mitral. Any *post-mortem* clots which may be adhering to the edge of the valve are removed with care, so as not to disturb the position of the cusps. An aperture sufficiently large to admit the brass nozzle is next made into the apex of the left ventricle in the line of incision which will afterwards be required to lay the ventricle open. The nozzle is then introduced, and air driven in by the bellows in the manner before described. The heart should meanwhile be suspended by the cut edges of the auricle. A series of hooks fixed on supports is useful for this purpose, the supports being movable so as to allow of the proper apposition of the cusps.

The slightest incompetency can be detected quite as much by the irregular manner in which the cusps mutually adjust themselves, as by the escape of air between them.

The same means of demonstration is to be followed in the case of the tricuspid, and, after its condition has been ascertained, the incision in the left ventricle is carried up as close as possible to the root of the aorta, without, of course, injuring the aortic ring. The heart is now suspended, as before, by the cut edges of the ventricle, and the nozzle is tied into the distal end of the aorta. The aorta should either be cut off at the termination of the ascending part of the arch, or be laid open up to this point. The nozzle both in the case of the mitral and in that of the aortic may be simply held in with the hand, or it may be tied in; or, what is even preferable, a simple clamp apparatus may be attached to its extremity, which seizes the cut edge of the vessel or cavity into which the nozzle is introduced, and holds it air-tight or nearly so. In watching the action of the valve, the ventricular aspect should be turned upwards, the air being driven in from below.

The pulmonary artery orifice is next tested in the same manner. In a perfectly healthy heart it will be found that the aortic, mitral, and pulmonary artery valves are quite competent, but, so far as my present experience goes, I have never met with a tricuspid in man which will stand the test. There is always a slight leakage, even when the cusps are perfectly free to move.

THE PROCESS OF COMPENSATION AND SOME OF ITS BEARINGS ON PROGNOSIS AND TREATMENT.

By BYROM BRAMWELL, M.D., F.R.C.P. EDIN.,

Assistant Physician to the Edinburgh Royal Infirmary; Lecturer on the Principles and Practice of Medicine in the Extra-Academical School of Medicine, Edinburgh, etc.

(Concluded from page 840.)

Compensation in Cardiac Lesions.—One of the most common and obvious methods of compensation is the increase (hypertrophy) of muscular tissue which takes place in muscular tubes and muscular organs when an increased effort is frequently and repeatedly demanded of them. The hypertrophy of the bladder, œsophagus, and intestine in cases of organic stricture are familiar examples; but there is, perhaps, no organ in which compensatory hypertrophy is more beautifully developed than in the heart. But, although hypertrophy of its muscular walls is by far the most important manner in which valvular lesions are compensated, it must also be remembered that in some forms of valvular disease a certain limited amount of dilatation and alterations in the frequency of the cardiac contractions also take part in the compensation, and assist the hypertrophy to restore and maintain the balance of the circulation. It may be stated broadly that in obstructive valvular lesions compensation is effected by muscular hypertrophy, aided, in some cases, by slowing of the cardiac action, while regurgitant lesions are compensated by hypertrophy, aided by a certain but strictly limited amount of dilatation, and it may be by increased frequency of the cardiac contractions. The manner in which the different valvular lesions are compensated is briefly as follows:—

In stenosis of the aortic orifice, compensation is chiefly effected by hypertrophy of the left ventricle, by means of which an increased quantity of blood is forced through the narrowed orifice at each ventricular contraction. The duration of the ventricular systole is at the same time lengthened, while the frequency of the cardiac contractions is diminished rather than increased, with the result that more time is afforded for the hypertrophied left ventricle to discharge its contents. In aortic stenosis, dilatation of the left ventricle affords no aid as a means of compensation, and when it does occur, it is a secondary and injurious result.

In stenosis of the mitral orifice, compensation is partly due to hypertrophy of the left auricle, but chiefly to hypertrophy of the right ventricle. Prolongation of the ventricular diastole, increased duration of the auricular systole, and slow action of the heart are also, when present, important factors in the compensation, since they facilitate the flow of an increased quantity of blood through the stenosed orifice during each cycle of the heart. At the best, however, the compensation in severe mitral stenosis is imperfect, much less perfect than it is in many cases of aortic stenosis, for even when fully developed the pulmonary circulation is subject to some strain. The most that the compensation can do in mitral stenosis is to enable the left ventricle to supply an adequate quantity of blood to the peripheral tissues, and to protect the organs and tissues which are situated behind the right heart from the effects of venous engorgement. In mitral stenosis, the pulmonary circulation is exposed to engorgement and all its secondary consequences, for the obstruction in front (that is, at the mitral orifice) is not suddenly relieved, as it is in mitral regurgitation, by the bursting open of the mitral valve and the suction action of the left ventricle. Even in those cases, then, in which the compensation is most perfect, the blood-vessels of the lung are exposed (in consequence of the obstruction in front and the powerful action of the right ventricle behind) to increased pressure and the injurious consequences which may result therefrom.

In stenosis of the pulmonary orifice, compensation is partly due to hypertrophy of the wall of the right ventricle, but chiefly in congenital cases (which comprise the great majority of cases of pulmonary stenosis) to the fact that the foramen ovale remains patent and allows a portion of the blood to avoid the lungs and to make a short cut, as it were, from the right to the left heart. The imperfect aëration of the blood which results from this arrangement is, of course, a disadvantage, and the compensation is, consequently, always imperfect; but if the foramen ovale did not remain open, the circulation could not be carried on with the extreme contraction of the pulmonary orifice which is present in some cases of this description, for the blood which could get through the contracted pulmonary orifice and lungs to the left heart would be inadequate to distend the arterial system, while the engorgement of the venous circulation behind the lesion would be too great to allow of the long continuance of life.

In incompetence of the aortic orifice, compensation is effected chiefly by hypertrophy of the left ventricle, partly by a certain but limited amount of dilatation of the left ventricle, and partly by shortening of the ventricular diastole, and, it may be, by increased frequency of the cardiac contractions. When the dilatation of the left ventricle is just sufficient to accommodate the blood which regurgitates through the aortic orifice, and, at the same time, to receive the normal quantity of blood from the left auricle, and, when the hypertrophy is sufficiently great to enable the cavity to empty itself at each systole, compensation is as perfect as circumstances will allow. The circulation in the parts behind the mitral valve is little, if at all, interfered with; and, provided that the arterial anæmia, due to regurgitation from the aorta during the ventricular diastole, is not great, there are few, if any, symptoms. But in free aortic regurgitation, compensation can never be perfect for any length of time, for a greater amount of dilatation than the theoretical minimum required for the perfect restoration of the balance of the circulation is sooner or later established; this necessitates a proportionate excess of hypertrophy if the cavity is to be completely and satisfactorily emptied, and this dilatation and hypertrophy tend to go on increasing. Again, the increased frequency of the cardiac contractions and the diminished duration of the ventricular diastole prevent the normal amount of blood flowing into the left ventricle from the left auricle, and consequently tend to cause some obstruction to the blood-flow behind the mitral valve and left heart. The sudden and excessive distension of the arterial system is also apt to produce dilatation in the aorta and the vessels arising from it.

In incompetence of the mitral orifice, compensation is effected in a still more complicated manner—namely, partly by dilatation and hypertrophy of the left auricle, partly by hypertrophy of the right ventricle, partly by dilatation and hypertrophy of the left ventricle, and perhaps to some slight extent by diminished duration of the ventricular diastole and increased frequency of the heart beats.

When the dilatation of the left auricle is just sufficient to accommodate the blood which regurgitates through the mitral orifice, and at the same time to receive the normal amount of

blood passing to it from the lungs; when the hypertrophy of the right ventricle and left auricle is sufficient to drive the normal amount of blood through the lungs and to empty the left auricle of the normal and abnormal (regurgitant) blood which it is just sufficiently dilated to contain; when the left ventricle is sufficiently dilated to receive the abnormally large quantity of blood from the left auricle, and sufficiently hypertrophied to expel all the blood (part of it passing forwards into the aorta in the normal course, and part passing back into the left auricle), the compensation is as satisfactory as it is possible to be under the circumstances; but in free regurgitation satisfactory compensation does not, as a rule, last long.

In mitral regurgitation, so long as the compensation is good, the engorgement of the pulmonary circulation is not so continuous as it is in mitral stenosis, for relief is afforded and pressure is removed with the occurrence of ventricular diastole and the bursting open of the mitral valve. But in free mitral regurgitation the theoretical minimum of dilatation of the left auricle required for satisfactory compensation is sooner or later exceeded; the lungs become engorged, and there is a tendency to dilatation both of the left and right ventricles, to venous engorgement in the parts behind the right heart, and to arterial anæmia in the parts in front of the left heart, and hence to the production of symptoms.

In *incompetence of the tricuspid orifice*, the compensation can never be satisfactory if the regurgitation is at all free. Compensation in this case entirely depends upon the condition of the right auricle. If the cavity of the right auricle could become just sufficiently dilated to accommodate the blood which regurgitates from the right ventricle and at the same time to receive the normal amount of blood passing into it from the *venæ cavae*, and if its wall could become sufficiently hypertrophied to expel all this blood into the right ventricle, the compensation would be as perfect as it could be under the circumstances. As a matter of fact, too much dilatation and too little hypertrophy are almost always the result; and even in the earlier stages of anything like free tricuspid regurgitation, engorgement of the peripheral venous circulation takes place.

The relative order of gravity of the different cardiac valvular lesions is a question which has given rise to a good deal of debate, though authorities are now pretty well agreed, except perhaps as regards the relative gravity of aortic regurgitation and mitral stenosis. I shall be glad to hear the opinion of members on this point. For my own part, I believe, for the reasons advanced above, that free aortic incompetence is, as a general rule, more progressive and more rapidly fatal than advanced mitral stenosis. It seems to me that in free aortic regurgitation the lesion must of necessity get the upper hand, and the compensation prove inadequate before very long, but that there is no such necessity in advanced stenosis, provided that the patient leads a quiet, vegetative life. The following is a case in point.

Some few years after entering practice I examined *post mortem* the body of a woman, aged 56, who had been under my father's observation with advanced mitral disease for more than thirty years. For the greater part of this long period she had been a helpless cripple from left-sided hemiplegia, the result of embolic plugging of the right middle cerebral artery, and had lived a vegetative life. The mitral valve was so much contracted that it would not admit of the tip of the little finger; and, still more remarkable, considering the extreme mitral stenosis and the comparative absence of all lung symptoms, the left lung was, to a large extent, useless, being compressed into small size by a very much dilated left auricle, and bound by adhesions to the upper and back part of the chest.

Now I very much doubt if a relatively great aortic incompetence could have been compensated and tolerated (even under the same circumstances) for such a length of time. I am prepared, therefore, to allow that a free and progressive aortic regurgitation of necessity tends to a more rapid breakdown of the compensation than an advanced degree of mitral stenosis, even when all the other conditions for compensation are as favourable as could be wished. I believe this is the general rule when the lesion is a serious one, but there are certainly exceptions to it—cases, on the one hand, in which free aortic incompetence is tolerated for a long time, and, on the other, cases of mitral stenosis in which compensation fails much more quickly than is usually the case; but when the lesion is slight in degree or stationary, aortic incompetence is I think in many cases—and this is perhaps the general rule—less serious than mitral stenosis. Slight aortic incompe-

tence causes, in my experience, less disturbance of the circulation, less discomfort to the patient, is compatible with a more active and often also with a longer life, than the same (relative) amount of mitral stenosis. The "accidental risk" of aortic incompetence (death from syncope) is also, I think, less in such (relatively slight and not progressive) cases, than the "accidental risk" of mitral stenosis, namely, embolism.

From this description it will be apparent that hypertrophy of the muscular wall of some one or other of the cardiac cavities is the chief means by which valvular lesions are compensated. When, therefore, an organic valvular lesion is established, and more especially when that lesion is a serious one, or when, though slight at first, it is progressive, the prognosis depends largely on the probability of a sufficient and satisfactory amount of muscular hypertrophy being produced.

It must, of course, be remembered that in forming a forecast or prognosis in cases of cardiac valvular disease, many other considerations—besides the capability of a sufficient amount of muscular hypertrophy being produced—have to be taken into account. In fact, the more important points which enable us to determine the prognosis are as follows: (1) the form of valvular disease and the risk of its causing sudden death; (2) the severity of the lesion; (3) the stationary or progressive character of the lesion; (4) the capability of compensation in the heart itself and the power of resistance possessed by the peripheral tissues; (5) the occupation, circumstances, and surroundings of the patient, and the possibility or probability of his carrying out the treatment which is recommended; (6) the mental temperament of the patient; (7) the presence or absence of complications, more especially the presence or absence of disease of the kidney.

Time does not permit me to enter into details with regard to all these matters; nor, indeed, do I desire to do so, for I have already considered most of them elsewhere.⁵

But I may perhaps be allowed to say that for the production and maintenance of a sufficient and satisfactory muscular hypertrophy, the following conditions are necessary:—

1. The muscular tissue must be healthy, or at all events sufficiently healthy to become hypertrophied. When the wall of the heart is in a condition of fatty degeneration or fibroid disease the hypertrophy is never very satisfactory.

2. The cardiac muscle must receive a sufficient supply of healthy arterial blood.

3. The waste products of its combustion must be sufficiently quickly removed.

4. Its trophic nerve apparatus must be healthy. In short, the heart muscle must possess the conditions which are essential for rapid development and healthy growth.

The age of the patient, the etiology of the case, and the facility with which the cardiac muscle responds to cardiac tonics (digitalis and strophanthus), together with the general condition of the patient and the state of the other tissues and organs, are the chief means by which we endeavour to form an opinion as to the condition of the cardiac muscle.

In young persons the muscular tissue is, as a rule, healthier, and the capabilities of repair and of the production of compensatory hypertrophy are generally greater than in older people. When, too, the valvular lesion has resulted from acute endocarditis, the capabilities of repair are, speaking generally, greater than when the lesion is due to atheroma or senile or degenerative changes. There are, however, many exceptions to this general statement. The endocarditis may have been complicated with pericarditis, or with myocarditis, and the valvular lesion may therefore be associated with a damaged cardiac muscle, a condition which may interfere with the production of satisfactory hypertrophy, and materially add to the gravity of the case. It must not, indeed, be supposed that in cases of cardiac valvular disease all the advantages are on the side of youth. In old people who lead quiet vegetative lives there is little strain put upon the damaged heart; and for this reason, if other things were at all equal, the valvular lesion would be more likely to remain stationary, and the call for repeated and renewed compensation would certainly be less than in children, youths, and active adults, who are with difficulty kept quiet, and who, notwithstanding all the advice which we can give them, are only too apt to over-exert themselves and to put an injurious strain upon the damaged heart.

In trying to ascertain whether a sufficient amount of arterial

⁵ See my *Diseases of the Heart*. Sections devoted to the general pathology of the heart and the prognosis in valvular lesions. Also my *Practical Medicine and Medical Diagnosis*. Sections devoted to the prognosis.

blood is supplied to the cardiac muscle, and whether the products of its waste are satisfactorily removed, we observe the condition of the peripheral, arterial, and venous circulations.

A badly filled radial pulse or atheroma of the superficial vessels is suggestive that the coronary arteries are also badly filled or diseased, and therefore that the supply of arterial blood to the muscular wall of the heart is deficient. Angina-like pain in the region of the heart is also suggestive of disease of the coronary arteries, and of fibroid or fatty changes in the wall of the heart.

A dilated right heart and engorgement of the systemic veins suggest that the veins of the heart are also engorged, and that the removal of waste products from the cardiac muscle is defective.

As yet we have no satisfactory means of ascertaining the condition of the trophic nerve apparatus connected with the heart. Indeed the exact nature of this mechanism is not as yet thoroughly understood, though Gaskell's brilliant researches seem to show that the vagus exerts some sort of stimulating and trophic influence upon the cardiac muscle. But be that as it may, the course of cardiac valvular lesions (and perhaps, too, the amount of muscular hypertrophy and compensation which are developed) is largely determined by the condition of the nervous system, and by the mental temperament of the patient. In no affections are the beneficial results of a healthy nervous system, and of a serene, happy, placid disposition more manifest than in cardiac valvular lesions.

The power of resistance possessed by the peripheral tissues and organs to the injurious effects of cardiac valvular lesions depends, as in the case of the heart itself, upon (1) the condition, healthy or otherwise, of the peripheral tissues; and (2) the fact whether the conditions for their nutrition are satisfactory or not.

Here, as in the case of the heart muscle itself, the tissue must receive an adequate supply of healthy arterial blood; the waste products of its metabolism must be satisfactorily removed, and its trophic condition must be satisfactorily regulated by the nervous system.

It is quite obvious, therefore, that the presence or absence of complications, more especially of diseased conditions of the stomach and other organs which manufacture the nutrient fluid; of the kidneys, liver, and intestines and other organs, which purify it; and of the nervous system, which exerts such an important influence upon all the processes of nutrition and resistance are most important factors in enabling the peripheral tissues to resist the injurious effects of valvular lesions, and to protect themselves (in some cases by the production of an increased amount of connective tissue) against the injurious effects of venous engorgement. And, *vice versa*, that the presence of disease in any of these organs adds most materially to the gravity of the prognosis.

The important bearing which these facts have in the treatment of cardiac valvular lesions is obvious. It is now an acknowledged axiom in cardiac therapeutics that the muscular hypertrophy, which was at one time thought to be injurious, and, therefore, a condition to be attacked and removed by treatment, is the best thing which can result under the circumstances, and is, therefore, to be encouraged. Our therapeutic efforts are now directed to sustaining the vitality of the cardiac muscle. Our main object is to reduce and remove the lesion on which the cardiac hypertrophy depends, but, so long as the lesion persists, to sustain the hypertrophy, which is Nature's chief means of compensating it. The more I see of cardiac valvular lesions, the more I am convinced that digitalis, strophanthus, and other remedies of a similar kind, are much too freely and too frequently used in the earlier stages, and that the indiscriminate use of these remedies is often most injurious. It is only when compensation fails, either in the earlier stages, as the result of some temporary overbalance of the compensation, such as is so frequently occasioned in mitral cases by an intercurrent attack of bronchitis, or, in the later stages, when the period has at length arrived at which Nature's reserve is exhausted, when the lesion has gained the upper hand, when the cardiac muscular fibre is undergoing degenerative changes, and when dilatation is succeeding to hypertrophy, that digitalis or other remedies are called for. So long as the natural compensation is sufficient to meet the lesion, our chief efforts should be directed, *first*, to removing all sources of cardiac strain and excitement. *Secondly*, to maintaining the general health in the best possible state of efficiency, by (a) careful regulation of the diet, exercise, rest, sleep, whole mode of life, and general hygienic condition of the patient; (b) by attention to the condition of the

blood-forming and blood-depurating organs, more especially of the stomach, liver, intestine, kidneys, and skin; (c) by the administration of such general tonics as arsenic, quinine, and strychnine; and (d) by avoiding all causes of nervous exhaustion, and by promoting, as far as possible, a tranquil, happy, and healthful state of mind. And here I must again emphasise the all-important influence which the mental condition has upon all the processes of nutrition and repair, and the value of what I term mental therapeutics—the influence produced on the mind of the patient by a judicious and hopeful medical adviser, by the avoidance of all worry and anxiety, and by cheerful and pleasant surroundings. *Thirdly*, to avoid all conditions which are likely to aggravate the lesion or to produce those complications and accidents to which the disease predisposes and renders the patient liable.

The Importance of forming a Correct Estimate of the "Reserve," for the Purposes of Prognosis.—In forming an opinion as to the chances of recovery, both in acute and chronic affections, it is of the utmost importance to endeavour to estimate correctly the amount of reserve on which the patient can fall back, and the power of compensation which he possesses. In order that this estimate may be correct, it is necessary (1) to be acquainted with the mental temperament of the patient; for this, as I have elsewhere tried to show, exercises a most important influence on the chances of recovery, more especially in cases of acute disease (*Practical Medicine and Medical Diagnosis*, page 141); and (2) to gauge correctly the condition, not only of the part which is primarily affected, but of all the tissues and organs. In illustration of the importance of the latter point, I need only instance the extreme gravity of acute croupous pneumonia in the presence of a weak or fatty heart, and of typhus or typhoid fever when the kidneys are diseased. The advantage which a knowledge of these particulars gives the ordinary medical attendant over the consultant is so obvious that I need hardly refer to it.

Compensatory Protection.—Closely allied to these processes, which we truly term compensatory, by which Nature endeavours to counteract or minimise the effects of lesions such as I have already described, there are many other methods by means of which the causes of disease are got rid of, or the injurious effects of disease prevented. The vomiting and diarrhoea which are due to the introduction of irritants into the stomach or intestine; the contraction of the peripheral blood-vessels which results from exposure to external cold; the board-like rigidity of the abdominal muscles which is present in perityphlitis; and many reflex nerve alternations, such, for example, as the beautiful reflex process by which the peripheral blood-vessels are relaxed when too great strain is suddenly thrown on the left ventricle of the heart, may be mentioned as illustrations.

Probably the process of suppuration, as Mr. Bland Sutton has so ingeniously theorised, is also a protective and compensatory condition, the pus corpuscles endeavouring to take up, devour as it were, and carry off the irritative particles on which the inflammatory condition depends.

Whether the pyrexia which so frequently follows the introduction of noxious materials—more especially particulate organic poisons—into the blood has any beneficial and compensatory action, or whether it is wholly injurious, we do not at present know.

The adhesive inflammation which prevents the production of pneumothorax in cases of phthisis and the perforation and peritonitis in some cases of ulcer of the stomach, and the obliterative process which seals up the blood-vessels and prevents the occurrence of hæmoptysis in cases of phthisis are also beautiful examples of Nature's protective processes.

The Value of the Local Application of Cold in the Treatment of Hæmoptysis; and the Treatment of Acute Perforative Peritonitis.—And here I would raise two practical questions: First, what, in the opinion of the members of the Branch, is the value of the local application of cold to the exterior of the body in the treatment of hæmoptysis, and indeed in the treatment of localised internal inflammations such as acute pericarditis or acute peritonitis?

Although personally I have until comparatively recently been in the habit of recommending the application of an ice-bag to the surface of the chest, at a point superficial to what appears to be the source of the bleeding in cases of hæmoptysis, I have never felt thoroughly satisfied as to the practical value of the treatment, and for some time past I have had grave doubts as to the correctness of the theory on which it is based. It is supposed

that the local application of cold produces, by means of reflex nerve action, contraction of the blood vessels of the internal organ, which is situated beneath the part of the surface to which the cold is applied. But the experiments of Dr. Milne Murray, and the practical experience both of surgeons and gynaecologists have conclusively shown that the application of very hot water is a much more powerful means of arresting hæmorrhage and of producing marked and sustained contraction of the blood-vessels than the local application of cold water. The question naturally arises whether, instead of applying an ice-bag, we should not rather apply a bag containing very hot water over the part of the surface which is superficial to an inflamed or bleeding part. I have hopes that Dr. Milne Murray will clear up this very important practical question by an additional series of experiments; but pending such direct evidence I shall be glad to elicit the opinion and to learn the practice of the members of the Branch on this point.

Secondly, I would ask has any gentleman present seen a case of recovery from peritonitis due to the perforation of an ulcer of the stomach; and what practice is it advisable to adopt in the treatment of this most serious accident?

Personally I have never seen a case of recovery, though cases do without doubt occur. For my own part, I am disposed to think (since by means of transfusion we are able to temporarily restore the patient, and so greatly minimise the danger of death from shock during or soon after the operation) that the proper plan of treatment is first to transfuse the patient, and then to open the abdomen, wash out the peritoneum, and stitch up the opening in the stomach. I am not of course oblivious of the gravity of this procedure and of the difficulty, perhaps the impossibility, that there would often be of carrying it out in private practice; but I shall be glad to hear the opinion of the surgeons on the point.

Euphoria.—Whether the feeling of well-being and hopefulness which is present in some affections, notably in many cases of phthisis, has any special or beneficial effect on this disease, and why this peculiar condition of mind is associated with some affections, and present in some cases and not in others, it is perhaps impossible to say. That the opposite condition of mental depression, which is often associated with derangement of the functions of the liver, has a distinctly prejudicial effect, no one, I presume, will deny. The causation and significance of the remarkable hopefulness which is present in some cases of advanced organic disease requires, I think, more attention than that which it has hitherto received. One of the most remarkable cases of the kind which has come under my own observation occurred in the person of a medical friend, aged, I think, 56. While in charge of an important appointment in the Indian Medical Service he began to fail in health. He was seen by several medical men, and a small hard tumour about the size of an egg was discovered in the epigastric region. The patient was of an extremely nervous and anxious disposition, and since the exact nature of the tumour was

not very obvious, it was not thought advisable to inform him of its presence. He was therefore advised to come home on sick leave; this he accordingly did. He arrived in Edinburgh some two months after the tumour was first noticed, under the firm belief that there was nothing of any importance the matter with him, and that his great weakness and extreme emaciation were entirely due to the effects of very small doses of morphia, which he had been in the habit of taking on the voyage to relieve the uneasiness (not actual pain) which he felt in the abdomen. Both the patient and his wife, a most intelligent, sensible, and sober-minded lady, were fully persuaded that there was no serious organic disease, and that the patient had only to come to Edinburgh to get thoroughly restored to health. I saw the patient a few

hours after his arrival, and my wonder was that he had been able to complete his journey. Profoundly cachectic-looking, emaciated to the last degree, so weak that he was unable to walk across the room, with an unhealthy sloughing ulceration of a few days' duration on the cornea, it was obvious, without any further examination, that he was dying from some serious organic disease. On uncovering the abdomen the cause was at once apparent, for a large tumour, fully the size of a child's head, of great hardness, and evidently malignant, occupied the upper and central part of the cavity. The new growth appeared to be (chiefly, at all events) an enlargement of the abdominal glands; but as no *post-mortem* examination was made, the exact connections of the tumour and the organs which were involved were not ascertained. Both the patient and his wife seemed amazed when the presence of the tumour was pointed out to them. How it could possibly have escaped the attention of the patient, a most cultured and intelligent medical man, I am utterly unable to conceive, for it was impossible to place the hand on the abdomen without at once recognising the large tumour and its dense, hard, character. Nothing, of course, in the way of an operation was possible, though Dr. Keith was kind enough to see the patient with me, and to corroborate my opinion on this point. The patient died, I think, on the second day after I saw him first.

Advanced Organic Disease without Symptoms.—The manner in which the most serious disease is tolerated, and, indeed, in some cases borne with-

out any apparent suffering or distress, is sometimes very remarkable. Why there should be this extraordinary tolerance in some cases and not in others, it is difficult to say. One of the most remarkable cases of the kind which has come under my own personal observation was a case of aneurysm of the thoracic aorta. All physicians and pathologists are, of course, familiar with the fact that sudden death not infrequently results from the rupture of a thoracic aneurysm whose presence was unsuspected during life. In most cases of this kind the aneurysm is of small size, usually deep seated, often arising from the first part (root) of the aorta; and seldom so situated as to have compressed such important structures as the œsophagus and trachea.

Fig. 8.—The heart and thoracic aorta in the case of J. G., from the front, showing an enormous aneurysm of the ascending and transverse portions of the aortic arch (copied from a photograph of the dried preparation). The size of the preparation is 10½ inches vertically and 7½ inches transversely. The figure measures 4½ inches vertically and 3½ inches transversely. The letter A points to the descending thoracic aorta; B to the innominate artery, from which a piece of cotton-wool is projecting; C to the left common carotid, and D to the left subclavian arteries; E to the trachea; F to the œsophagus; G to the aneurysm; H to the heart; and P to the pulmonary artery.

The case which I am about to relate is a remarkable exception to these statements. It is probably unique when the enormous size of the tumour is considered (it is, I think, the largest entirely intra-thoracic aneurysmal sac which has come under my own notice), and when it is remembered that there is an entire absence of symptoms, the patient having regularly followed his employment without ever making complaint of illness until an accident to the eye caused him to seek admission to the infirmary, where the aneurysm was discovered almost by accident the very day before the fatal rupture occurred. Further, it is important to note that not only was the main tumour of such great size, but that a small secondary sac (which cannot be seen in the figures) sprang from the primary tumour, and was in direct contact with the trachea. It is remarkable that, so far as I have been able to ascertain, the patient did not complain of shortness of breath until the night before he died, for the small secondary aneurysmal sac was not only in contact with, but had evidently pressed upon and flattened the trachea. The notes of the case are as follows:—

Enormous aneurysm of the ascending and transverse portions of the aortic arch, entire absence of symptoms, death from rupture into the left pleural cavity.

J. G., aged 58, was admitted to the Edinburgh Royal Infirmary under the care of Dr. Argyll Robertson on March 11th, 1885, suffering from an injury to the right eye. The patient stated that on March 9th a piece of wood struck his eye with great force. Upon admission the lens was found in the anterior chamber; it had already caused a considerable amount of irritation.

On March 12th, Dr. Robertson (to whom I am indebted for this clinical note) removed the lens with the scoop. On the evening of March 14th, the patient complained of difficulty of breathing, and when examined by the clinical clerk, Mr. Pettie, he was sitting up and labouring under considerable dyspnoea. In the course of a little time, and after the administration of an antispasmodic, the difficulty of breathing subsided. Mr. Pettie then examined the chest, and found extensive dullness and very marked heaving pulsation over the whole of the upper part of the front of the thorax, more especially on the left side. The following day, March 15th, the patient suddenly died, before any further examination of the chest had been made.

The post-mortem examination was made by me on March 18th, 1885. The body was well nourished, and the patient had evidently been a very muscular man. The capacity of the thorax was great in proportion to his height, the length of the body being 66 inches, and the circumference of the thorax 41 inches.

The left pleural cavity was filled with blood, for the most part clotted, which had escaped from a huge aneurysmal sac, which occupied a large part of the thoracic cavity (see Figs. 8 and 9). The aneurysm involved the terminations of the ascending and the whole of the transverse portion of the arch of the aorta. It was globular in form, and measured transversely (from a point a little

above the letter P, to a point a little below the letter G. in Fig. 8) 21 inches; and in circumference, the tape being passed round the distended and dried sac between the left subclavian artery (D) and the trachea (E, in Fig. 8) 16 inches. Without any exaggeration, the distended dried sac is larger than a child's head.

A small secondary sac, about the size of a walnut, sprang from the upper and posterior surface of the large tumour, and pressed directly upon the trachea. This pressure had evidently been considerable, for the trachea was distinctly compressed and flattened. The aneurysm contained a large quantity of laminated clot; the sac had ruptured just above the root of the left lung, and a very large quantity of blood had escaped through the wide laceration into the sac of the left pleura. The aneurysm was in contact behind with the trachea, oesophagus, and spinal column; there had apparently been little if any pressure on the spinal column, for there was no erosion of the vertebræ. The left lung was extensively adherent to the exterior of the tumour; and the left recurrent and vagus nerves were incorporated in the anterior wall of the sac.

The sac wall was remarkably friable, and on microscopic examination the structures composing it were found to be markedly fatty. The heart was of small size; it was not weighed, for this would have necessitated detaching it from the aneurysmal sac. The exterior of the heart was covered with a moderately thick layer of fat; the muscular tissue of the organ was in an advanced condition of fatty degeneration; its valves were healthy.

The question naturally arises whether this fatty condition of the heart and the low arterial tension which we would expect in such a condition may not to some extent account for the absence of symptoms in this remarkable case. The descending thoracic aorta and the great vessels arising from the sac were dilated. The lungs were deeply pigmented, but otherwise healthy. Permission was restricted to the cavity of the thorax; the other organs were consequently not examined.

The history of the case seemed so remarkable that I wrote to Messrs. Tancred, Arrol and Co., in whose employment the patient was; and also to Dr. James Hunter, the medical officer of the Forth Bridge Works.

I append the answers which they kindly sent me. It is, perhaps, right to mention that the men employed at the Forth Bridge Works can have the benefit of Dr. Hunter's advice and attendance simply by asking for it. It is, therefore, very unlikely that this man could have been ill without Dr. Hunter hearing of it.

Copy of Letter from Messrs. Tancred, Arrol and Co.

"Forth Bridge Works, South Queensferry, N.B., June 16th, 1885. Dear Sir,—Referring to yours of yesterday, we have the pleasure in giving you the following information. The man you refer to worked here under the name of J. G. for about two years, first as a fitter, but latterly in looking after the planing machine, neither of which is heavy or severe work. So far as we can make

Fig. 9.—The heart and thoracic aorta in the case of J. G., from the back, showing an enormous aneurysm of the ascending and transverse portions of the aortic arch (copied from a photograph of the dried preparation). The size of the preparation is vertically 10½ inches and transversely 7½ inches. The figure measures 4½ inches vertically and 3½ inches transversely. The letter A points to the descending thoracic aorta; B to the superior vena cava; C to the left common carotid artery; D to the trachea; E to the oesophagus; and H to the heart. The heart, aneurysm, arteries, trachea, and oesophagus were first filled with cotton wool; the preparation was then dried, varnished, and photographed.

out he was not under medical care here, nor did he complain of illness at any time. We trust the above information will suit your purpose, and are, yours faithfully, TANCRED, ARROL AND CO.
—Dr. Byrom Bramwell."

Copy of Letter from Dr. James Hunter.

"The Priory, Queensferry, December 29th, 1887. Dear Dr. Bramwell,—J. G. was a member of the Forth Bridge Workmen's Club, in virtue of which he was entitled to medical advice at any time. Up to the date on which he met with the accident to his eye I had not been asked to see him professionally. Indeed, I am not aware that he was on any occasion absent from his duties at the bridge.—Yours sincerely, JAMES HUNTER."

The details of another case of aneurysm (which is reported in the *Edinburgh Medical Journal* for April, 1888) were also given, in which the patient was able to work for a period of eight years with an aneurysm projecting through the front wall of the thorax.

CASE OF SERIOUS HÆMORRHAGE FROM THE CAVITY OF THE TYMPANUM IN AN INFANT (OTITIS MEDIA HÆMORRHAGICA).

By THOMAS BARR, M.D.,

Surgeon to the Glasgow Ear Hospital; Lecturer on Aural Surgery, Anderson's College, etc.

BLEEDING from the ear may occur in various affections of that organ. Thus it is not uncommon in purulent diseases associated with polypi, granulation tissue, or caries; in these conditions, however, it is rarely more than sufficient to tinge the discharge. A moderate escape of blood is also not unusual in otitis externa and in myringitis, from the rupture of hæmorrhagic elevations, either on the osseous portion of the external canal or on the tympanic membrane. In otitis media hæmorrhagica the source of the hæmorrhage, as the name implies, is the middle ear, especially the cavity of the tympanum, the mucous membrane of which is in a state of acute inflammation. The walls of the distended and engorged vessels give way, blood is poured into the cavity of the tympanum, the tympanic membrane is ruptured, and hæmorrhage takes place externally. The following case is probably unique in respect of (1) the tender age of the patient, and (2) the serious loss of blood. A baby (girl) of 9½ months was sent to me on November 17th last by Dr. Sinclair Kennedy, of Perth. I was informed that, five weeks previously, the baby had become restless, uneasy, and sleepless, apparently from severe pain. These symptoms continued for about a week, and then seemed to be explained by the appearance of muco-purulent secretion from the left ear. There was also noticed a considerable swelling in the neck below the left ear. This escape of matter was on the evening of Friday, and on Sunday night the mother awoke, and was dismayed to find the upper part of the child's dress saturated with blood; she was of opinion that at least a cupful of blood must have escaped. Dr. Kennedy says, in a letter of November 15th, "I was sent for at 5 A.M. on Monday, when I found the child deluged with blood from the left ear. I used a weak injection of acetate of lead, and enjoined rest. The bleeding returned once or twice during the day, but not to so great an extent. I had Dr. Bramwell seeing her with me in the afternoon, and we tried to see the state of the tympanum, but she was so restless we left her alone. We thought she must have had some inflammatory mischief of the middle ear, with rupture of the membrane. Under the ear there was a swelling as large as a walnut, and tender to touch, with a brawny feeling around. She had no pain over the mastoid process, but did not like to be touched over the head of the lower jaw-bone. She was put on iodide of potash, with liq. hydrarg. perchlor. and tinct. of iodine. Iodoform was put into the ear on cotton-wool, and iodoform and vaseline applied to the swelling under the jaw. Some hours after the first bleeding a troublesome dry cough came on, without any chest symptoms, and so we concluded it was aural. The bleeding again returned on Tuesday morning, and I put her on gallic acid internally. From this time there was no return of the bleeding. In a few days the swelling under the jaw went completely away. The discharge from the ear by Thursday, the 21st, was colourless and odourless. On her recovery I tried to examine the ear, but she was restless with me. However, I felt satisfied. I saw a ragged tear of the under half of the tympanum."

When I examined the patient, there was neither discharge from the ear nor perforation of the tympanic membrane; but a small cicatrix on the antero-inferior part of the membrane indicated the recent existence of a perforation. The membrane had not the ordinary lustre, and the vessels of the manubrium were somewhat injected. The Eustachian tube was permeable. I had no doubt that a perforation had recently existed in the tympanic membrane, and that the hæmorrhage had its source in the middle ear. The child was decidedly anæmic, but, according to the statement of the parents, the pallor was much less than it had been soon after the bleeding, when the face was quite blanched.

In regard to the explanation of the hæmorrhage in a case such as this, it has been argued that the dilated and engorged arterioles in the tympanic mucous membrane give way under the influence of the partial or complete vacuum produced in the tympanic cavity from the closure of the Eustachian tube. It is urged that just as rarefaction of the air in the tympanum may so act upon the blood-vessels of the mucous membrane of the middle ear as to produce a serous exudation (*hydrops ex vacuo*), so when acute inflammation exists with, as a consequence, dilated and engorged arterioles on the surface of the mucous membrane, the suction may in time lead, not simply to transudation of serum through the coats of the vessels, but to their rupture, with free hæmorrhage into the cavities of the middle ear. In this case, however, there was probably perforation of the tympanic membrane two days previous to the hæmorrhage, which would remove the vacuum, unless, indeed, which is quite conceivable, the aperture in the membrane was afterwards closed temporarily by inspissated secretion, so as again to lead to partial rarefaction of the air. But while admitting the possibility of the vacuum theory, it must be pointed out that such conditions frequently exist—in every case of otitis media, a very common affection—without hæmorrhage; and therefore it is probable that in cases attended by hæmorrhage there is, in addition, some peculiar weakness of the walls of the vessels of the middle ear. Such weakness may be the result of morbid changes due to Bright's disease, and several cases are on record of hæmorrhage from the mucous membrane of the tympanum associated with, and probably the result of, chronic Bright's disease, corresponding, as has been suggested, with the retinal hæmorrhage of the same disease. In this child there was no evidence of the existence of Bright's disease, and, indeed, the age was quite opposed to the supposition of changes in the arterial coats from chronic Bright's disease. Apart, however, from Bright's disease or any other chronic affection, there was probably in this child a hæmorrhagic tendency which predisposed to the hæmorrhage. Just as ophthalmic surgeons recognise a form of retinal apoplexy in the young, due simply to a hæmorrhagic tendency, so may such a case as this (tympanic apoplexy) have its origin mainly in the same predisposition. The immediately determining cause may have been the action upon the dilated and engorged vessels of the partial vacuum in the cavity of the tympanum. In view of the predisposition to the hæmorrhage which probably existed in this child, the parents were instructed to use every precaution in order to avoid, if possible, future inflammatory attacks in the ear.

The following authors may be consulted by those interested in the literature of this subject:—

Dr. Roosa's *Practical Treatise on Diseases of the Ear*; Politzer on the Membrana Tympani (p. 95), translated by Drs. Mathewson and Newton; Schwartze in a paper in *Archiv für Ohrenheilkunde*, entitled "Ein Fall von Bluterguss in die Paukenhöhle bei Morbus Brightii." Bd. iv; Dr. McBride on Otitis Hæmorrhagica in *Archives of Otolaryngology*, vol. xv.

LUMBAR COLOTOMY; THE CAUSES OF FAILURE IN FINDING THE COLON, AND HOW THEY MAY BE OBTIATED.

By HERBERT WM. ALLINGHAM, JUN., F.R.C.S.,

Surgeon to the Great Northern Hospital; Demonstrator of Anatomy at St. George's Hospital.

MANY surgeons commence the operation of lumbar colotomy under the impression that it may be impossible to find the colon, and almost all of us have seen the best operators experience difficulties and even failure in finding the gut. Cases, too, have been reported in which the small intestine has been opened by mistake. Knowing this, and having read Mr. C. B. Lockwood's interesting