Substernal aneurism : cases and observations on its diagnosis, and relation to disease of the heart / by A. Halliday Douglas.

With the Authors Compliments

SUBSTERNAL ANEURISM;

CASES AND OBSERVATIONS

ON ITS

DIAGNOSIS,

AND

RELATION TO DISEASE OF THE HEART.

BY

A. HALLIDAY DOUGLAS, M.D.,

FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS, EDINBURGH;
PHYSICIAN TO THE CHALMERS HOSPITAL FOR SICK AND HURT;
FORMERLY PHYSICIAN TO THE ROYAL INFIRMARY.

EDINBURGH: OLIVER AND BOYD, TWEEDDALE COURT.

MDCCCLXIII.

REPRINTED FROM THE EDINBURGH MEDICAL JOURNAL, OCTOBER AND NOVEMBER, 1863.

SUBSTERNAL ANEURISM.

PART I.—ITS RELATION TO DISEASE OF THE HEART.

It is with reference to their early diagnosis that thoracic aneurisms are chiefly interesting to the practitioner. Their early treatment is desirable; and it is pre-eminently true of aneurisms as it is of diseases of the heart, that mistake is not more apt to occur in the early stage, than mismanagement may be hurtful: the measures suitable to other affections of the chest are singularly inappropriate to cases of aneurism.

In writing on this subject, my principal object is to illustrate the grounds on which the diagnosis of thoracic aneurisms usually rests. Recent writers have indicated a disposition to bring different classes of symptoms into a kind of antagonism, setting one means of diagnosis against another, when really none can be dispensed with, and each is important. I think, too, that there is a tendency to forget or overlook the results of past experience; for, in point of fact, some of the most reliable means of diagnosis have been settled long since by the distinguished cultivators of this branch of pathology.

It is no part of my plan to attempt a complete review of the relations or diagnosis of thoracic aneurism. Various attempts of this kind have been made; but in a great measure these resolve themselves into fragmentary illustrations of the subject. Probably the best attempts of this kind are to be found in the recent statements given by Dr Stokes and Dr Walshe in their masterly books on the diseases of the heart. I find, however, that I have been led into the discussion of the correlation of disease of the heart and arteries; and I propose to dispose of this before writing what I have to say on the questions of diagnosis.

In not a few cases of aneurism, the diagnosis will be assisted by observing the co-existing lesions of the heart, lungs, etc.; but the relation of aneurism of the aorta to disease of the heart has an important reference to the pathology of aneurism. In this place I may state the following as among the more weighty considerations connected with the subject.

A diseased state of the arterial coats often exists in conjunction with hypertrophy of the heart; and there is reason to think that the disease of the arteries may affect the system extensively, and that the aorta may become dilated.

The diseased condition of the arteries necessarily impairs the elasticity of their coats; and hypertrophy of the heart may arise as the consequence of their impaired tonicity.

Thus, aneurism of the aorta and hypertrophy of the heart may co-exist as the effects of a common cause.

The co-existence of aneurism of the aorta and disease of the heart is recorded by many writers as of frequent occurrence; but the inquiry into their mutual relation is disposed of in a sentence by even our best authors. Dr Walshe,[1] in his most admirable chapter on aneurism, says,—"The heart may become hypertrophous, especially if the sac originate near the sigmoid valves; but such effect is by no means constant. I have known its size fall quite within the limits of healthy bulk under the circumstances." Dr Stokes[2] holds that the occurrence of hypertrophy is accidental; "and hence we commonly find a healthy heart co-existing with a vast aneurism." On the other hand, an apparently opposite and yet a not really inconsistent view of the question is given by Rokitansky[3]:—"Large aneurisms on the trunk of the aorta have a tendency to produce active dilatation of the heart, and this tendency is the more marked in proportion to their vicinity to that organ. They give rise to this disease either in association with insufficiency of the aortic valves—which is, however, generally the case—or independently of this affection." Elsewhere he puts the matter even more strongly. "Large aneurisms of the aorta give rise to consecutive disease of the heart in the form of dilatation, with a readiness proportional to their vicinity to the heart. It is, therefore, in consequence of the venosity and cyanosis occasioned by the latter disease that aneurism of the aorta affords a decided immunity against tuberculosis."

Of the two former quotations, I may be permitted to say that they present a very partial view of the subject. In the statement by Rokitansky there is a singular want of precision, involving, indeed, the whole subject in obscurity and confusion. It is with diffidence I call in question the views of one so learned and so justly held in reputation, but surely there is a singular looseness in the argument that would ascribe the absence of tubercle in cases of aneurism to dilatation of the heart, which it is admitted results from co-existing disease of the valves. In short, this distinguished pathologist appears to ascribe dilatation, venosity, and consequent immunity from tubercle, equally to aneurisms of the aorta and disease of the valves.

The apparent differences in the statements of these authors, I believe, originate in the different descriptions of cases on which they have founded their reasonings. I am the more disposed to take this view as my own observations have led me to think that cases of aneurism differ from each other in their essential or accidental relation to disease of the heart.

[1] Practical Treatise on the Diseases of the Heart and Great Vessels. 3d Edit. 1862. P. 472.

[2] The Diseases of the Heart and Aorta. 1854. P. 579.

[3] Pathological Anatomy. Translated by Dr Day. Vol. iv. pp. 289 and 296.

Before I proceed to the illustration of the more immediate subject of this part of my paper, I wish to restate a doctrine which lies at the foundation of all my reasonings, viz.,—That the pathological effects of disease of the heart depend chiefly upon hypertrophy and dilatation of the organ. The effects of chronic endocardial and exocardial lesions are physical and local. They do not exert any specific and essential action on distant organs or on the system at large; their primary action is on the muscular substance of the heart. Consecutive hypertrophy arises from the effort of nature to obviate the pre-existing difficulty of the circulation; and, at a subsequent stage, and in unknown states of the system, the increasing capacity of the cavities of the heart, associated or not with an altered condition of the muscular fibre, at length establishes a preponderating dilatation—the immediate consequence of which is embarrassment, obstruction, and venosity of the circulation; and, practically, the effects of a dilated hypertrophy are the same as if the case were simply one of dilatation.

This doctrine was an old one when I endeavoured to illustrate it in the year 1850 in the pages of this Journal. Subsequent experience has increased my confidence in the view that dilatation is the natural termination of all cases of disease of the heart; and that hypertrophy is the result of nature's effort to counterbalance an existing difficulty in the way of the circulation. Moreover, this theory receives confirmation from the history of thoracic aneurisms.

A large number of cases of aneurism of the aorta are accompanied by disease of the aortic valves. Indeed, this is the most intelligible link between co-existing aneurism and disease (*i. e.*, dilatation) of the heart. The disease of the valves and that of the artery have a common origin in coincident atheroma or other lesion of the parts. Accordingly, it is in some sense true that aneurisms situated near the commencement of the artery are associated much more frequently with valvular disease than when the more distant parts of the artery are affected. Now, in such circumstances, the arterial aneurism really has no appreciable relation to the consecutive disease of the heart, which results here, as in simpler cases, from the valvular disease. I shall not multiply cases of this sort: it will suffice to narrate the one that follows.

Case I.—Mrs ——, æt. 53, placed herself under the care of my late friend, Dr Richard Mackenzie, in March 1850. When consulted by Dr Mackenzie, I found the patient in great anguish from dyspnœa, cough, and angina pectoris. She was sitting up, stooping forwards, in bed. She had fixed substernal pain, and pain lancinating to the right shoulder. The action of the heart was tumultuous.

History.—The existing symptoms had been increasing steadily since the preceding autumn, and for some time the slightest effort brought on an attack of angina. Her breath and the heart's action had been liable to disturbance for two years. Chronic articular rheumatism had existed, but there had been no acute attack.

Physical Signs.—There was fulness, without tumour, in the sternal notch. The impulse of the heart was diffused and feeble, and the pulse was small, soft, and irregular. The præcordial dull space was increased, especially in the

transverse diameter, and to the right of the sternum. On the upper bone of the sternum there was dull percussion in a space one inch in transverse diameter. The sounds of the heart were confused,—the second superseded by a murmur loudest at the base. Upwards on the sternum, the first sound acquired a murmurish character.

Progress.—The patient lived six weeks. Slight œdema occurred; dyspnœa became aggravated, with the sound of obstructed expiration. Leeching and stimulants gave temporary relief, and the patient died after two days of peculiar anguish.

Post-mortem Examination.—The heart was bulky, globular, and distended with blood. Both ventricles dilated; the apex cordis was formed by the right. The ascending and transverse portions of the arch of the aorta were dilated, the circumference being five inches. The aortic valves were puckered and insufficient. The aorta was extensively atheromatous; and an aneurismal sac the size of a filbert communicated with the transverse portion of the arch by an orifice, a quarter of an inch in diameter, in the posterior wall. The tumour rested on the trachea, causing a bulging in its interior. The vagus and recurrent nerves were traced; and the left recurrent was found embedded in a dense semi-fibrous mass, which had suppurated along the course of the nerve, and which densified the parts around the tumour, compressing the trachea.

I shall not discuss at present the different questions suggested by this case. It is narrated as illustrating the condition commonly met with, in which the valves participate in the contiguous disease of the artery. In determining the antecedence of the aneurismal, the valvular, or the cardiac lesion, a good deal must be left to conjecture; the priority of these lesions will be indicated better by reference to the history and symptoms of the case,—the anatomy only shows their co-existence. The view I take of the case is, that the combined disease of the artery and of the aortic valves was of long standing, and had originated in the rheumatism which had become her constitutional habit; that about two years before death, disease of the muscular substance of the heart began to declare itself, with the formation of a gradually preponderating dilatation, which became fully developed subsequently to the preceding autumn. About that time she first experienced a more or less complete incapability for exertion; the angina became easily excited; she subsequently suffered from tumultuous action of the heart, dyspnœa, venosity of the blood, and, later, from dropsy, with transversely extended dull percussion, and extended, but feeble, impulse of the heart. The dilated hypertrophy was, doubtless, consecutive to the lesion of the valves and artery; but what part each of these had played cannot be affirmed. It is not improbable that peripheral dilatation of the aorta will be found in some cases to have an influence in superinducing consecutive disease of the heart. In the preceding case, however, the results were not different from what is seen in cases of simple valvular insufficiency; and I ascribe the dilated hypertrophy to that lesion.

There is no evidence that the aneurismal sac in front of the trachea had recently undergone any enlargement. The increased pressure on the trachea, which became so distressing before death, was not improbably connected with the suppuration in the densified tissue surrounding the sac. The pressure of this tumour was

doubtless the cause of the persistently obstructed expiration during the last fortnight, and to it I ascribe her usual posture,—stooping forward in bed. The peculiar angina might be connected with the disease of the aorta; but in some cases of dilated hypertrophy of the heart there is an anguish for which we have no other name.

It is unnecessary to insist that, in this case, and in many such, not the aneurism, but the valvular disease, with its consecutive hypertrophy and dilatation of the heart, was the source of the obstructed circulation that characterized the case in its leading symptoms and mode of death.

Cases have come under my notice which lead me to think that consecutive hypertrophy and dilatation of the heart are more apt to occur in conjunction with dilatation of the aorta (that is, uniform or peripheral aneurism) than in cases of saccular aneurism. It will appear in the sequel that this inquiry involves different questions which have an intimate connexion. There is the question of the capability of the dilated state of the aorta to act mechanically or otherwise on the muscular substance of the heart; and there is that of the relation of the disease of the general arterial system to disease of the aorta and the muscular substance of the heart.

The following case illustrates very well the relation of hypertrophy of the heart to disease of the arteries and apoplexy. I take the case as an average specimen of a large class; and though there is difficulty in determining the sequence of the principal lesions, the class of cases affords good ground on which to rest this inquiry. The case presents features of interest in other respects, to which I shall have occasion hereafter to refer.

CASE II.—*Hypertrophy of the Heart; Dilatation of the Aorta; Disease of the Arterial Coats; Bronchitis; Cerebral Apoplexy.*

Walter Dairsie, æt. 50, a saddler; admitted under my care in the Royal Infirmary, 11th January 1848. A robust man of short stature, and temperate.

He applied for advice on account of dyspnœa; sense of oppression of the chest; cough, and increasing weakness. His breathing was slightly laboured, and wheezing in expiration. He had no pain.

History.—He had been attacked eight months previously with acute chest symptoms after exposure. On recovery, he suffered little for about six months; but then he felt unable for work owing to dyspnœa and debility. He had had an attack of acute rheumatism many years before, but regained perfect health.

Physical Signs.—The præcordial dulness extended from the third left rib, and from the right margin of the sternum five inches in vertical as well as transverse diameter. The upper part of the sternum was dull on percussion. The impulse of the heart was increased in force, and, at the top of the sternum, there was perceptible a sense of the flap corresponding to the second sound of the heart. The *apex cordis* beat in the fifth intercostal space nearly five inches to the left of the mesial line. The heart-sounds were free from murmur; the second was intensified, and had a hollow ringing tone most marked over the course of the aorta. The sternal end of the right clavicle pulsated synchronously with the ventricular systole; the arterial pulse was visible—60 in the minute.

The murmur of respiration was mingled with copious purring mucous rattle in the dorsal regions.

Progress and Treatment.—He was discharged from hospital, and re-admitted again and again; and took little care of himself when out.

In March he was attacked suddenly with vertigo, incomplete insensibility, and imperfect paralysis of the left limbs and right side of the face. He was bled to 30 oz., and he revived shortly. At this date, a murmur was recognised with the first sound of the heart along the course of the aorta. He rallied for a time from this attack, but soon lapsed into a state of lethargic imbecility and gradually lost ground.

He had a good deal of cough, with expectoration. He became subject to paroxysms of dyspnœa, chiefly at night, which gradually became more frequent. Latterly, dyspnœa became permanent, not urgent; and slight œdema occurred. During the earlier paroxysms he was repeatedly cupped with relief; and tartar emetic was well borne. The impulse at the top of the sternum never increased; and the first-sound murmur was at times wanting.

Post-mortem Examination.—The volume of flesh was small. There was slight œdema of the right arm. The heart was bulky, firm, well formed and contracted; it weighed 27 oz. The valves were normal. The endocardium of the left ventricle presented a few atheromatous patches. The aorta was roughened internally by atheroma; and it had a diffused pouch-like dilatation at the commencement of the ascending arch, in the convexity. The circumference measured 4½ inches. Hypertrophy was less apparent in the right ventricle than in the left.

Both pleuræ were partially adherent, and contained a little serum: the right was coated with recent lymph. Lungs gorged.

The arteries of the brain were rigid, calcareous. The lateral ventricles contained two ounces of limpid serum. In the right optic thalamus, there was an old cyst, containing a dark-coloured areolar tissue.

The liver and kidneys were healthy.

Such cases as this are met with very frequently; and I fancy that the usual explanation of them is, that the aneurism or dilatation of the aorta is the effect of the hypertrophy of the heart. I shall not disturb this view of the case; but it leaves altogether out of view considerations of great interest, which tend to throw additional light on the mutual relations of aneurism and disease of the heart. This case, and innumerable others of the same description, illustrate the twofold reference of cerebral apoplexy to disease of the arteries and to hypertrophy of the heart. I do not think it is necessary to maintain, with Rokitansky, that "the occurrence of hæmorrhage in the brain is entirely mechanical," or that "all the predisposing causes are mechanical;" but it will be found in a large proportion of cases of apoplexy in which disease of the cerebral arteries exists, that there is also hypertrophy of the left ventricle, and, though their co-existence should prove less frequent than I think it is, still an important and a practical question arises, viz.:—What the mutual relation of the two lesions is. Now, if it can be made out that the arteries of the system at all generally partake in the altered state so often presented by the cerebral arteries in conjunction with hypertrophy of the heart, then it appears to me that an important point is gained in explaining the relations and pathology of hypertrophy, with reference especially to the mutual relation of arterial aneurisms and disease of the heart.

I am not aware of any observations that conclusively prove one way or other the state of the systemic arteries in such cases; but there is a proof in the state of the arterial pulse which will aid us in the inquiry. The importance of a visible state of the arterial pulse as a sign of hypertrophy of the left ventricle was pointed out by

Dr Henderson nearly thirty years ago; and, after an experience of twenty years, I regard it as a reliable indication of the lesion. I do not remember to have seen any explanation of this state of the arterial pulse; but I fancy the one usually received is, the increased action of the left ventricle. It is probable, however, that in many instances the visible pulse, in its abnormal degrees, is closely connected with an altered condition of the coats of the arteries, and that it may be received as a symptom of a decrease of their elasticity, as well as of increased force of the heart's action. Besides, there are not wanting cases to prove that the arteries may pulsate visibly, and may become tortuous from disease of their coats, though not accompanied by any great degree of cardiac hypertrophy. Now, if an altered state of the arteries, as indicated by this visible state of the pulse, be so frequent, if not invariable, an attendant of cardiac hypertrophy, and if the disease do at all usually extend to the arteries generally, it will be admitted that we have a state of matters fitted to exert an influence on the heart, exactly in proportion to the part taken by the healthy arteries in the circulation of the blood. And, here, let me suggest, that the effect produced on the heart by diseased arteries probably does not depend on the degree of the change their coats have undergone so much as on the extent to which they are affected. Indeed, it will be enough that the elasticity of the coats be at all extensively impaired. For, though the elasticity and action of an individual artery may be insignificant, the impairment of this function throughout the system must cast no inconsiderable burden on the left ventricle.

We perceive thus a twofold condition of the arteries, each having direct reference to the state of the heart, viz.,—such an alteration of the coats of the aorta and other arteries as impairs their elasticity, and may excite hypertrophy of the heart; and a dilated condition of the aorta, partial or peripheral, which may be the effect of the cardiac hypertrophy.

In cases in which a partial dilatation occurs, as in the one narrated, in the greater curvature of the ascending aorta, it is difficult to escape from the conclusion that it is superinduced by the overaction of the hypertrophied left ventricle. I confess, however, that I have felt it difficult to believe that aneurisms of any kind depend directly on the mere force of the action or overaction of the heart. I think there are other influences in operation, especially in causing peripheral dilatation. But, whatever be the mutual relation of hypertrophy of the heart, and partial or peripheral dilatation of the aorta, their usual co-existence with disease of the smaller arteries, and the frequency of cases such as this, appear to establish the fact of an essential pathological correlation of peripheral aneurism and hypertrophy of the heart as effects of a common cause—the impaired tonicity of the arteries. And hence, there is established, through the medium of the disease of the arterial coats, a correlation, of certain cases of aneurism and disease of the heart, which is not accidental but essential.

Notwithstanding the obscurity that invests the subject, it cannot be gainsaid as a foregone conclusion, that hypertrophy and dilatation of the heart are equally distinct from each other in their essential nature and in their effects, and consequently in their separate existence; and, however conjectural our conclusions may be, still it is necessary to trace by their different effects the distinction of each from the other; although in practice they do usually occur combined, probably for this reason that cases do not always come under medical observation till dilatation has set in. Dairsie's case sets forth the ordinary features of hypertrophy of the heart:—force of the circulation; oppression of the chest, without venosity of the blood, or obstructed circulation; the symptoms local; the chief signs physical; the greatest risks accidental, until dilatation sets in; and no such derangement of other functions nor of the constitutional health as might be looked for in spontaneous disease of this organ. It is this remarkable absence of secondary complication in the progress of undilated hypertrophy that has led me to regard it as the natural compensation for some existing physical defect in the powers that maintain the circulation.

The conviction that, in physical diseases of the heart, hypertrophy is nature's mode of preventing embarrassment of the circulation, increased my difficulty in regard to cases of so-called primary or spontaneous hypertrophy of the heart, but now I see it to be a reasonable belief that hypertrophy is always a secondary lesion; and in cases, such as this, of disease of the arteries, it arises in a manner quite analogous to that in which it is induced by physical diseases of the heart, with the view of counterbalancing the obstruction of the circulation which otherwise would arise. In such cases, therefore, we have hypertrophy arising as one of the earliest recognisable indications of a diseased condition of the arteries. We cannot inquire here what is the source or proximate cause of this state of the arteries. It is not a satisfactory solution of the question that it may result by deposit from the arterial blood contained in the affected vessels. There is no conclusive evidence that any change of the blood has specifically to do with atheromatous and other degenerations of the arteries. It is not improbable that the lesion once established will affect the arteries more or less extensively; and in the present state of our knowledge we may regard it as one of the results of rheumatism.

At present I need not write minutely on the influence of the concomitant pulmonary affections from which the patient suffered, especially the acute attack and the chronic bronchitis which appeared to result. The condition of the patient during the later months of his life was largely influenced by the paralytic and mental disturbance arising out of the apoplectic seizure.

There is abundance of evidence to prove that peripheral dilatation of the aorta does not necessarily occasion hypertrophy of the heart, and that such a state of the artery does not require for its

production that the left ventricle should be in a state of hypertrophy. Perhaps one of the most remarkable illustrations of this is to be found in a case recorded by Dr Law of Dublin.[1]

The arch of the aorta was dilated to three times its natural circumference, and the coats of this artery and those of the great vessels arising from it were converted into an osseous tube. The orifice of a large aneurismal sac existed at the commencement of the descending aorta. The heart was of the natural dimensions, and it was not displaced.

It is not to be concealed that there are peculiar difficulties connected with the demonstration of this subject. The difficulties, too, are increased by the limited number of completed observations which individual physicians have the opportunity of making and recording; and from the difficulty of giving such descriptions of morbid conditions, which cannot be very precisely defined, as to render our observations available to other writers: this is true especially in regard to hypertrophy and dilatation of the heart in their relations and combined proportions.

It is not more evident than it is intelligible that, in a great proportion of cases of aneurism of the aorta, there is no tendency to the production of any disease of the heart, and its occurrence is truly accidental. We have seen that even in instances of peripheral dilatation this is the case, owing, I believe, to localization of the disease of the artery. And, no doubt, it is for the same reason that, in saccular aneurisms, the heart usually preserves its normal condition, and hypertrophy and dilatation are unlikely to arise, unless there be disease of the valves of the heart, or coexisting and extensive disease of the arteries.

Moreover, it appears that cases now on record prove beyond dispute that saccular aneurisms of the ascending aorta, even though they should be close to the heart, do not involve disease of the heart as a necessary consequence. It is scarcely necessary to quote cases illustrative of this view; but I prefer narrating cases already published to giving observations of my own.

Dr F. Robinson communicated to the Pathological Society of London[2] the case of a soldier, æt. 49, in whom the heart was small. The valves were competent, but the aorta and its semilunar valves were atheromatous, and the coats of the artery were thickened. A saccular aneurism, which burst into the trachea, existed in the ascending aorta; the orifice of the aneurism presented a valve-like and abrupt fold of the coats of the artery.

Dr Crisp, in his appendix,[3] narrates the case of Mrs Walworth, æt. 40, whose case was supposed to be one of tubercular disease. She had frequent hæmoptysis, and suddenly she died after profuse bleeding from the mouth. The right lung adhered, and was carnified throughout. The heart was small, and the valves healthy. A saccular aneurism, as large as the fist, existed to the right side of the ascending aorta; its orifice was about one inch and a quarter

[1] Dublin Journal, 1842, Vol. 21. [2] October 15, 1862. [3] Treatise, p. 392.

in diameter. The aneurism had discharged into a large bronchial tube.

I do not quote in evidence the often recurring cases of saccular aneurism at the origin of the aorta close to the heart, because it may be argued that their usually early rupture into the pericardium, auricles, etc., does not afford them time to give rise to their effects on the heart. Such cases, however, substantiate the truth of the view I entertain, that mere contiguity of the aneurism does not involve the heart in consecutive disease.

Without insisting upon conclusions arrived at, I conclude this part of my paper by submitting the following as considerations arising out of this subject, fitted to advance and simplify our views of the pathology of aneurism and disease of the heart.

1. Hypertrophy of the heart is probably in all cases a secondary lesion, and is the result of nature's effort to counterbalance a pre-existing hindrance to the circulation.

2. Dilatation of the heart is the natural resolution of local diseases which may, in the first instance, excite hypertrophy. Embarrassment of the circulation, with venosity of the blood, dropsy, etc., usually arises with this consecutive dilatation.

3. Valvular disease of the heart often co-exists with aneurism of the aorta, especially peripheral aneurism; the sigmoid valves most usually are affected, perhaps from contiguity.

4. In such cases the consecutive condition of the heart does not materially differ from that which occurs in cases of simple valvular insufficiency.

5. A diseased condition of the arterial coats often exists in conjunction with hypertrophy of the heart, as is commonly seen in cases of apoplexy with disease of the cerebral arteries.

6. There is reason to think that the disease of the arterial coats may exist extensively in the system, though the aorta and the arteries at the base of the brain usually present its most advanced effects.

7. In this state the aorta is apt to undergo dilatation, constituting usually peripheral aneurism.

8. The diseased state of the arteries destroying their elasticity, the circulation is to that extent obstructed, and the left ventricle, under the additional burden, undergoes hypertrophy to compensate the lost tonicity of the arteries.

9. In such circumstances hypertrophy arises in a way analogous to that which occurs in cases of disease of the valves, etc., of the heart.

10. Consequently, hypertrophy of the heart and peripheral aneurism stand associated together as effects of the same diseased condition of the arteries.

11. Consecutive preponderating dilatation may occur in such cases; but the progress to that stage is liable to be hindered by the accidents of hypertrophy, as, for example, of cerebral hæmorrhage, which is promoted by the co-existing disease of the arteries.

12. Aneurism of the aorta is often altogether local in its origin,

and has no tendency to involve the heart in associated or consecutive disease.

13. Such aneurisms are usually saccular, but they may be peripheral, and they suggest the probability of localization of the disease of the coats of the artery.

14. Proximity to the heart in such cases does not affect that organ.

PART II.—DIAGNOSIS.

The cases of substernal aneurism recorded in this part exemplify some of the more usual, and therefore more important conditions in which the disease occurs. They are intended as clinical illustrations of the most reliable indications of the lesion, and the narratives of the cases will be trusted to for the illustration of many of the signs; while, in the commentaries, some points only will be referred to. No attempt will be made to discuss particular questions, nor the details of the cases; indeed, the only question considered with any minuteness is the diagnostic value of aneurismal second sounds; the supreme importance of that subject has induced me to remark on it at some length, under Cases IV. to VIII. The subjects of *pain*, *respiratory obstruction*, and *tracheotomy*, also, are specially referred to; and Cases X. and XI. are given as instructive instances of the disease masked by co-existing lesions.

The cases now to be given chiefly belong to the class which, in the former part of this paper, were designated *saccular*, and which in general have no essential relation to disease of the heart. The occasional co-existence of *peripheral* aneurism (dilatation) of the aorta in its slighter degrees, does not interfere with their clinical character as saccular aneurisms. For clinical purposes, it is better to view substernal aneurisms in these classes, founded upon their external figure, rather than to arrange them according to the injury or disorganization of one or several of the coats of the arteries. To prevent misunderstanding, I may define in this place what is meant by the terms peripheral and saccular aneurisms. Peripheral aneurism indicates true aneurism and dilatation of the artery. Saccular aneurism embraces false aneurism, whether it be in the form of a sac or merely a deep pouch, widest at its communication with the artery.

Perhaps some will question the advantage of such records of cases in which so many particulars necessarily repeat themselves; but the never-ending variations in the symptoms of substernal aneurisms render the study of recorded cases essential. Indeed, I am persuaded that the most instructive reading on the subject of the diagnosis of substernal aneurism, are condensed narratives of carefully observed cases. No reference occurs to the changes in the pupil of the eye connected with thoracic aneurisms, for the reason that the symptom did not occur in any of my cases. The papers of my friend Dr W. T. Gairdner[1] on that subject will repay

[1] Clinical Medicine, 1862.

perusal; but probably results will prove that the symptomatic value of these conditions has been over-estimated. Connected with that subject, the very important investigations of Dr Ogle[1] are well worthy of study, not with reference to aneurism alone, for the subject involves a larger inquiry.

I may appear to limit my view too exclusively to the physical relations of the cases; but it is scarcely possible to do so. It has become the fashion to talk of the "general," the "functional," and the "physiological" symptoms of aneurism: and in a limited sense I do not object to these phrases; but it is of consequence to keep in view that the conditions referred to are for the most part the accidental results of a purely mechanical cause, whether the organs suffering lie contiguous to the aneurism or distant from it. Experience confirms the remark of Dr Hope,—"Where an aneurism is buried deep in the chest, and not capable of being detected by sight and touch, it does not present a single general sign which is peculiar to itself, and therefore pathognomonic of its existence." There is something fixed or essential in the general or vital symptoms of any disease, which is wanting in aneurism;—accordingly, as it is somewhere written, "in any case presenting symptoms connected with the organs in the chest, and an apparently sound state of the general health, aneurism should be suspected." This is in a degree exemplified in the following case:—

CASE III.—*Aneurism of the Ascending Aorta; no Aneurismal Sounds; Signs antecedent to Prominence and Impulse; Sudden Death.*

On the 20th June 1862, I was consulted by Dr Turnbull of Coldstream in the case of his patient, an energetic and robust man, who had led an active life as an extensive farmer. His age was 50, but he appeared younger. He had lost a stone in weight. He complained of pain in the upper part of the chest, on the right side, extending from the front to the scapular region. His breathing was very slightly embarrassed, and his other functions were unaffected. Pulse 80.

History.—At no time had his health been materially disturbed; the pain of his chest had been severe for about two months, especially when cough occurred; and in the previous autumn he had consulted Dr Turnbull on account of pain which at the time appeared to be muscular.

Physical Signs.—On the right front of the chest there was slightly defective movement during inspiration; and the percussion sound was impaired as low as the fifth rib, with tenderness in the third intercostal space. On the right summit there was developed expiratory sound amounting to tubularity, and the vocal resonance and thrill were slightly augmented. No rattle of any kind accompanied respiration. The heart-sounds were unaltered, but subsequently they became sharper on the second and third right costal cartilages.

Progress, etc.—During the week following, as I learned from Dr Turnbull's first letter, his patient suffered much—his pulse at times as high as 100, and he "could not lie for a moment off the affected side." His gums were soon but slightly affected by the calomel and opium which I had advised, and repeated small blisters rose well. Digitalis and nitre were then administered; and on the 7th July the patient was "much better, free of pain even on forced inspiration, and able to enjoy a little claret with his chop. Pulse 77." He now thought himself quite well. He visited the International Exhibition before the end of July, and soon regained nearly the weight he had lost.

[1] Med. Chir. Transactions, 1858, vol. xli.

In the end of August he suffered from return of pain; and on the 8th September, when again I saw him, he looked well. I found the dulness of percussion diminished in extent, but decided on the second, third, and fourth right costal cartilages, and on the sternum. The cardiac sounds were slightly sharper and more distinct in this situation than in the region of the heart. No impulse could be detected. His pain was not affected by posture nor by forced breathing. He often felt uneasy without pain when first he lay down at night, and he lay awake more than formerly. The treatment now was restricted to the alternate application of iodine and belladonna, with general care and quietude.

From this date the advance of the disease may be said to have been progressive. I did not see the patient till the 20th March, when I found the evidence of aneurism complete, with slight tumour and pulsation. I had been prepared for this by Dr Turnbull's letters, to which I am indebted for all I have to say of the subsequent progress of the case. In December, Dr Turnbull applied to me for a statement of my views of the case, to be submitted to Dr Watson, of London, whom it had been resolved to consult, in consequence of the unfavourable opinion I had given in September. Dr Watson's opinion was decided, that an aneurism of the ascending aorta existed. He referred to "a spot midway between the clavicle and the level of the nipple where pulsation existed, with prominence and soreness on pressure."[1] From Dr Turnbull I learn that during January and February his patient suffered little or no distress, and "could with great difficulty be persuaded that anything was seriously wrong with him." His appetite was good, he enjoyed his claret, and took the tincture of the muriate of iron with little intermission from the time he had seen Dr Watson. "His chief complaint was of distress experienced on first lying down in bed." In May the prominence of the right costal cartilages began greatly to increase, with tenderness and impairment of the respiratory murmur of the right side; the cardiac sounds were unaltered to the last. Pain and dyspnœa were never continuous till within a few days of his death.

In the end of July he had alarming paroxysms of dyspnœa, subsequent to which he rested best in an easy chair, and he preferred to stoop forwards, leaning his elbows on his knees. During the last month his voice was occasionally changed; he had a sharp ringing cough, and, latterly, occasional fits of dysphagia. A few days before his death swelling of his face occurred; his feet became œdematous, and dyspnœa continuous. On the 27th August he suddenly exclaimed he was dying. His face was intensely purple, but became pale; his tongue, also, was very pale; his breathing laboured, and his pulse full. In half an hour he sank. There was no external hæmorrhage. Immediately after death the surface became extremely blanched. Of the treatment, Dr Turnbull says, "Anodynes administered internally never gave any relief. The greatest degree of relief was from iodine, atropine, or chloroform, applied locally. Latterly, the only comfort he obtained was from leeching or the free application of chloroform."

The value of this case depends in a great degree upon the opportunity of observing the symptoms before prominence or impulse of the aneurism existed. The difficulty, in the first instance, was to determine whether the apparently pleuro-pneumonic state of the upper part of the right lung was primary or secondary; and having

[1] Since the publication of this case, Dr Watson has favoured me with a copy of his minute and singularly complete report of the case, dated 9th and 12th December. In reply to my question, as to the state of "impulse," Dr Watson says, "Certainly it was obscure." In the words of his report:—"The heart-sounds, and even I think a beating, can be heard and felt at the suspected spot, more loudly and more jerkily than over the heart itself." . . . "Had much pain last night. The pain 'cuts' the suspected spot on a full breath. At night, a gnawing pain extends thence down the right arm."

The value of these observations is very great, showing that it was between November and March that the impulse first occurred; and confirming what I have said at page 26, regarding pain in cases of aneurism.

decided that it was secondary, what evidence was there that the dull percussion over the sternum resulted from aneurism, and not from a new growth in the mediastinum. The former of these questions was decided, not without hesitation, and only after apparent improvement in the state of the lung, with, at the same time, increasing dulness of percussion over the mediastinum. To dispose of the second question, we find the following considerations:—The comparatively unbroken state of the patient's general health; his muscular and energetic habit; the history of his pain, alternately acute and intermitting completely, with a tendency to fix itself in one situation, where tenderness soon occurred; comparative freedom from cough and expectoration; the existence of pleuro-pneumonia in an unusual situation; dulness of percussion over the supposed seat of tumour, and increased distinctness of the cardiac sounds.

These symptoms leave no doubt as to the presence of a tumour. That it was an aneurism could only be conjectured; but, on the ground chiefly of the unbroken health, and the history of the pain, it became what Dr Latham calls a "sober conjecture;" and too soon it was verified. Of the other symptoms in this case I shall have occasion to write in the sequel; of his pain, at page 26; of pulmonary inflammation, at page 31; and of the stooping attitude, at page 29. Of the mode of death, too, I shall have a word to say in connexion with Cases IV. and X.

The absence of direct stethoscopic signs is frequent, and may be the source of most perplexing difficulty. In such circumstances, as in this case, sharpness and increased distinctness of the heart-sounds, away from the region of the heart, acquire an importance we cannot afford to overlook. It was not until my second examination of the case that this sign duly impressed me, when it was accompanied by circumscribed dulness of percussion. Isolated, this sign would be valueless; but with other grounds to suspect the presence of aneurism it has a value. At the same time, it must be borne in mind that in cases of aneurism one or both sounds may be less distinct than natural.

Before recording the immediately succeeding cases, a few preliminary remarks on aneurismal second sounds are here necessary; as, however, I desire to limit my remarks to a purely clinical illustration of this subject, I shall not discuss their mechanism. The sounds heard over substernal aneurisms are of two kinds—murmuring or pure, and these may be systolic or diastolic: I refer, of course, to the systole and diastole of the heart.

The aneurismal diastolic or second sounds have been known for long to possess a much more precise diagnostic value than systolic sounds do; and their diagnostic value in substernal aneurisms makes it the more remarkable that they are not met with in abdominal and in external aneurisms. This renders it very probable that the phenomena of the second sound in substernal aneurisms depend more or less upon communication from the heart;

as occurs in such an experiment as the following:—Let an india-rubber bag, with a tube attached, be distended with water, by means of an elastic forcing bag having a valve in the nozzle which communicates with the tube. The sudden expansion of the forcing bag (its diastole) is followed by the audible flap of its valve; but when the india-rubber bag is fully distended the sound produced at the valve is now heard in the distant bag, intensified and having a cavernous tone, from the diffusion of the sonorous wave in the globular bag of fluid, though at a distance of several feet from the valve of the forcing bag.

As a general rule, the *character* of an endocardial or arterial murmur has no diagnostic value,—it may be harsh or soft, musical or blowing. It is otherwise, however, with the cardiac second sound, which is liable to changes in its intensity and in its timbre. Increased loudness or sharpness of the second sound, in the aorta or in the pulmonary artery, accompanies a variety of conditions of the heart and of its action, and these possess more or less diagnostic value; but the timbre of the second sound is apt to acquire a peculiar intensified, ringing or metallic, quality of great diagnostic value. The difficulty of describing the quality of a sound has led to a vast number of terms being employed to designate this one. In clinical teaching I was accustomed to style it "intensified, with a hollow metallic ring or boom." Dr Stokes terms it "ringing second sound." Dr Walshe "twanging" or "pumping." The most recent reference to it is by Dr J. W. Begbie,[1] who adopts the name of "accentuated second sound." In the year 1836, Dr Henderson[2] demonstrated the importance of this altered timbre of the second sound, and compared it to the closing of a pump-valve. Dr Henderson's patient was a man about 50. He presented dull percussion on the left front, as low as the sixth rib, with well-marked impulse. "After the slow impulse in these parts had terminated, and while the heave of the chest produced by it was subsiding, a remarkably clear and loud sound was audible, bearing a very striking resemblance to the sudden shutting of a pump-valve in the immediate vicinity of the ear. The sound was of nearly equal intensity at all points where the impulse existed." In this case, the heart was of natural size. A large aneurism occupied the space from the left clavicle to the fifth rib in front, and it extended to the spine. The aneurism commenced an inch from the aortic valves, and involved the left wall of the arch as far as the origin of the left subclavian artery. The sac contained a maze of laminated clots, with a dirty grumous-looking fluid intervening.

The "equal intensity" of the sound in this case probably arose from the size of the sac, its diffused proximity to the surface, and possibly the extent of its communication with the aorta. The case was admirably suited for unfolding the value of this state of the

[1] Edinburgh Medical Journal, No. 96. June 1863.
[2] Edinburgh Medical and Surgical Journal, vol. 45.

second sound, the peculiar and distinctive character of which does not depend upon its *accent* so much as upon its *timbre*. In its higher degrees it has a metallic twang, but sounds as if produced in or communicated to a cavity full of fluid, and the facts substantiate this resemblance. I would therefore suggest, as a fitting description of it, that it be termed the cavernous second sound, or cavernous intensified second sound. Booming or ringing expresses the quality of the sound defectively, as the flap of the valves terminates the sound abruptly, and prevents the prolonged vibration implied by these terms.

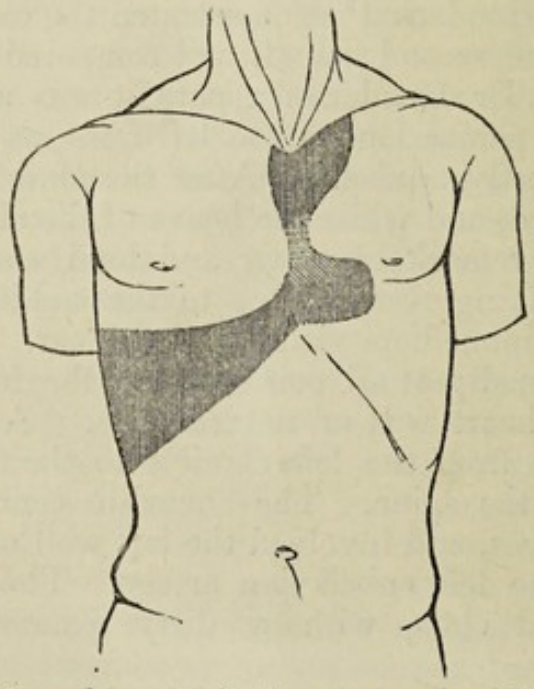
The immediately following cases, No. IV. to No. VII., illustrate some of the conditions in which this state of the second sound is observed.

CASE IV.—*Saccular Aneurism of the Arch of the Aorta; Sac large and empty; Cavernous intensified Second Sound; Cardiac Sounds at the base of Heart, normal; Death by Hæmorrhage into the Cavity of the Pleura.*

Georgina Macpherson, æt. 34, house-servant, admitted under my care in the Infirmary on account of trivial symptoms with amenorrhœa, an ulcer of the leg, etc. In the end of October 1851, the following report was taken:—She complained of fixed and lancinating pain in the upper part of the left front of the chest, extending to the shoulders and left upper extremity. She had difficulty in resting on the left side, and her breathing was very slightly impeded.

History.—She had experienced pain for some months, and she had coughed without expectoration. She had not suffered any acute attack, and stated that her previous health had been good. She had derived temporary benefit from opiates.

Figure 1.[1]



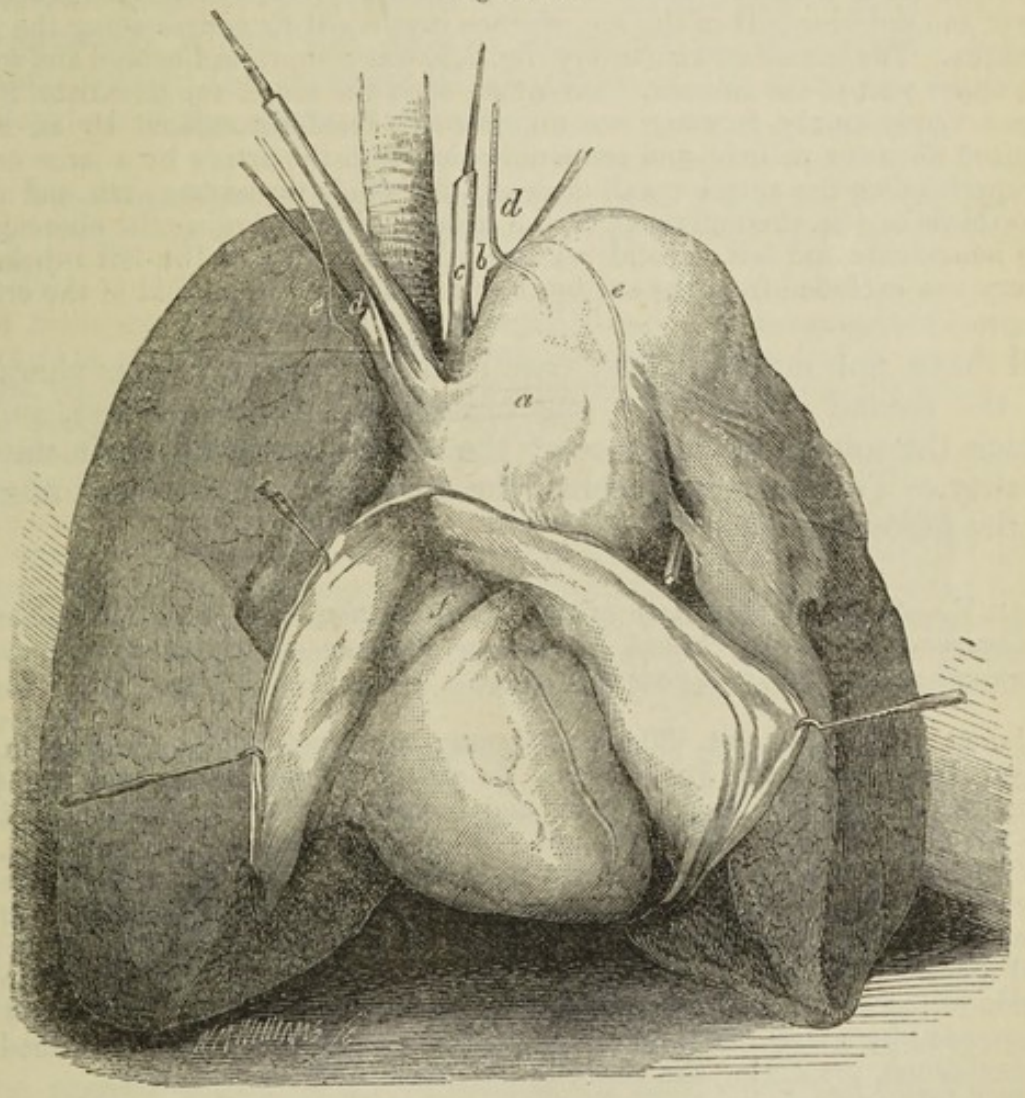
Physical Signs.—Coverings of chest were spare, and there was slight prominence of the left subclavian region, and diffused visible pulsation, with sense

[1] Figure 1 shows the extent and situation of dull percussion over the aneurismal tumour in the left subclavian region, and over the heart and liver. The paler shading on the middle part of the sternum represents the more resonant tone of percussion where the adherent lung intervened between the heart and the tumour.

of impulse more marked than that of the heart. Pulse was wanting in left wrist. There was acute tenderness on percussion of the left front, and there was dulness on the inner half of the left subclavian region, and upon the upper two inches of the sternum. The præcordial space dull on percussion, commenced in the fourth intercostal space. The hepatic dulness was as usual. The precise site of the apex cordis could not be determined. In the fifth left intercostal space the heart sounds were faint, the second being somewhat more distinct, with an occasional reduplication. At the fourth intercostal space, close to the sternum, the second sound was distinct, clear, and not intensified. As examination was made upwards, the second sound became more and more intense, and in the centre of the subclavian space acquired its greatest loudness with cavernous metallic boom; and the first sound acquired a harsh prolongation. Behind, there was dull percussion on the left summit,—feeble respiration and suppressed vocal vibration.

Subsequent Progress.—During examination, and at other times, she complained much of pain and restraint in lying supine. Her cough was only occasional and slightly husky. She gradually lost strength and became almost incapable

Figure 2.[1]



of moving from acute lancinating pain in the upper part of the left side of chest and shoulder. She lost flesh and became slightly sallow. About the end of

[1] Figure 2.—*a*, Aneurismal tumour. *b*, Left subclavian artery, its cut extremity concealed and compressed behind the tumour, with a director in the orifice. *c*, Left carotid artery. *d*, *d*, Pneumogastric nerves, the left lost in the sac wall. *e*, *e*, Phrenic nerves. *f*. Aorta at its commencement, within the pericardium.

December she became more feeble and languid than previously, and on the morning of the 28th she became suddenly faint. She rallied, but died in the course of an hour or two.

Post-mortem Examination disclosed a large clot of blood in the left pleura. There were firm cellular adhesions of the upper lobe of the lung posteriorly. The heart and its valves were healthy, except glistening white spots on the pericardial surface. The upper part of the left lung was displaced by an aneurismal tumour considerably larger than the fist; and the compressed upper lobe of the lung was closely adherent to the outer and back surface of the tumour. Rupture of the sac by a very small slit, had taken place at the outer and lower part of the tumour, where screened by adherent lung. On the left side, the par vagum (fig. 2, *d*) was found incorporated with the wall of the sac, at its upper part, so that it could scarcely be traced; it was displaced outwards, and was lost where the lung adhered to the sac. The recurrent nerve of this side was found in the first part of its upward course, in firm contact with the posterior wall of the aneurismal sac. Tracing it downwards, it was found to emerge from the sac-wall, but could not be traced to its connexion with the vagus nerve. At the upper part, the phrenic nerve (fig. 2, *e*) on this side was found entering the sac-wall, in which its fibres could be traced imperfectly; it emerged at the lower and anterior part of the sac, whence it pursued its course along the pericardium. The left subclavian artery (fig. 2, *b*) was compressed behind and within the upper part of the tumour. On laying open the sac of the aneurism, it was found nearly empty, forming one uniform sac, lined throughout by an unorganized fibrinous pellicle, and communicating with the artery by a large orifice comprehending the anterior wall of nearly the whole transverse arch, and about two-thirds of the circumference of the vessel; thus including the openings of the innominate and left carotid arteries. The aperture of the left subclavian artery was excluded from the sac by the projecting marginal fold of the orifice.

I have not met with any case in which the cavernous character of the second sound over the tumour was more marked, and in which the sounds at the base of the heart preserved their natural quality. The same condition of the second sound was very marked in the following case.

CASE V.—*Saccular Aneurism of the Arch of the Aorta at its termination; Cavernous intensified Second Sound over Tumour—at the Heart, both sounds normal; Death from Effects of Fatigue.*

James Henderson, æt. 30, flax-spinner; admitted under my care in the Royal Infirmary, 12th June 1845. A well-developed man, suffering from cough with expectoration, dyspnœa, and hoarseness, accompanied by pain in the left upper front of chest, extending to left shoulder.

Physical Signs.—There was slight prominence of left front, and an obscure impulse in this subclavian space, distinct from the impulse of the heart. In the same situation, there was dull percussion with tenderness. The præcordial dull space was of the natural extent. The cardiac sounds were normal in the region of the heart; but upwards, in the subclavian space, the first sound was superseded by a sharp bellows murmur; the second sound was intensified and hollow-toned, presenting the characteristic metallic cavernous ring.

The left internal jugular vein was permanently distended; the left radial pulse was small; and the respiratory murmur was impaired in the left lung.

History.—He stated that the existing symptoms had arisen within ten weeks, and that his illness originated in the effects of exposure to cold, with rigors, headache, oppression of the chest, cough, with expectoration and hoarseness. He had been relieved by active treatment, but the hoarseness had undergone no improvement.

Progress, etc.—He left the hospital on the 22d June, contrary to advice, and travelled to the country a distance of ten miles. Before he recovered from the fatigue of the journey, he became affected with increase of the dyspnœa, and pain of the upper and left side of the chest. He died exhausted after three days' distress.

Post-mortem Examination was performed in the country. The only lesion of importance was an aneurism of the arch of the aorta. The tumour was large and irregular; it occupied the left subclavian space, and extended upwards behind the clavicle, and in front of the great vessels of the neck. The vagus nerve of this side was involved. The trachea was compressed, and showed a mulberry-like bulging in its interior. The circumjacent tissues were infiltrated with a dense exudation. The sac was partially loculated, some of the loculi contained dense coagula, but the general cavity was chiefly empty; and it communicated with the aorta by a large irregular orifice in the anterior wall of the arch before it descends.

The imperfections in the report of the preceding case do not affect its bearing on the diagnostic value, and the variations of the cavernous change of the second sound. In the case which follows, the situation where the altered character of the second sound existed declared the presence of dilatation of the aorta, in addition to aneurism of the innominate artery.

CASE VI.—*Aneurism of the A. Innominata, with Dilatation of the Aorta; Cavernous Change of the Second Sound most marked over the Ascending Aorta.*

Mrs Scott, æt. 69, consulted me on the 22d Dec. 1849, on account of increasing swelling of the belly, and slight breathlessness. She had no cough nor distress otherwise; but she pointed my attention to a fulness at the lower part of her neck. Pulse was 72, of good strength.

Physical Signs.—The tumour occupied the sternal notch and the space above the right clavicle; it was diffused, of irregular form, and pulsating; its greatest length was two inches, transversely. Percussion was dull on the right side of the sternum as low as the second intercostal space, and two and a-half inches to the right. The upper margin of the præcordial dulness was on the third rib. The cardiac second sound was intensified, especially on the tumour and on the right margin of the sternum above where the second costal cartilage articulates, and had a hollow cavernous timbre. No irregularity of pulse, nor of respiration.

History and Progress.—Her disturbed health had existed for some months, and she had been relieved by laxative medicine. She could not tell when the tumour appeared; it had existed for years. I saw her occasionally for a year afterwards, when she left Edinburgh.

These cases exemplify the cavernous intensified second sound in a very instructive manner. In all of them the diagnosis was a matter of no difficulty, and on that account they are the better fitted for our present purpose. In Cases IV. and V., the altered timbre of the second sound had its greatest intensity over the aneurismal tumour; and in No. VI., the cavernous timbre of the second sound was appreciable over the aneurism of the innominate artery—a condition I have met with in other cases, as in No. IX. of this series—but from the greater distinctness of the sign in the second right intercostal space, where the aorta lies close to the surface, conjoined

as it was with extended dulness of percussion, I concluded that dilatation of the ascending aorta existed along with the aneurism. In such circumstances, I do not think the greater nearness to the aortic valves a satisfactory explanation of the increased loudness of the second sound, which became less intense at the base of the heart; its more cavernous character at the second intercostal space is to be explained rather by the fact of the dilated artery coming into more extended contact with the parieties in that situation. The aortic valves were adequate to their office in all of these cases; and the necessity of this practically affords a solution of the vexed question, whether the cavernous tone is a cardiac or aneurismal second sound. The practical solution of the question, as a clinical fact, appears to be, that this condition of the second sound cannot arise without the sonorous flap of the sigmoid valves, and the sonorous vibration or undulation, reaching the cavity of the aneurism, is multiplied and intensified, in the manner observed, in a distended elastic bag, connected with a valve by a continuous tube, even of considerable length. The important fact is, that we are to look for this altered state of the second sound over the aneurism, not over the sigmoid valves. This was conspicuous in Case IV., and was true in all; although in many cases, and possibly for a variety of reasons, the cavernous second sound may be heard as loud at the valves as at the seat of disease. Moreover, it is not incompatible for this cavernous tone to exist in conjunction with diastolic murmur from aortic regurgitation, as I shall take occasion to explain in noticing Case No. VIII.

This state of the second sound not unfrequently accompanies cases of hypertrophy of the heart. To distinguish such cases does at times involve real difficulty, which, however, may usually be surmounted by attention to the collateral signs, especially the extent of percussion dulness over the heart and aorta. At the same time, so far as I have observed, in cases of hypertrophy, the altered second sound does not acquire the cavernous timbre in its highest degree—it is more apt to assimilate to the normal flatness in the tone of the second sound, more or less intensified. Besides, in hypertrophy, as in peripheral aneurism of the ascending aorta, the cavernous sound exists at the seat of the valves; while it may, or may not, in cases of saccular aneurism.

Case No. IV., and others similar, impressed me strongly that a fluid state of the contents of the sac is necessary for the production of the cavernous timbre; but in several cases of the present series it was well marked, although the sacs were in part occupied by coagula. In the case on which Dr Henderson founded his original observation, we are told that the sac contained a "maze of clots." It appears, then, that entire fluidity of the contents is not essential to the production of the cavernous tone, and its occurrence in cases of hypertrophy of the heart, and its modifications in different aneurisms, confirm the opinion that the aneurismal and arterial

walls, as well as their contents and the force of the blood current, are concerned in the mechanism of the sound.

Of diastolic bellows murmur in aneurisms I have little to say; but the negative statement I have to make is not without value. In the course of a now lengthened experience I have not met with any well-marked instance of diastolic bellows sound originating in, and audible only over, a substernal aneurism. This is enough to prove its rarity; but still, its existence would afford conclusive evidence of the presence of a saccular aneurism. The two succeeding cases illustrate the difficulties in discriminating aneurismal diastolic murmurs. No. VII. is of interest in other respects, especially as regards the advanced effects of the disease in protracted cases. The narrative has been condensed from a minute and necessarily very lengthened record of the case.

CASE VII.—*Saccular Aneurism of the Arch of the Aorta, Dilatation of the Aorta; External Tumour, with Rupture; Diastolic Murmur; Cavernous Second Sound; Death from Exhaustion.*

Anne Stewart, æt. 53, hawker, was in hospital on various occasions under my care, between July 1850 and February 1853. An intemperate person, and bloated. Subject to dyspnœa and palpitation. A large pulsating, painful tumour occupied the upper portion of the sternum and contiguous parts.

Physical Signs.—These necessarily underwent variation during so long a period. The space dull on percussion over the tumour measured above five inches in diameter. Præcordial dull percussion was displaced downwards, and was extended. At the level of the fourth costal cartilages, besides the systolic impulse, there was perceptible a sharp impulse as of the flap of the sigmoid valves, at times accompanied by a thrill. The cardiac sounds varied from time to time. At the apex, in the sixth intercostal space, the first sound was muffled,—the second was occasionally accompanied by a diastolic bellows murmur. At the level of the third and fourth cartilages, the cardiac first sound always presented a muffled, murmurish character; and the second, a cavernous intensified timbre, audible only in a limited space, and increased by excitement. On the tumour there was perceptible to the ear only the shock of the impulse. Over the carotid arteries a hoarse systolic murmur existed.

History and Progress.—She had an attack of acute rheumatism about seven years previously, in the course of which she was cupped in the region of the heart. The tumour appeared not long after.

Gradual increase of the tumour took place, accompanied occasionally by acute pain locally, and interscapular aching. On several occasions, sudden increase occurred, with purple colouration of the tumour and acute tenderness.

In the end of 1850, slight dysphagia occurred, with deep-seated pain, and followed by impeded breathing and ringing cough for about three months.

In the beginning of 1853, with one of the sudden attacks, rupture of the tumour occurred, and slight hæmorrhage, which was easily restrained, and the wound cicatrized. Her head had become immovable without the assistance of her hands. Soon after, sudden and large expansion of the tumour occurred, with diffused purple coloration, and sloughing of the cicatrix. She had acute pain, with febrile symptoms, which were relieved by bleeding from the arm, and by leeches. She sank till 6th February, when she died.

Post-mortem Examination.—The external tumour occupied the lower part of the neck, and extended as low as the fifth rib; there was a slough on its surface. The aorta was dilated and calcareous. The orifice of the aneurism was about an inch in diameter at the arch. In the sac was a dense coagulum,

having a small cavity, and weighing 16 oz. The coagulum was partially separated at the orifice of the aneurism, and a thick, dark-coloured clot of blood lay between it and the sac-wall. Posteriorly a saccule existed, with dense surrounding tissue the size of a filbert, and in contact with the trachea. The upper two-thirds of the sternum were wanting. Both the clavicles rested on the coagulum, and had their periosteum separated from their sternal ends, with recently effused blood interposed, continuous with the soft clot that lined the sac. The heart was hypertrophied. No lesion of the valves. The vagus and recurrent nerves were free from the tumour. Both lungs were slightly emphysematous.

With reference to the want of agreement between the reported diastolic murmur in this case, and the apparently normal state of the sigmoid valves, I can offer no explanation, nor shall I argue as to its sigmoid or aneurismal seat. In evidence of the care taken to determine its nature, I quote the following notes from the record of the case, dated 8th January 1852 :—"At the apex cordis the second sound is converted into a prolonged bellows murmur. Over a spot on the sternum, on the level of the third intercostal spaces, and corresponding to the lower boundary of the morbid dulness of percussion, both heart sounds are free of murmur." Upwards, on the tumour, no bellows sound existed. On the 12th July 1850, it was noted,—"The murmur reported with the second sound at the apex is doubtful."

In the existing state of our knowledge, the supposition is more than justifiable that this diastolic murmur, appearing and disappearing as it did, originated in the aneurismal orifice; but if the fibrinous mass contained in the sac prevented it being heard on the tumour, it is difficult to explain how it could reach the apex cordis.

Further, the aneurismal diastolic murmur is described by authors as being peculiarly a faint murmur, and therefore liable to be overlooked. And there is reason to suspect that the murmur of aortic regurgitation in such cases as the following, might be confounded with it :—

CASE VIII.—*Saccular Aneurism of the Ascending Aorta; Second Cardiac Sound cavernous, with Bellows Murmur; Insufficiency of the Aortic Valves; Hypertrophy of the Heart.*

William Scott, æt. 53, stonemason, admitted into the Royal Infirmary under my care. A man of robust frame, but spare, and suffering from dyspnœa and palpitation.

History.—He had been under treatment for some weeks previously to admission, with relief to severe bronchitis. He had been unfit for work for a month, but had experienced occasional distress for twelve months.

Physical Signs.—Coverings of the chest spare, with præcordial fulness and diffused cardiac pulsation; that of the apex was perceptible in the fifth space to the left of the usual situation. In the second right intercostal space, close to the sternum, there was very slight visible pulsation, synchronous with the cardiac beat. There was permanent fulness of the external jugular vein, and visible irregular pulsation of the internal. Radial pulses equal, quick in action, not decidedly diastolic, visible. Præcordial space dull on percussion, extended vertically from the third left cartilage five inches downwards, and transversely from a line an inch left of the mesial line, four inches leftwards. There was

dull percussion on the upper part of the sternum and the contiguous parts of the third and fourth right intercostal spaces. Percussion also was dull under the right clavicle, and behind from the middle of the right interscapular space upwards.

The heart sounds at the apex cordis were free of murmur. The first was muffled; the second was intensified and hollow-toned. Over the ventricles, and at the base, the second sound acquired more of the developed hollow and metallic tone, but its termination was accompanied by a soft prolonged bellows murmur. This bellows murmur had its greatest intensity in the second right intercostal space close to the sternum; it was inaudible on the upper bone of the sternum. The cavernous intensified second sound, on the other hand, was well heard over the front, and was intense in a limited place on the sternum and contiguous second right intercostal space.

The murmur of respiration was distinct and pure in both subclavian regions. Behind, where percussion was dull, in the upper part of the right lung the expiratory murmur was prolonged and tubular. The altered second sound was also distinctly audible, though faint, and the thrill of the voice was increased.

He left the hospital and was lost sight of.

The conjunction of a well-marked second sound, with a *terminating* murmur of aortic regurgitation, is not unfrequent; and here it occurs in circumstances in which, if care were not taken to guard against the mistake, the murmur might appear to have its source away from the valves, and lead to the supposition of diastolic aneurismal murmur. This case was referred to at page 22, in illustration of the statement that a degree of insufficiency of the aortic valves may exist along with the cavernous intensified second sound in cases of aneurism.

The immediately preceding cases illustrate what is so commonly and so painfully witnessed in practice—that this disease has made hopeless progress before attention is fixed upon it. This was most impressively shown in our fourth case. The patient, a young woman, applied for the relief of symptoms apparently so trivial and disconnected with the idea of aneurism, that no examination of her chest was made for several weeks. At length, in consequence of neuralgia of the shoulder, and sense of discomfort on first lying down, her chest was examined, and the physical signs afforded unmistakable evidence of the large aneurismal tumour represented at page 19.

In contrast with this, and other cases of this series, especially No. VI., we see in No. III. the attention of the patient attracted by his local and other symptoms ten months before the physical signs of the disease were very decided. Perhaps the only considerations which cast any light on the probable cause of these differences are connected with the constitutional habit of the patients, and the situation of the tumour; although an invariable rule cannot be laid down, the former is worthy of attention. Patients, such as Dr Turnbull's (No. III.), of muscular and vigorous frame, and sound general health, will often be found to suffer sooner and more acutely. Again, in cases such as No. IV., the tumour, from the nature of the surrounding parts, may expand

to a much greater extent, and perhaps rapidly, without the same degree of suffering or injury to contiguous organs.

The more I see of this disease, the more I am led to attach a high value to pain as a symptom. General statements regarding the value of this or any other symptom of substernal aneurism are very difficult, and very apt to leave on the mind a felt sense of practical defect. Authors have given systematic statements of the character, seat, and connexions of pain as a symptom; but, probably, its worth will be better estimated by tracing it in the narratives of individual cases. We have already seen how valueless may be the most reliable sign of substernal aneurism, if it be isolated; and this is pre-eminently true of pain. Conjoined, however, with the evidence of an obstructed bronchus, or with circumscribed dulness of percussion, pain may afford all but conclusive evidence. Moreover, the frequent absence of pain proves nothing against its value as a symptom. No doubt, Dr Law,[1] in his valuable cases and observations, puts the matter too strongly when he says, pain is "a pathognomonic sign;" but, he says well and truly, that "it is important to know that it does sometimes exist when we most stand in need of it to guide us."

The diagnostic value of pain in these cases depends less upon its character and seat, than upon the circumstances in which it occurs. It has been mentioned at page 16 how important an influence the pains had in the diagnosis of case No. III., chiefly in consequence of the attendant dulness of percussion and the general condition of the patient. The pain in that case did possess a marked peculiarity in its history as well as in its seat. The length of time during which it had recurred, with long-continued and complete intermissions, and the sudden severity of its attacks latterly. His early and acute suffering seems to illustrate what some have insisted on,—the more painful character of saccular aneurisms of the ascending aorta. The completeness of the intermissions in the case was observed of the other symptoms as well as of the pain. After the result of the case, Dr Turnbull, writes:—"From first to last I have been much struck with the variableness of the symptoms." The patient also complained expressly of the twofold character of his pain, similar to what Dr Law dwells upon in connexion with aneurism of the abdominal aorta,—fixing itself in the seat of disease, and at the same time lancinating elsewhere.

The history of the symptoms in case No. IV. contrasted very strikingly with what has just been said of Case III. In the former, so far as the patient's statements can be trusted, she appears not to have suffered till the last four months; probably because not till then did the tumour attain a size which, by stretching, or

[1] Dublin Med. Journ., 1842. Vol. xxi.

otherwise affecting the phrenic nerve, could act upon the branches of the cervical plexus. In commenting on the next case, I shall draw attention to the correlation of substernal aneurisms and the phrenic nerve; but, connected with the pain and neuralgia, I wish to point to the excessive stretching and displacement of the left phrenic nerve in case No. IV.; and connected with that, the intense anguish in the seat of the tumour, which latterly interfered with all movement, and which was accompanied by severe neuralgia of the shoulder. This was conspicuous, also, in case No. V., where, there is reason to think, the left phrenic nerve was interfered with.

CASE IX.—*Bilocular Aneurism of A. Innominata; Compression of Trachea; Obstructed Expiration; Cavernous Second Sound; Vomiting.*

Feb. 24, 1853.—An artist, æt. 40, having the general appearance of good health, but a constrained position of the head, bent forwards. His breathing was noticed to be noisy, his voice hoarse, and he had a husky ringing cough. He complained of embarrassed breathing, acute darting pains in the right shoulder and right side of the neck, and occasional sudden vomiting of ingesta. There existed a diffused, firm, pulsating tumour at the lower part of the throat, slightly to the right side.

History.—The altered tone of voice and cough had existed for three months. His health had been uniformly good till neuralgia of both shoulders became distressing, eight months previously.

Physical Signs.—The tumour at the root of the neck occupied a space which was irregularly triangular, and had for its base the inner half of the right clavicle; its apex on the right side of the cricoid cartilage; and the swelling extended in front of the trachea. There was distention of the veins of the neck, and visible pulsation of the great vessels. The left radial pulse was small as compared with the right; but of the carotids, the right was the less distinct. Percussion was dull on the upper portion of the sternum and contiguous part of the right subclavian space. In the region of the heart, dulness commenced on the third rib. The sounds of the heart were natural, except in the vicinity of the sternal notch, where the second had a distant but decidedly cavernous intensified tone or timbre. Over the tumour there was perceived only a sound or sense of impulse. Heart sounds obscure. The action of the heart intermitted occasionally. The murmur of respiration was nowhere disturbed appreciably.

Subsequent Progress.—Little change occurred during the next ten days; the dyspnœa then became aggravated and more or less permanent, with painful paroxysmal attacks. He was slightly relieved by leeching. The constrained bowed position of the neck increased; he usually sat up stooping.

Cough, in violent paroxysms, became more frequent, with eight or twelve ounces of glairy mucous expectoration daily. The obstruction of respiration affected both expiration and inspiration more than previously. He experienced but slight and temporary relief from the remedies used, and sank rapidly in about three weeks.

Post-mortem Examination.—The general volume of flesh was considerable.

The tumour consisted of an aneurism of the arteria innominata. It was the size of an egg, and of irregular shape; elongated vertically, and divided in the same direction into two lobes by a deep sulcus, the larger being to the right and in front of the trachea; the smaller dipped deep to the left of the trachea, which thus was closely invested by the tumour. The par vagum and the recurrent nerves of the right side were free from the tumour. On the left side the par vagum anteriorly, the recurrent posteriorly, were in contact with the

left lobe of the tumour; some fibres of the par vagum being incorporated with the sac. The carotid artery, but in a greater degree the subclavian of this side, were pressed upon by the deep and firmer portions of the tumour.

The interior of the aorta showed extensive atheromatous deposit, and the arch was dilated chiefly by two shallow pouches. The orifice of the arteria innominata was irregular in form, but little enlarged. The aneurism had its origin in the anterior wall of the artery, leaving a free communication for the current from the aorta to the carotid and subclavian arteries. The sac was bilocular, corresponding to the right and left lobes of the tumour, as seen externally. The septum was complete except below, where its crescentic border overhung the orifice of the artery. The right or principal saccule was nearly filled by a dense lamellar and adherent clot. The saccule on the left was nearly empty; its cavity was irregular; and some small dense decolorized coagula existed in its sinuosities, giving hardness to the posterior part.

On laying open the trachea from behind, its rings were found bulging inwards from the pressure of the firm tumour; and several points of minute ulceration of this part of the mucous membrane existed.

The heart was healthy. The bronchial tubes contained much mucus, and the mucous membrane was injected.

In several respects this was a remarkable case. The stethoscopic phenomena confirmed the proofs already given, that in saccular aneurisms the cavernous intensified second sound is to be looked for over the sac, and not at the base of the heart.

I have not met with the description of any case in which the bilocular division of the aneurism was so complete; and this singular limitation of the aneurismal expansion to the anterior wall of the artery, leaving a free passage for the blood from the aorta to the carotid and subclavian arteries, would interfere with one of the most available means of distinguishing aneurism of the innominate artery from that of the aorta. I refer to the diminished pulsation of the tumour by compressing the carotid and subclavian arteries. This was not tried in the case; but I cannot doubt that in such an anterior situation of the sac, in a degree disconnected with the current from the aorta to the great vessels beyond, the pulsation of the sac would not have been diminished to the degree it would if the current of blood had passed more directly by the sac to the arteries.

This case is the only one I have met with in which frequent and sudden fits of vomiting, chiefly of ingesta, occurred. In connexion with this, as well as the neuralgia of the shoulders and neck, I regret that the relation of the phrenic nerves to the tumour has not been recorded. I had overlooked this in making the *post-mortem* examination, but from the situation of the lobes of the tumour on each side of the trachea, it is probable that both phrenic nerves were implicated. I make this statement with the view of directing attention to the relation of substernal aneurisms to the phrenic nerves. This case does not admit of any reasonings on the subject, but it justifies the suggestion that irritation of both phrenic nerves might result in spasmodic fits of vomiting. In cases No. IV. and No. V. we have the interference of the tumour with one of the phrenic nerves without vomiting; but, to the encroachment on that nerve,

the neuralgia of the left shoulder may be ascribed. I do not remember to have seen this correlation of the phrenic nerves with aneurisms referred to ; and it appears well worthy of the attention of those who have opportunities of carefully recording the clinical and *post-mortem* conditions of such cases.

The pressure of the trachea was very remarkable in this case, from the manner in which the aneurism had expanded itself round the front of the air tube. It was in consequence of this that the patient presented the very expressive stooping position of the neck, which is also referred to in case No. I. Dr Turnbull describes a similar attitude in his patient, Case III., in the following words, "he was seldom able to lie down in bed, and latterly he was easiest in an easy chair, leaning forwards with his elbows resting on his knees."

With reference to the performance of tracheotomy for the relief of obstructed breathing in cases of substernal aneurism, I have not met with any case in which it was demanded. It cannot be denied that in possible circumstances a few hours, possibly days, might be added to the existence of a patient; but this case, and many similar, in which there is an encroachment upon the calibre of the trachea, suggest a very weighty consideration in connexion with the question. Probably, in all such cases, the operation is inadmissible ; and in this case, as well as in No. I., I wish to point to the obstructed expiration which accompanied the difficult inspiration, and was permanent—not paroxysmal. Now, in cases of aneurism, and in some other diseases, this noisy and obstructed expiration is a simple and conclusive evidence of organic or physical encroachment on the calibre of the trachea, which, in cases of aneurism, is conclusive against the performance of tracheotomy. Of course, in laryngeal obstruction from spasm merely, in which inspiration alone is affected, the conclusion may be otherwise.

Copious expectoration of glairy mucus is not a usual accompaniment of substernal aneurism. I have met with it occasionally in connexion with pressure on the trachea.

The narratives of the two following cases present the disease in a form that is often met with; and yet such cases are not unlikely to mislead.

CASE X.—*Masked Saccular Aneurism of the Arch of the Aorta ; Chronic Pleuro-Pneumonia ; Death by Hæmorrhage into the Air Tubes.*

James Carr, æt. 40, admitted into hospital on the 24th January 1845 ; a delicate-looking man of spare habit of body ; he had a husky wheezing cough with hoarseness, and he complained of pain of the left side of the chest, with difficulty of resting on that side, owing to increase of cough. Pulse 85, soft. There was slight ulceration of the left tonsil.

History.—He had not suffered from any acute attack. The existing symptoms had been developed gradually during fourteen weeks. Pain did not distress him except from coughing. He admitted occasional sweating in sleep.

Physical Signs.—His chest was small. Percussion was dull throughout the left side, with respiratory silence; on forced inspiration faint wheezing sounds were audible. The vocal vibrations were suppressed; no effect from change of posture. The heart appeared to be slightly displaced downwards, and to the right; its sounds were normal, but unusually distinct in the left subclavian space.

Progress.—Little or no improvement occurred in the symptoms; with the exception of blisters to the left side of the chest, the treatment was palliative. On the 1st March, profuse fatal hæmorrhage from the air passages occurred suddenly.

Post-mortem Examination.—The volume of flesh was small. The left lung was adherent, except at the lower part, where above a pint of straw-coloured serum was contained in the pleura. This lung was contracted and dense throughout, with mingled lobular masses of grey and red, and large masses of black consolidation. The heart was normal, and externally the aorta appeared to be so.

The inner surface of the aorta presented some atheromatous deposit; and in the lesser curvature of the arch, just before it descends, there existed an angular aperture half an inch in diameter, communicating with the sac of an aneurism about the size of an egg. It occupied the space of the arch and compressed the left bronchus, in the interior of which there was the bulging tumour of the aneurism, with a dark, sloughy, ulcerated surface, which was perforated.

The left carotid artery and the A. innominata originated together from the aorta; the carotid separating immediately, the innominata crossed the trachea to the left, and divided into its branches.

CASE XI.—*Masked Aneurism of the Arch of the Aorta; Ulceration of Tonsil, etc.; Death after Gradual Exhaustion.*

18*th November* 1858.—A country labourer, æt. 40, a patient of Dr ———, who requested me to examine the case. He was pale, feeble, and emaciated; and he had a husky ringing cough, which, at times, occurred in paroxysms with expectoration of glairy mucus. The breathing was slightly embarrassed with noisy inspiration; expiration free. No pain, but distress of the chest, with sense of oppression.

Physical Signs.—The impulse of the heart was obscure, but when the patient leaned forward the apex was felt to beat in the sixth intercostal space. The cardiac dulness was extended; and circumscribed dulness of percussion existed on the sternum, at the level of second and third ribs. The dull space inclining to the left at its lower part, measured about one inch and a half in the vertical as well as the transverse direction. No impulse could be detected away from the heart. The murmur of respiration was absent throughout the left side of the chest, as also was the vocal fremitus. The radial pulses were of equal size.

History.—For six or eight months he had been liable to alarming paroxysms of dyspnœa, and to cough as described above. He had suffered from ulcerated sore throat.

The subsequent history of the case was that of progressive decline, and he died exhausted on the 2d January 1859. For long he had been free of the paroxysms of cough and dyspnœa, and he suffered comparatively little at the last.

The former of these cases was admitted into hospital, and treated by me as a case of chronic pleurisy; the latter was sent for my opinion by his medical attendant, who felt dissatisfied with the result of advice received from two practitioners, surgeons of acknowledged eminence, that the case being one of chronic laryngitis, was

to be treated by the use of caustic solution locally, and the administration of the iodide of mercury.

In Case X. the difficulty was created in a great measure by the dull percussion which pervaded the left side of the chest, and made it impossible to define the dull space over the tumour; and the husky cough had not the metallic clang. In Case XI., on the other hand, it was manifest from the cough, the dulness of percussion, the vocal and respiratory silence of the left side of the chest, and the displacement of the heart, that a tumour existed deep in the chest. It appeared doubtful whether the tumour was a new growth or an aneurism, and after his death his medical attendant wrote, if the case was one of aneurism, death must have resulted from pressure on the thoracic duct, as he was reduced to a perfect shadow. This is a most probable view of the case—the aneurism occupying a position where it could compress the left bronchus, the pneumogastric nerve, and the thoracic duct.

In such cases the chief practical difficulty will be solved, if it can be determined whether the respiratory obstruction is central or peripheral. In case No. X. this difficulty is exemplified:—Obstruction existed at the surface of the lung as well as at the root, and the evidence of pleuro-pneumonia, though negative, was conclusive. Strict attention, however, to the absence of vocal vibration throughout the left side of the chest would have disclosed the undiscovered central obstruction of the aneurism; because in pleural effusion the thrill of the voice is usually increased on the upper part of the side affected. In Case XI. the state of percussion-dulness made the case less difficult; but the interruption of respiratory sound and vocal thrill on the upper part of the chest was an important guide to the deep-seated disease.

The correlation of substernal aneurism and disease of the lungs and pleura involves interesting but most abstruse problems, which it is impossible to discuss here. I have not met with any case of thoracic aneurism in which tubercular deposit existed in the lungs. Chronic pleuro-pneumonia, or at least a densified contracted state of the lung, is a frequent accompaniment of aneurism, and in some cases it has a useful symptomatic value. The source of the lesion is matter of dispute; and there are great practical difficulties in the way of determining whether the altered state of the lung is the consequence of direct pressure, of obstruction of the nutrient vessels, or of interference with the pulmonic plexus of nerves. These remarks apply chiefly to cases such as No. X.:—in Case III. the state of matters is not strictly analogous. There, I believe, the pleuro-pneumonia, which occurred fifteen months before death, was the result of direct pressure; as indeed it may be in such cases as No. X. The condition of the lungs has not been frequently recorded in the earlier stages of aneurism; but in cases such as No. III., in which inflammation arises in a part of the lung contiguous to the supposed aneurism, and not usually affected primarily, the concur-

rent pulmonary lesion may be accepted as an indication of the aneurism.

The cases recorded in this paper afford illustration of various modes of death in substernal aneurisms. I refer only to the instances of hæmorrhage. And it is to be observed that death does not appear to result from the quantity of blood lost. For example, in cases such as No. X., the patient is destroyed by asphyxia, not by syncope. In Case IV., in which hæmorrhage occurred into the pleura, the patient survived the first attack of bleeding, and even rallied in a degree. In Case III., the suddenness of the fatal attack, with prostration, bloodlessness of the surface, and speedy dissolution—though the result was delayed with revived vital power, it is probable that death arose from the same cause; and in both cases the shock and prostration of the system were altogether disproportioned to the loss of blood.

It is not essential to the object and general intention of this paper to give any categorical statement of the results to which these cases or my experience otherwise have led me, as my purpose has not been to discuss, so much as to illustrate the subject. Those who do not possess opportunities of watching many cases, or of studying them with minuteness, will admit the practical advantage of this mode of illustrating the diagnosis of substernal aneurisms.

I conclude with the following statement of some of the considerations that arise out of these cases, and which were of value in their diagnosis:—

1. The all but invariable importance of dulness of percussion.
2. The great diagnostic value of a cavernous intensified character of the cardiac second sound;—heard over the seat of disease rather than at the sigmoid valves (Cases IV. to IX.).
3. The rarity and uncertainty of diastolic murmur as a sign of aneurism (Cases VII. and VIII.).
4. The importance of pain as a symptom; observing its neuralgic form and its conjoined fixed or local and lancinating character (Cases III., IV., and IX.).
5. The relation of pain of the shoulder to interference with the phrenic nerve (Cases IV. and IX.).
6. The occasional vomiting of ingesta as a consequence of irritation of both phrenic nerves (Case IX.).
7. The occasionally temporary character of laryngeal symptoms (Case VII.).
8. The diagnostic value of concurrent inflammation of the lungs (Cases III. and X.).
9. The import of difficult expiration, as indicating mechanical obstruction of the trachea (Cases I. and IX.).

PRINTED BY OLIVER AND BOYD, TWEEDDALE COURT, EDINBURGH.