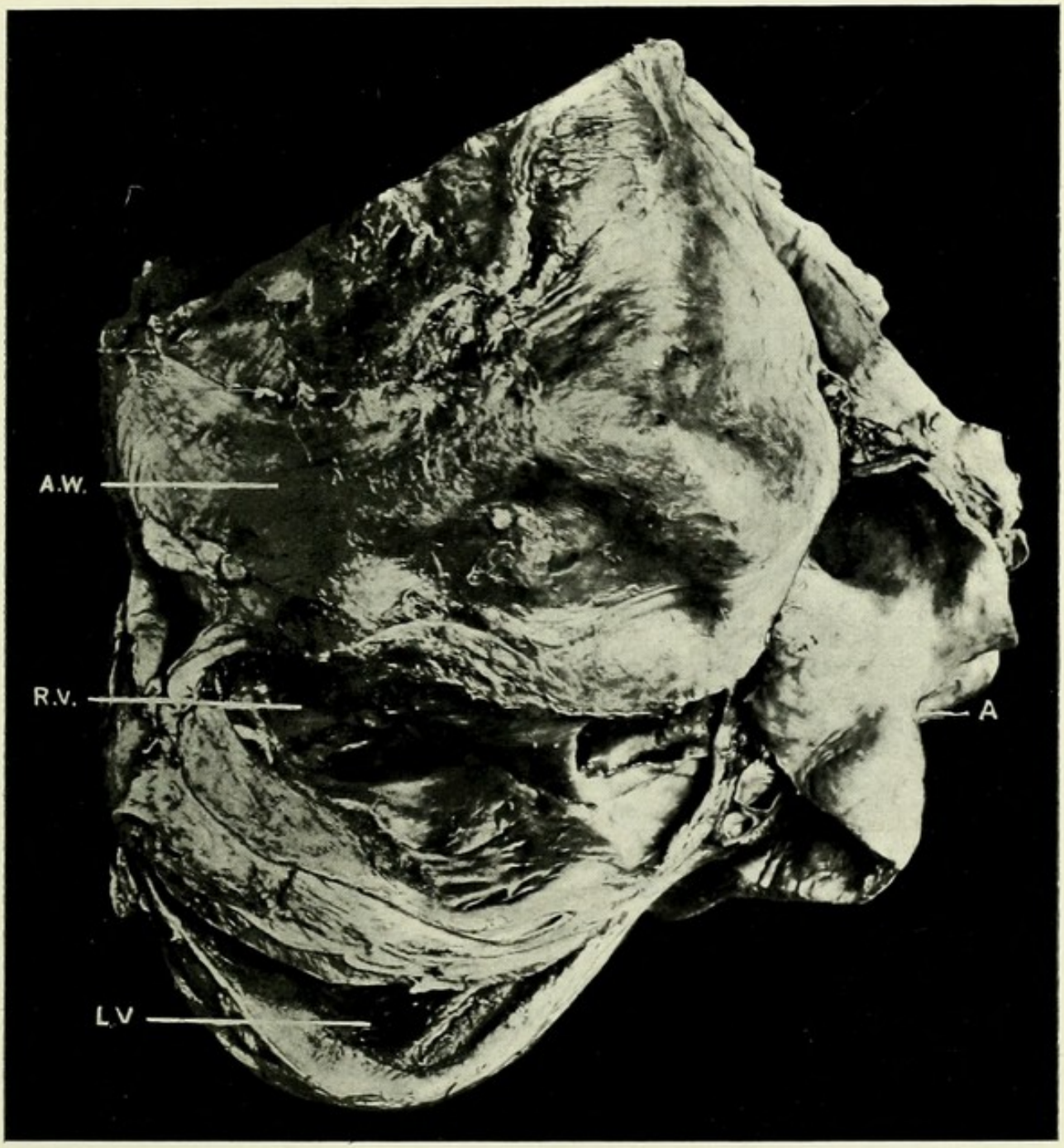
But the interest of bilateral palsy of the vocal cord does not end here ; there comes in the question of the relative predominance of the adductor and abductor muscles respectively of the vocal cords—a matter of great clinical importance. It is now a well-founded belief that the abductors are the muscles to be first weakened under paralysing influence. The fact has little importance, perhaps, when the affection is of one vocal cord only, but it is quite otherwise when there is bilateral paresis or paralysis of the abductors, permitting the adductors to close the glottis without any pronounced degree of spasm and simply by lack of the opposition which the abductors should supply under normal circumstances.

FIG. 1.



From a case of endothelioma of the pleura. The illustration shows the internal surface of the parietal pleura. The pleura is very much thickened and shows many nodules, some of them as large as cherries.

FIG. 2.



Oval saccular aneurysm of the ascending aorta, the size of a cocoanut. It is adherent to and incorporated with the wall of the right ventricle. Adhesive pericarditis is well seen. A.W., Anterior wall of aneurysm. R.V., Right ventricle. L.V., Left ventricle. A., Aorta.

Another consideration, which I do not think has received all the attention it deserves and which was the subject of a thesis in 1865 by Professor Wyllie of Edinburgh University, relates to the concavity of the upper surfaces of the true vocal cords, whereby the edges of the cords may be forced into apposition and, in valve-fashion, the glottis be closed by the *in-rush* of air, especially when the current is forcible and the glottis small and narrow. Physiologists have long known that this valve action of the vocal cords is specially demonstrable in the case of young animals whose recurrents have been cut. Granted any weakness of abduction of the cords, the predominance of adduction will in all probability be rendered more dangerous by this valve action to which Wyllie called attention.

Sir George Johnson in 1875, with reference to a case described by him, remarked: "Looking into (the) larynx with the mirror, I saw the vocal cords of their natural colour nearly touching each other in the middle line and nearly motionless. ..... During inspiration the glottis did not expand as in the normal state, but, on the contrary, the cords appeared to be pressed nearer together by the inspiratory current of air, while in expiration again the cords were slightly pushed apart by the outgoing stream of air."

The greater the dyspnœa the more forcible will be inspiration, and consequently the greater this tendency of the true cords to act in valve fashion in this dangerous manner. The condition in which the abductors of both cords are weak is all the more likely to be overlooked without the laryngoscope, inasmuch as the voice is unaffected, and in quiet breathing there may be little or no stridor, although on any exertion and in sleep inspiratory stridor is usually pronounced. When there is direct pressure of an intrathoracic tumour, especially aneurysmal, upon the trachea, the stridor, as already noted, is usually double, though the inspiratory sound is the better developed as a rule. An interesting clinical point concerning the site of obvious obstruction—in the trachea or at the glottis respectively—is, in the latter case, the so-called excursion of the larynx downwards with inspiration and its return in expiration. While palsy of one cord may affect the voice little or hardly at all, direct pressure of a tumour or aneurysm on the lower part of the trachea may interfere to some extent with and diminish its tone, the clang of the cough being usually exaggerated in both cases.

Lastly, the two conditions, palsy of laryngeal muscles and direct pressure upon the trachea, are quite compatible in the same case. As regards the clinical diagnosis of one palsied cord, this can be reliably accomplished only by means of the laryngoscope, inasmuch as the degree of interference with the voice and the presence of stridor on forcible inspiration seem to vary a good deal in different cases.

With the production of adduction resulting from nerve-irritability and spasm there will again come into play what may be called the valve action of the vocal cords, and the greater will be the predisposition to this effect the more forcible is the inrush of air, as in dyspnœa. A very slight degree of spasm of the adductors, the result of nerve irritability, may then determine a very dangerous degree of closure of the glottis. To such result, however, spasm may contribute only by bringing the cords within the range of their dangerous mechanical valve action.

Affection of the Sympathetic Nerve.

Both in intrathoracic aneurysmal and neoplasmic tumour the cervical sympathetic nerves may be implicated, affection of the pupil, palpebral fissure, and sweat secretion resulting. The tumour must, of course, extend upwards high enough to involve the sympathetic fibres, leaving the cord by the first and second dorsal roots to ascend in the sympathetic chain, and it must extend in the backward direction. As Gairdner stated: "Intrathoracic aneurysm to affect the sympathetic may be expected to arise from the upper and back part of the arch ..... or its primary branches, the sac projecting backwards in the direction of the sympathetic trunk or its ganglia and of their communications with the spinal system." Before he published a case of the kind in 1855, Dr. MacDonnell, of Montreal, had called attention to a case of malignant tumour producing pressure on the sympathetic in connexion with contracted pupil and ptosis on the affected side. The usual affection is paralysis, with the familiar contracted pupil, diminished palpebral fissure, and interference with sweating on the affected side of the head and neck, though the opposite effects may result, it is believed, from irritation of the nerve. Dr. Purves Stewart has shown how well the area of skin rendered sweatless may be mapped out by dusting charcoal over the head and neck. After general sweating has been induced by pilocarpine the charcoal adheres to the normal sweating side, while it is easily blown off the dry side. Doubt has been recently thrown upon the exclusive causation of unequal pupils in the manner stated, and there has been supposed to exist a relationship between the state of the pupils and of the arteries—"with low blood pressure in one carotid the pupil on that side is dilated, with high pressure contracted." The various other causes of unequal pupils that may accidentally and independently coexist with intrathoracic tumour must, of course, be given due consideration in the investigation of a case before the symptom be admitted to have weight in evidence.

Nerve Pressure.

Affection of the brachial plexus, causing weakness and sensory disturbance of the arm, is often included among the symptoms of intrathoracic tumours, especially aneurysmal, but it is obvious that the tumour whatever its nature must extend high up in order to produce the symptom. The affection of the arm in true angina pectoris, with implication of the coronary orifices, must not be confused with that just referred to.

Paraplegia.

One of the rarest clinical manifestations of an aneurysm of the descending aorta is paraplegia. When again the spinal column is invaded by malignant growth, secondary to like mediastinal growth, the same symptom may be developed. It was quite suddenly developed in the case of an aneurysm I witnessed during my hospital residence, the patient, while I was going round the ward, calling out with intense pain in his back and rapidly becoming paraplegic. Secondary malignant growth in the spine is a much less rare cause of paraplegia than aneurysm.

Dysphagia.

It is a familiar fact that dysphagia may be a merely transitory and little pronounced symptom in cases of intrathoracic aneurysm, when the position of the aneurysm, in relation with the gullet, would seem to indicate a good deal of interference with the function of the latter. Moreover, the patient may never have been conscious of such interference under like circumstances. It is a good rule of practice to be careful in passing a bougie for dysphagia, lest the cause of the dysphagia should be an aneurysm.

In malignant tumour of the gullet dysphagia is, of course, an all-important symptom, and the possible implication of the recurrent laryngeal nerves in the growth has to be borne in mind with regard to the larynx.

Hæmorrhage.

Prolonged repeated slight hæmoptysis has long been recognised as significant of intrathoracic growth involving the lung. The late Sir William Gairdner, again, called attention to the oozings of blood that may result from an intrathoracic aneurysm. The important fact, in our present

consideration, is that slight hæmoptysis may occur over a considerable period of time in the course of intrathoracic tumour—neoplasm or aneurysm. Such slight hæmoptysis, as the result of aneurysm, seems to be, as a rule, short and not much over a month in duration. It is curious to note, however, that a much more profuse single hæmorrhage may be survived for years. Sir William Gairdner related a case in which a profuse hæmoptysis occurred nearly five years before the death of the patient. For several years after the first hæmorrhage the patient "continued occasionally to bring up a more or less tinged expectoration—sometimes rusty, sometimes purple, almost never of anything approaching pure blood." At last this patient died after the sudden loss of 8 or 10 ounces of blood which suffocated him. Post-mortem examination revealed a very large aneurysm of the descending aorta "adherent over a considerable space to the left lung and opening freely into the lung and bronchus and trachea. ..... The left bronchus was stretched over the sac and had its posterior wall absorbed throughout its whole length. ..... The sac was filled with firm coagulum" to which no doubt the patient owed the prolongation of his life.

It may be said that hæmoptysis is often a late symptom in aneurysm and an early and frequently a persistent one in neoplasm. In one of my own cases of tumour the patient attended the out-patient department of the infirmary for a slight hæmoptysis, which in the absence of definite physical signs was naturally regarded as probably of tuberculous origin. To increase the resemblance of the case to a tuberculous one, a large tumour mass was ultimately formed at the upper part of the right lung and actually became excavated, but there was fulness in the infraclavicular region, not the usual depression of the tuberculous process.

Hæmorrhage from Complicating Pulmonary Tuberculosis.

When we consider the great frequency of tuberculosis in general, it is not surprising that the disease should be found occasionally complicating intrathoracic tumours—neoplasmic or aneursymal. The affection may be old and obsolete. On the other hand, I have found it active miliary and the cause of pyrexia in a case of aneurysm. It is obvious, therefore, that hæmoptysis may be caused by tubercle, though an aneurysm be present. Rarely, again, hæmoptysis may proceed from lung tissue directly invaded by, and incorporated with, an aneurysmal sac, without the integrity of the wall of the latter being compromised. On the other hand, without any tuberculosis or direct implication of the lung by the aneurysm, blood from the sac may be aspirated into the lung structure, as pictured by Gairdner in his "Clinical Medicine," pp. 482-3.

Hæmatemesis.

Hæmorrhage into the gullet, usually soon, if not immediately, fatal is, of course, a well-known accident of intrathoracic aneurysm affecting the descending part of the arch. However confidently the diagnosis of intrathoracic growth had been previously made it would be a singular trial of one's faith in it, if the case terminated suddenly by a profuse hæmatemesis, and yet this practically happened in a case reported by the late Dr. Lindsay Steven.[3] That the blood did not appear externally is nothing to the point. The patient, aged 57, had suffered from dysphagia, which began in December, 1889. A No. 16 œsophageal bougie passed 13 inches and was then arrested. The diagnosis of cancer of the gullet was made, and on April 1st, 1890, Dr. Steven recorded that he was "quite satisfied that the case was one of cancer of the œsophagus." A great improvement in swallowing and consequent increase of the patient's weight, however, occurred. Dr. Steven was naturally led to doubt the accuracy of his first diagnosis and consider whether or not the dysphagia "might not be due to the presence of an aneurysm or solid tumour at the bifurcation of the trachea." In October he had the "gravest suspicion of aneurysm at the bifurcation of the trachea." On the 18th the patient spat up a small quantity of dark blood, and for the first time complained of pain in the chest. He died suddenly on the 29th, having "raised himself in bed suddenly because of some distress referred to the chest," and then sank back and speedily died with indications of internal hæmorrhage. At the post-mortem examination, on laying open the thoracic aorta (removed from the body) from below upwards "a circular opening with thin and somewhat ragged edges [was] found," which led "into a ragged cavity." On laying open the gullet "at the level of the obstruction ..... a large rugged ulcerated cavity ..... was found in its wall." The upper margin of this excavation was raised and rather sharp and presented the typical characters of the margin of a malignant ulcer. "The stomach [was] greatly distended, being filled with red blood. ..... The duodenum and several feet of the upper part of the small intestine were similarly distended."[4]

Cutaneous Hæmorrhage.

Cutaneous hæmorrhages in the form of petechiæ or ecchymoses may be only expressions of a general hæmorrhagic diathesis in the most malignant sarcomatous growths of the mediastinum, while, in the same case, hæmoptysis,

[3] Glasgow Medical Journal, 1891, vol. xxxvi., p. 123.
[4] Ibid., p. 125.

and other hæmorrhages from within, may result from the same cause. On the other hand, the sac of an aneurysm (generally of the arch) coming forwards may be the source of actual cutaneous hæmorrhage. At first there is usually only blood-staining of the skin, but a local inflammatory condition is often set up, with redness and tenderness, so that, as in a well-known case, the patient may regard the red, tender, and possibly elevated spot as a "boil." The deposition of fibrin in layers is the great preventive of rupture externally as in other directions, and even when hæmorrhage does occur it is often, as Gairdner said, "a mere oozing or succession of oozings filtered through these obstructions." On the other hand, as I have seen, the issuing stream of blood may spout up a considerable distance above the surface of the chest of the recumbent patient. In a case I witnessed, as a hospital resident, the patient survived the rupture many days. Dr. Walshe recorded a case, indeed, in which the patient "must have lived for nearly two months with a gradually increasing extent of his chest wall and aorta replaced by lint."

Physical Methods of Diagnosis.

Inspection.

It will often happen in cases of intrathoracic tumour and aneurysm that the mere inspection of the chests puts the observer almost at once in possession of the diagnosis. It is customary in chest diseases to limit the application of inspection, regarded as a physical method of diagnosis, to the thorax itself, but, specially in the case of intrathoracic neoplasm, it is better to include all that may be obvious to the observer possessing a bearing on the diagnosis under consideration. Thus enlargement of the lymphatic glands above and below the clavicle or in the axilla may afford valuable evidence of malignant growth within the thorax. The significance of external tumour on the thorax, either from projection of an internal one, or of a superficial and essentially parietal one, requires little comment, care of course being taken to exclude superficial tumours, that have existed for many years and clearly have nothing to do with recent developments. Significant tumours in our present consideration have usually appeared recently, or there may be the scar of a surgically-removed tumour.

Intrathoracic aneurysms frequently form local projections, neoplasms less often, and in the latter case independent growth in the chest wall has to be distinguished from projection of internal growth. In the case of aneurysm the seat of projection is usually, as might be expected, the upper part of the thorax often involving the middle line in some degree and extending more frequently to the right than to the left of it. But the projection may be altogether to

the right of the sternum and extend under the nipple itself, encroaching on the situation of a distended right auricle, or in rare cases of aneurysm of the descending aorta, it may be situated to the left of the spinal column. In almost all cases careful inspection over its surface will reveal pulsation, though it is astonishing how easily pulsation may escape observation when there is no projection of an aneurysm. This year only I regarded a dull area as due to intrathoracic growth because I, and those associated with me, failed to observe pulsation carefully looked for. A few days later a somewhat accentuated aortic second sound and diminution of a radial pulse were allowed more weight than at first in the diagnosis, which was changed to that of aneurysm. The patient proved absolutely intolerant of iodide of potassium, which produced quite grave symptoms, and he died in a short time. Post-mortem examination revealed a large aneurysm. Why visible pulsation was absent in this case, overlooked if you will, is a mystery to me to this day. The only explanations I can offer are: that there was much deposition of clot in the sac, and that the heart was evidently greatly depressed, which probably accounted also, to some extent, for the ill-development of accentuation of the second sound, that, along with the absence of pulsation, at first led to the erroneous idea that neoplasm rather than aneurysm existed.

The distended, enlarged, often tortuous, veins coursing over the thorax and obvious in the neck and arms form a conspicuous feature of many cases of mediastinal tumour and aneurysm. This condition is again often associated with œdema of the face, upper thorax, and arms which may reach an extreme degree. It is commonly held, I think, that such a condition is more frequently present in the case of neoplasm than in that of aneurysm, a statement with which I cannot quite agree. At any rate, I have seen most pronounced examples of the condition in aneurysm, one case in a lad of 20. Enlarged veins in a lesser degree may course over the abdomen, the circulation within them passing downward to the tributaries of the inferior vena cava.

Inspection again may indicate important disturbance of lung functions, especially immobility more or less of one side, expansion being always more affected than elevation during inspiration. A difference between the two sides—unilateral change—is often much more obvious to the eye as regards movement than as regards mere shape. For instance, it is a familiar fact that the changes in the contour of the chest produced by copious effusion into a pleura are bilateral rather than unilateral, though difference between the movements of the two sides is marked and obvious.

With unilateral *retraction* of the chest as the result of intrathoracic tumour there will generally go excessive play of the unaffected side. Complete obstruction of the main bronchus is probably in most cases the predominant factor

in the production of unilateral retraction, though the retraction may be more apparent than real owing to the abnormal activity of the sound side.

"Tracheal tugging" is usually regarded as a palpation sign of aneurysm, but the diagnostic value of the sign is much enhanced when the movement of the larynx is so pronounced as to be visible and therefore to become an inspection sign. When this happens there is probably always aneurysm present.

Palpation.

One of the specimens I have brought with me shows a most unusual condition, and one, I may say, therefore peculiarly difficult of diagnosis. I have not been able to find a similar case recorded, though Sir William Osler makes reference to such an one in his article on Aneurysm in Allbutt and Rolleston's "System of Medicine," vol. vi., p. 658—a case of Dr. Gee's. During life in my patient there seemed to be two apex beats—if I may so say—one including and not extending much beyond the normal apex situation and the other quite separate and approaching the left mid-axillary line. Moreover, the whole heart seemed to be thrust forwards in systole, much as it is when a solid mass or an aneurysm lies behind the heart and between it and the spine. This led me to think that there was an aneurysm of the descending aorta behind the heart, causing the heart to be thrust forwards in each systole, though there was always, in this view, the difficulty of explaining the absence of pulsation between the spine and the more external of the two impulses. The curious fact was ascertained by examination post mortem that an aneurysm *in front of* the heart can occasion a pulsation closely resembling that of an aneurysm or neoplasm situated *behind* the heart, so much so indeed as to cause clinically confusion between two opposite conditions. About the same time, curiously, I had in the wards a closely similar case. Although a necropsy was made, it was made on condition that no part of the body should be removed.

The peculiar thrusting forwards of the heart in systole that occurs even when a solid tumour is situated *behind* the heart, no doubt depends on the ventricles of the heart becoming a solid mass of rigid muscle in systole, which mass thrusts itself between the spine and the sternum, with its adjoining cartilages. The spine cannot yield backwards, therefore the sternum has to yield forwards. What may be called the special impulses of the heart are quite lost in a *general* heave forwards. No doubt the instruments of precision we now possess may distinguish a dual pulsation in the case of an aneurysm situated behind the heart, but my old-fashioned ideas make me apprehensive of such blending of the two impulses in the case I refer to as would make their

distinction one of peculiar difficulty. Of one thing I am convinced ; it is : that to the eye, at any rate, the two very different conditions—a solid tumour and a large aneurysmal sac behind the heart—occasion to the clinical observer a very similar condition, while I doubt if the most sensitive hand could convey the double impulse that must, one would think theoretically, be present in front in the case of aneurysm of the descending thoracic aorta.

A valuable sign of intrathoracic aneurysm that has its place under palpation is the diastolic shock, representing the closure of the semilunar valves, that may be felt over a superficial aneurysmal sac. This shock, no doubt, is the palpation-sign corresponding to the peculiarly valuable auscultatory sign of accentuation of the second sound, to be considered under auscultation. Thrills too, generally systolic, rarely double and possibly diastolic only, felt under like circumstances come better under the discussion of auscultatory signs, with the murmurs, to which indeed they for the most part correspond. That neoplasmic tumours may occasionally pulsate seems to be unquestionable, and the fact that the large arteries are often found post mortem undiminished in calibre, though surrounded by new growth, is noteworthy.

Arterial Effects of Aneurysm.

In the distinction between aneurysm and neoplasm within the thorax, evidence from the condition of the arteries beyond the tumour may prove of great value. A difference between the radials, for instance, is a familiar sign of aneurysm of the aorta, though the interference with the affected radial may be brought about in different ways, certainly not always by the interposition of an elastic sac between the heart and the artery examined. Sir William Osler records a case in which there was "no trace of pulsation and not the slightest throbbing in the abdominal aorta or in the peripheral arteries of the leg." The patient "had a very large area of pulsation in the left scapular region" and presumably an aneurysm of the descending aorta. Dr. Fagge described "a case once in Guy's Hospital in which the pulses at the wrists became quite imperceptible." I had myself a similar case. In such cases the warmth and vascularity of the pulseless limbs are well maintained.

Although Stokes referred to the "subclavian or carotid arteries" becoming "obliterated" as a result of malignant growth within the thorax, it is a pathological fact already considered, that the large arteries may maintain their calibre in the very centre of a neoplasmic mass. I think it may be granted that interference with peripheral arteries, in weighing evidence between aneurysmal and neoplasmic intrathoracic tumours, must go to the side of the former.

So called "tracheal tugging" has been already referred to as an inspection sign of aneurysmal tumour of the arch, but it was originally described as a palpation one. The difficulty with regard to it is that with the finger and thumb applied to the lower border of the cricoid cartilage, an impulse, distinct enough to count the pulse-rate by, may be felt in cases in which there is only some amount of dilatation of the arch and no true aneurysm. By making this sign an inspection one we get rid of its minor developments, which are of doubtful import and may only lead us into error.

Percussion.

I have discussed at such length the percussion changes concerning the lungs which arise in the thorax as a result of intrathoracic tumour, that I must be brief here and speak only of such dull areas as the tumour (neoplasmic or aneurysmal) itself may occasion. The most typical area of dulness may be designated a "mediastinal dulness" and is situated at the level of the manubrium sterni and characterised by its *crossing the median line and reaching to neither costo-acromial angle.* Such dull area may or may not be united to the cardiac dulness by a neck of varying breadth and length, and it may barely cross the median line on one side while extending for a considerable distance on the other side. The lower border of a mediastinal dull area is usually convex downwards, whether or not there be a neck uniting it with the cardiac dulness. An aneurysmal sac is specially apt to extend to the right of the sternum, while such a sac springing from the transverse part of the arch may get to the left of the bone, and the more so the further from the valves it has its origin, but the less likely, on the other hand, is it then to displace the lung and reach the surface. An aneurysmal sac low down to the right of the sternum may cause a dull area entirely to the right of the sternum, lower down but at a varying level. It rarely happens that an aneurysmal sac *per se* causes a dull area behind, to the left side of the spine. In the case of neoplasm I have found such an area only when the ribs themselves were the seat of growth. The lungs may in that case escape involvement, and the parietal growth may not acquire such thickness as to annul the lung resonance.

Auscultation.

There is, I think, a curiously widely spread belief that murmur is almost invariable, at least very common, over aneurysmal tumour. This is, of course, very far from being the case. Directing our attention to the physiological sounds of the heart first of all, their departure from the normal is often in the opposite direction, so that a frequent combination indeed is a practically absent first sound and an

extremely loud second sound. During systole the auscultator is aware rather of an impulse—jog or push—than of sound—murmur or sound in the technical sense; of such valuable impression I presume the fashionable double or flexible stethoscope of to-day can give no indication. In many of the cases referred to in which the first sound is indubitably absent, systolic murmur is present, though very feeble and short. The second sound, on the contrary, is of great loudness and intensity—and as we have found on palpation the hand often receives a corresponding shock. The late Dr. James Warburton Begbie called special attention to this sign of aneurysm in 1863 in a paper which Sir Dyce Duckworth has preserved for us in his Sydenham Society "Memoirs" of Begbie.

We know that there are minor degrees of accentuation of the aortic second sound commonly met with, over simply dilated aortas, and, as so often happens with regard to clinical signs, it is sometimes hard to draw the line between such accentuations and that which denotes aneurysm to the practised observer. The impression given to me by the true aneurysmal-accentuation is that of *extreme tension of the valves*, the result of the greatest force, indeed, that could be borne without actual rupture of their structure. I have no hesitation in giving such accentuation the first place among the auscultatory signs of intrathoracic aneurysm involving the ascending arch.

An interesting diagnostic point is the absence of this sign in aneurysms of the arch *that do not involve the ascending part*. An aneurysm of the posterior part of the arch separated from the valves by a non-dilated portion does not produce this sign, but it will be obvious how likely it is that the commencement of the aorta, being atheromatous, will be dilated, and, as a result, there will be a minor degree of accentuation of the aortic second sound. I have already referred to two cases having as the chief symptom paroxysms of dyspnœa, apparently not due to cardiac disease, in which the absence of accentuation of the aortic second sound seemed to negative aneurysm of the first part of the arch, the cases proving post mortem to be cases of aneurysm limited to the posterior part of the arch. The weak point of the sign, strong as it is when well developed, is the gradual transition from the *minor* accentuation of dilated aorta to the *major* accentuation of true saccular aneurysm.

Murmurs.

While I contend that the most valuable auscultatory sign of aneurysm is unquestionably accentuation of the aortic second sound, I admit, of course, that murmurs are very common. The most common murmur is the systolic, but such a murmur is of extremely frequent occurrence in many con-

ditions other than aneurysm, and so its diagnostic value *per se* is small. Among its many causes, may be enumerated actual aortic stenosis, a dilated aorta with the orifice actually larger than normal, degrees of valve-change from slight general thickening to the marked projection of an inflammatory nodule or rigid valve, a thrombus attached to a diseased cusp, and so on. Diastolic murmur when present in aneurysm is often the merest appendage to a greatly accentuated second sound. Even when such a murmur is loud and well conducted down the sternum, the accentuated second sound may still remain to point to the nature of the disturbing process in the valve function—i.e., enlargement of the orifice, *not* diminution of the valve surface. Of cases of aneurysm, involving even the first part of the arch, it must be admitted that in a certain proportion the second sound has ceased, although the presumption is strong that at one time it had been not only present with the murmur that finally replaced it, but had been actually accentuated. In these cases there is extreme dilatation of the orifice, such as to render the valves incapable of so closing as to result in a sound.

In the case, again, of double murmur—even with accentuated second sound—the risk of overlooking aneurysm proper and regarding the case as one of simple dilated atheromatous aorta is considerable. Moreover, pathogically a dilated aorta and a diffuse aneurysm—so-called—run into one another. I draw the distinguishing line for practical purposes at the production of *pressure symptoms* in the case of true aneurysm. I have occasionally had confidence in the diagnosis of simple dilatation of the aorta strengthened by the carotids and subclavians retaining their characteristic exaggeration of pulsation, while in the case of aneurysm these vessels are apt to have their pulsation interfered with. As far as the double murmur of combined aortic *stenosis* and incompetence is concerned, *general* diminution of visible arterial pulsation is of value in diagnosis. Moreover, in its case the second sound tends towards diminution, *not* increase. Local causes, especially interference with the orifice of one or more of the large arteries by a patch of atheromatous disease, may, of course, likewise occasion diminution in pulsation of one or more of the great arteries of the neck.

As a general rule the statement may be accepted that a diastolic murmur audible over an aneurysm of the arch is evidence of incompetence of the valves, and an accompanying accentuation of the second sound is corroborative thereof. There is no incompatibility between accentuation of aortic second sound and diastolic murmur—rather should marked accentuation of the sound lead to careful seeking for the slight murmur that gives evidence of commencing leakage.

So much for the question of the auscultatory phenomena of aneurysmal intrathoracic tumours.

Murmur with Intrathoracic Tumour: Illustrative Case.

The presence of murmur has been regarded as a diagnostic point in favour of aneurysm rather than neoplasm. The rule may be accepted, but there is such an amount of exception that its value is greatly diminished. One of the best examples of murmur in a case of intrathoracic tumour I have met with may be related in some little detail, inasmuch as it illustrates other features of the disease, and I have not yet related a clinical case in any detail. It was the case of a married woman, aged 26, who, when admitted to the Manchester Royal Infirmary on July 17th, 1890, had been five months ill. Paroxysms of cough and slight hæmoptysis had been the early symptoms, curiously relieved after a miscarriage. Dyspnœa and cardiac palpitation soon, however, supervened, and she had a peculiar desire to vomit or retch with a large flow of saliva (a symptom I have recently met with in intrathoracic aneurysm also), "large quantities of clear neutral fluid" being brought up. Loss of weight and strength were progressive. There were no distended veins on the front of the chest. The whole heart seemed to be thrust forwards in systole. The left side of the chest was dull all over with the exception of a trace of resonance in the supraclavicular region. The intercostal depressions showed no levelling, and though the dulness was so extensive it did not transgress the middle line in front. The left side was immobile and vocal fremitus absent over it. There was practically respiratory silence on the left side. Over the right side there was vesicular breath sound with a few rhonchi. The heart sounds to the left of the sternum were at first normal, with the exception of accentuation of the second sound of the pulmonary artery. Over the sternum and the whole of the right side of the chest a loud systolic murmur was audible. There was no laryngeal palsy and the voice was clear. The pupils were normal. The urine was normal. Looseness of the bowels was a troublesome symptom. On July 22nd pain on the left side was complained of, and it continued more or less to the end. The temperature throughout was elevated, the fever being of remittent, irregular type. On Sept. 11th a systolic murmur became audible over the apex of the heart. On the 16th there was œdema of the feet, and the intercostal depressions on the left side were less marked. On the 22nd the intercostal depressions were becoming obliterated on the left side, while the dulness extended to the right border of the sternum, and pulsation was visible to the right of this bone. Profuse perspirations occurred, and the left side of the chest became expanded. A small quantity of pus was withdrawn from the left side of the chest. The patient died on Sept. 27th. Post mortem: The left side of the chest was expanded. The left pleural cavity was obliterated over

the posterior two-thirds of the upper lobe and the posterior half of the lower lobe. The anterior and inferior portions of this pleura contained pus and blood-clot. Left lung consolidated, its lower lobe compressed. The visceral pleura showed apertures through which pus exuded. Lobes of lung adherent. Lung of firm consistence, grey colour, riddled with small, irregular cavities, with ragged walls and filled with pus. In the upper lobe near the hilus there was a mass of reddish-grey tissue continuous with a growth of the posterior mediastinum, the size of an orange, between the aorta behind and the right and left bronchi in front, and reaching as high as the lower end of the trachea and as low as the origin of the sixth intercostal artery. On the left side a process of tumour surrounded the left bronchus, the walls of which were formed of tumour tissue: a No. 12 catheter passed through it. The growth extended half an inch to the right of the vertebral column. On section the growth was found of soft consistence and pale greyish-white colour with pink softer centre. The pericardium contained a large quantity of yellow fluid and flakes of lymph. Its upper posterior third showed nodular growth. Heart thin-walled and dilated. No valve disease. Microscopical examination showed the growth to be a lymphosarcoma.

Published reports of cases referred to in the lecture.—THE LANCET, Oct. 27th, 1888, Case of Excavated Tumour of the Lung. Medical Chronicle, December, 1890, Two Cases of Intrathoracic Tumour. Ibid., April, 1901, On a Sign of Intrathoracic Tumour involving the Posterior Mediastinum. THE LANCET, Feb. 17th, 1894, Clinical Lecture on a Case of Tumour of the Lung.