the patients left the infirmary much better, but the cure was never complete, for in some of them the sac ruptured weeks or months afterwards. Apart from the pain which the subcutaneous injection of gelatine causes there is another risk that has to be borne in mind. Tetanus followed gelatine injections in one of the London hospitals, and as it is extremely difficult to sterilize gelatine, this risk, although no doubt remote, must be borne in mind. I have had a limited experience of galvanopuncture; I have seen one case of subclavian aneurysm thus treated and in which for several months the result was satisfactory until, after friction caused by the workman carrying his spade on the affected shoulder, the aneurysm redeveloped and burst into the surrounding tissues, causing a haematoma which began to ooze through the skin, but in which the blood subsequently coagulated, and the aneurysm was finally removed. One of the difficulties of treating thoracic aneurysm is in getting the subjects of the disease to be patient under treatment. Rest in bed and the restricted diet in time become irksome to the patients, who, once they feel rather better, long for freedom. If, when the pulsation of the tumour has lessened and the sac has become smaller and harder, patients can be got still to carry out the rest cure, the results are, as might be expected, more satisfactory.

REFERENCE.

1 BRITISH MEDICAL JOURNAL, 1898, vol. i, p. 1. The Diagnostic Significance of Laryngeal Abductor Paralysis, Sir Felix Semon.

A Lecture

ON

AORTIC DISEASE.

DELIVERED AT THE LONDON SCHOOL OF CLINICAL
MEDICINE.

By GUTHRIE RANKIN, M.D., F.R.C.P. EDIN.,
M.R.C.P. LOND.,

PHYSICIAN TO THE DREADNOUGHT AND ROYAL WATERLOO HOSPITALS.

FROM the point of view of etiology, this hospital furnishes an instructive object lesson in cardiac disorders. Its wards are never without cases of aortic disease; whereas mitral lesions, except such as are secondary, are seldom found. As you know, our patients are all seafaring men, who, whether as seamen or firemen, are, by the nature of their work, exposed to the wear and tear of an arduous life which involves irregularity of habits, both as regards food and sleep, hard physical work, and exposure to all varieties of weather and climate. Toilers on the deep are also liable to other dangers which militate strongly against the best interests of their health. They are not more intemperate than other classes of men—perhaps less so than many—for, in the nature of their occupation, they are perforce compelled, during the continuance of each voyage, to abstain from alcohol in every form. As a consequence, when they finally get into port, they are apt to rush to the other extreme, and, in the first gladness of being once more ashore, too frequently squander most of the wages they have so hardly earned by over-indulgence in alcoholic drinks which, as supplied to them, are potent and impure. The publican knows from experience that his sea-going customers like their drinks strong, and, by various methods of adulteration and faking, he succeeds in catering to their tastes. The sailor, therefore, who indulges in alcohol at all—and most of them do—not only takes it from time to time in excessive quantity, but he has also to accept it of villainous quality. The sailor, again, is no exception to the common rule that, under the influence of alcohol, he readily succumbs to the seductive attractions of Eve. His necessary continence throughout a voyage encourages a sudden lapse from virtue when the circumstances become propitious, and the Legislature is so little concerned for the welfare of those who go down to the sea in ships and on whom the commerce of the country so largely depends that it takes no trouble to supervise and regulate this traffic. In every seaport prostitution of the most degraded type is rampant, and of those who give way to temptation few escape without the penalty of venereal disease. It cannot be wondered that, under such loose regulations, syphilis is prevalent among seamen, and unfortunately its

early manifestations are usually regarded so nonchalantly that, in most instances, the disease has reached its secondary or tertiary manifestations before its victims come under treatment. Climatic exposure, strenuous toil, alcoholic excesses, and venereal infections combine to undermine the sailor's health and predispose him especially to arterial disease and the many serious consequences thereof. Therefore it is that in a hospital such as this we find unusual opportunities for studying not only all varieties of venereal and alcoholic disease, but also the many secondary disorders for which one, or other, or both are primarily responsible.

It is remarkable that acute rheumatism is not frequently met with among seafaring men. One would expect that the constant exposure to cold and wet which is their almost daily experience would cause rheumatic fever to be a frequent ailment among them; but it is not. Chronic rheumatism is common enough, but the acute manifestations of the disease are comparatively rare. Sailors themselves tell us that they never catch cold or rheumatism from salt water; and though it is difficult to see why this should be, it is at least offered as an explanation of their immunity, and rather fortifies the opinion that acute rheumatism is a different disease entirely from chronic rheumatism, and that its proper position in nosology is among the infective diseases. This immunity from rheumatic fever is further testified by the fact I have already referred to, of the absence from these wards of mitral disease. Rheumatic endocarditis may involve any or all the valves of the heart; but we know that the mitral valves are specially susceptible to its influence, and in severe cases seldom escape some degree of impairment.

Aortic disease, which is the subject with which we are specially concerned to-day, may be a sequela of acute rheumatism or it may be the result of degenerative changes in the arteries or heart. I am able to show you a case of each variety, because, fortuitously, one of the patients before you has suffered recently from rheumatic fever, to which event he refers, and probably rightly, the commencement of the cardiac disturbance which has brought him here. The other man is an example of that form of aortic disease with which we are daily made familiar in this hospital. Time does not enable me to enter minutely into their histories, but it will suffice for our purpose that I should tell you that these two patients resemble one another in their habits and occupation. Both have indulged themselves bountifully in alcohol, both have had syphilis, and both have been exposed to the rigours and vicissitudes of a seafaring life. The main differences between them are that the one man is younger and has suffered from an acute rheumatic attack which lasted over several weeks and involved many of his joints; the other is older by twelve years and, so far as he remembers, has never been seriously ill, though now he complains of stiffness and pains in some of his larger joints, which he attributes to rheumatism. When you come to examine their physical condition, you will find that both patients have a double aortic murmur, and present the usual signs and symptoms by which we are accustomed to recognize this condition. I would specially draw your attention, as you look at the men, to the tortuous and locomotive arteries, the pulsation in the suprasternal notch, the heaving cardiac impulse extending beyond the nipple line, and the absence of cyanosis or dyspnoea. These facts tell us a great deal, because we may infer from them that the vessels are thickened, that the aorta is dilated, that the heart is hypertrophied, and that compensation is well maintained. On palpation the impulse is extensive and vigorous, and you will observe in this patient that two of the intercostal spaces between the nipple line and the sternum are depressed with each cardiac systole. This is usually ascribed to pericardial adhesions, and it is not impossible that such may be the cause in this instance, but it should not be forgotten that where we have as great hypertrophy as exists here, the effect of atmospheric pressure in filling up the space created by systolic contraction of the enlarged ventricle is enough to account for thoracic recession. Percussion shows an increased area of dullness in both cases, which extends, as the apex thrust tells us, mostly down and to the left. When we come to examine the pulse it is found to possess, in both men, the water-hammer characteristics which belong to aortic regurgi-

tation; it is also noticeable that the wave of blood through the radial artery is delayed, so that if a finger of the free hand is placed over the carotid artery, whose pulsations synchronize with those of the heart, it will be found that an appreciable interval occurs between the pulse-wave in the carotid and that in the radial. The length of this interval is in direct ratio to the amount of incompetence, and in these patients it is demonstrable that the delay is greater in the one than the other.

The explanation of the collapsing character of the pulse is the loss of the support of the aortic valves; the greater, therefore, the destruction of the valves, the more sudden is the pulse-wave and the more complete the collapse of the vessel walls after it. To appreciate fully the distinctive features of Corrigan's pulse, the hand should be raised above the level of the heart to allow the ordinary laws of gravity to come into play. The delay in the pulse is due to loss of tone in the arterial walls and to the emptiness of the vessels between the cardiac pulsations. The visible pulsation of all the large vessels, but especially of the carotids, is always a more or less marked feature. In the case of one of our patients it was such a prominent symptom when he first presented himself in the out-patient room that he was suspected of having an aneurysm. Another interesting symptom often observed in cases of aortic regurgitation and well marked in one of these men is capillary pulsation. It is caused by the extension of the arterial relaxation into the capillaries, and is occasionally transmitted through them to the veins. It is best brought out by drawing a line across the skin so as to produce a patch of hyperaemia, the margin of which reddens and pales with each beat of the heart; or it may often be well seen through a microscopic slide, applied against the mucous lining of the lower lip. This capillary pulsation is not pathognomonic of aortic incompetence, but is also met with in other conditions where there is great relaxation of the peripheral arteries. Having learned this much from inspection, palpation, and percussion, we are now in a position to confirm the suspicions which these processes of examination have raised by the crucial test of auscultation. The two chests are not quite alike, but in both the prominent auscultatory fact is a double murmur, which is heard with greatest intensity over the aortic area, and is conveyed downwards along the sternum and upwards into the great vessels of the neck. But when this double murmur is investigated carefully it will be found to present certain important features in the one patient which are absent in the other. In the patient who has suffered from rheumatic fever, and whose lesion therefore falls into the endocarditic group of aortic disease, you will observe that no distinct second sound can be heard over the aortic cartilage; that the first sound at the apex is peculiarly loud and determinate; that it is succeeded by a short, rough, systolic murmur, which is not conveyed any distance into the axilla; and that the double bruit can be distinctly heard in the vessels of the neck. In his neighbour, who is older and presents evidences throughout his tissues of senile changes, the lesion belongs to the arterio-sclerotic type of aortic disease, and in him you will hear a second sound over the aortic cartilage which is loud and ringing; the bruits cannot be traced any distance along the cervical vessels, and the regularity of the cardiac rhythm is occasionally interrupted by a short beat. In both cases the double murmur is doubtless due to changes in the valves, which, in the one instance, are for the most part inflammatory, and in the other degenerative.

If we consider first the inflammatory condition the following points of interest may be noted. In all probability the stenosis is due to actual crumpling of the valve segments and the amount of regurgitation must be considerable. We come to these conclusions because of the absence of the second aortic sound and because of the conveyance of the murmurs into the vessels of the neck. Confirmation of the last fact is found in the marked delay of the radial pulse, in the pronounced collapse of the pulse after each cardiac systole, in the extensive pulsation of the arteries, and in the large extent of the ventricular hypertrophy. The sounds heard at the apex are interesting because when the patient first came under observation he was reported to have a presystolic mitral murmur in addition to the double aortic condition. Now, this presystolic mitral murmur is, as you all know, not uncommon in aortic disease, but in many instances where it has been

markedly present and where opportunity has been afforded for *post-mortem* examination, the mitral valve has been found normal. Attention was first called to it by Austin Flint, and to the present day the murmur is constantly called by his name. The most likely explanation of its production ascribes it to the impact on the anterior mitral segment of the regurgitant stream from the aortic orifice preventing its complete retraction and so interfering with the free flow of blood from the left auricle. But a difficulty arises in this case from the fact of the rheumatic antecedents, because during his attack of acute rheumatism the mitral may have been involved as well as the aortic valve, and, therefore, we may have to deal here with a presystolic murmur which is the direct result of organic changes with consequent true stenosis at the mitral orifice. This view is supported by the short systolic murmur heard at the apex which possibly results from slight regurgitation through the mitral orifice such as must inevitably accompany an actual stenosis. What leads to some hesitancy in accepting such a theory is the loud and rasping character of the bruit, and the absence of any convection into the axillary region. It is not impossible that the murmur heard in this case at the apex is the aortic murmur which is so plainly in evidence at the second right costal cartilage. If the mitral valve is structurally altered, we are at least able to assure ourselves that, so far, compensation is well provided for because there is an entire absence of any signs of backward pressure.

In the case of the older patient, the accentuation of the second aortic sound is to be regarded as evidence of dilatation of the aorta and increased arterial tension, and proves that, despite some regurgitation, the valves are still sufficiently preserved to produce a check to the column of blood in the aorta. In order to bring home more emphatically the value of the second sound in aortic disease, I have had two other patients temporarily removed to this ward that you may compare the sounds at the aortic cartilage of the one with the other.

This patient, since a fever of some kind ten years ago, has never been quite well. He has suffered from breathlessness and palpitation on exertion, with occasional attacks of pain in the chest and slight oedema of the ankles. The physical signs to which I wish specially to direct your attention are the situation of his apex-beat in the sixth space well within the nipple line; the heaving nature of the cardiac impulse; the slowness of the pulse, which is not more than 60; the contradictory smallness of the pulse when viewed in the light of the ventricular hypertrophy; and the presence at the aortic cartilage of a thrill and rough systolic murmur which is conveyed into the vessels of the neck, but is not followed by any definite second sound; indeed, the second sound is replaced by a soft, short, and faint diastolic bruit.

Compare this condition with that presented by our other patient, who has come to hospital on account of a bronchial attack. He is 64 years of age, has marked atheromatous changes in his vessels, and some emphysema. On inspection you will find no evidence of extreme hypertrophy. This may be due partly to the overlapping of the cardiac area by emphysematous lungs, but the apex-beat which can be felt in the fifth space, is diffuse and is not of the thrusting, forcible type we associate with a concentric ventricular hypertrophy. The pulse is of distinctive high tension and numbers 76. On auscultation over the aortic area a systolic murmur very similar to that heard in the other patient is heard and is conveyed into the vessels of the neck and across the manubrium, but here it is followed by a loud ringing second sound; the second pulmonary sound is also excessive, but it is not nearly so loud as its aortic neighbour. In all probability the murmur in this case is due to dilatation accompanied by loss of elasticity in the aorta itself and is independent of any change in the valve segments. A mistake we are all liable to make is to associate every aortic systolic murmur with a picture of stenosis of the orifice, but stenosis in the sense of organic change in the valve curtains is a comparatively rare affection, whereas the relative stenosis that depends upon dilatation of the aortic arch is very common in patients like this man whose vessels are becoming atheromatous, and in whom the aortic arch has undergone some degree of degenerative change. It should be remembered that a murmur in this situation may also be produced by

anaemia or by roughness of the valves without any actual narrowing of the orifice. From examination of these cases we are able, therefore, to make ourselves familiar with most of the characteristic features of aortic disease, and by comparison of one with the other we can bring home to ourselves the value of the second aortic sound as a guide both to diagnosis and prognosis. The existence of a systolic, or of a double systolic and diastolic, murmur is an easy matter to establish, but it is of considerable importance to read into these murmurs their full significance and importance.

Of the two cases in which there is regurgitation, the prognosis is most favourable in that which comes to us with a history of rheumatic fever. Should the patient be fortunate enough to escape future attacks of acute rheumatism, he may continue well over many years, and be capable of a considerable amount of hard and active work. So long as his compensation is fully maintained, he may keep fairly well even until he reaches advanced age. Then the degenerative processes of senility will tend to bring about sclerotic changes in the damaged valves and fatty alterations in the hypertrophied ventricle. In the case of the other patient, the position of affairs is quite different; degeneration has already set in, and is the cause of his lesion. By care he may get along with a fair degree of comfort, for a longer or shorter time, but his disorder is progressive, and, though further developments may be slow, they are sure, and must inevitably be in the downhill direction. The two cases, on the other hand, which present a murmur indicative of obstruction only at the aortic orifice, must be regarded prognostically in a reverse sequence. The life of the man with true stenosis is in greater jeopardy than that of the other, whose stenosis is relative. This would not be the case if we had to deal only with the existence of the murmur; in that event the prognosis, just as in the regurgitant cases, would be most favourable for him whose lesion was presumably inflammatory. But our opinion becomes modified at once when we refer to his history and find that recently he has, on several occasions, been confined to bed on account of pain in the chest, shortness of breath, and oedema of the ankles. In these three symptoms we have indubitable evidence of threatened failure of compensation, and when this begins we have no guarantee of permanent safety in the future.

Whatever the variety of aortic disease, there is no reason for serious anxiety so long as the lesions remain mute. The danger in all of them is of a twofold kind: failure of compensation, with all the consequent risks of backward pressure, and the spread of endarteritis so as to implicate the orifices of the coronary arteries. The compensatory hypertrophy which Nature provides to the walls of the heart in order that they may overtake valvular defects and difficulties may break down, either under the influence of increasing pressure in front, or from degenerative changes in the walls themselves, in consequence of which the vigour of the driving power behind is diminished. In addition, there is the added risk in aortic, as distinguished from mitral, cases, of anginal complications. No case of aortic disease occurring after middle life is free from the possibility of sudden death, which is, in most instances, due to angina pectoris. It is much more likely to happen in regurgitation than in stenosis, and in degenerative than in inflammatory conditions, but it ought never to be lost sight of as a factor to be considered when summing up the prognosis.

It is said that true aortic stenosis is the least dangerous of all valvular affections, but so many cases are called stenosis on no better evidence than the existence of a systolic murmur at the aortic cartilage that the generalization is probably too sweeping. At any rate, it is certain that if the risk of sudden death is less in stenosis than in regurgitation, the hopes of improvement after compensatory failure are also smaller, because, since the obstruction at the aortic orifice remains undiminished, and there is no augmentative help to be got further back than the left ventricle, its chance of recovering when once it has broken down is comparatively slight. The first evidence of failure of compensation in aortic disease is often said to be the establishment of a systolic murmur indicative of regurgitation through the mitral orifice, but I think we ought not to lose sight of certain earlier warnings which are sometimes given. The occurrence, as in one of the patients before us, of shortness of breath, pains in the

chest, and palpitations should always lead us to make a physical examination of the chest. If the patient be one whom we know, by previous examination, to be suffering from aortic disease, the subjective symptoms of which he complains will probably have light thrown upon their significance before the mitral valve has become leaky, by some alteration in the quality of the murmur, by some irregularity or intermission of the cardiac rhythm, or by some change in the second sound.

A loud and well-sustained murmur may always be regarded as expressive of a powerfully acting heart, and as indicative of comparative safety, but when it changes its character and becomes weak and short, it proclaims a note of danger. Similarly, regularity of rhythm, when supplanted by a type of cardiac action in which the systolic contractions are of unequal duration, indicates a change from normal to one of abnormal functioning, for which, in such cases, there may be a serious explanation. Finally, where a second aortic sound has been known to exist, its disappearance, or a diminution in its intensity, is to be regarded generally as a harbinger of evil. If a combination of these symptoms and signs be appreciated and their significance understood, rest and proper care immediately adopted and sedulously followed may often avert the further stages of broken compensation. This, as you will remember, passes through the successive phases of left-ventricle dilatation, regurgitation through the mitral orifice, pulmonary congestion, dilatation of the right ventricle, and regurgitation through the tricuspid orifice, to dyspnoea, cyanosis, anasarca, and death. It has been frequently stated that regurgitation through the mitral orifice should be regarded, in aortic disease, as an advantage rather than otherwise, because of the relief afforded to the over-distended left ventricle, but it must be obvious that if a ventricle, previously able to overtake the disabilities of aortic disease unaided, finds itself compelled to relieve the pressure of its work by such relaxation as permits of leakage backwards through the mitral orifice into the left auricle, the true meaning is diminution in expulsive power, and in that case the regurgitation marks a downward step.

As regards angina, which is the other great danger of these cases, I would guard against two errors: first, against confusing the true with the false variety; and, second, against estimating what is apparently the pseudo variety too lightly. Whenever anginal attacks occur with physical evidence of aortic disease, there is *prima facie* evidence that the angina is true, and therefore to be regarded with grave concern. But where the circumstances are such as to make us feel confident that there is no more serious cause for the attack than a functional storm, many of us are apt to conclude that, despite the alarm they create in the patients' minds and among their friends, there is really no cause for serious misgiving. This is a dangerous conviction, as I had occasion recently to see exemplified. A lady, at her menopause, 48 years of age, healthy and well to look at, active and vigorous, with no physical signs of disease anywhere except a moderately accentuated second aortic sound, had suffered at varying intervals over three years from anginal attacks. They were not severe in character, but quite distinctive. She was warned to be careful in the avoidance of fatigue and excitement, but was assured that she had no heart disease, and that when she was a little older and had reached the end of her menstrual life the attacks would probably disappear. One day, after walking quickly for a short distance out of doors immediately after luncheon, she developed the usual feeling of tightness in her chest which always ushered in one of her attacks. She got quickly home and went to bed, where under the influence of poultices and nitro-glycerine she seemed to be improving, as she had often done before. Suddenly and without warning she experienced an acute accession of precordial pain, became rapidly and deeply cyanosed, and almost immediately expired. *Post-mortem* examination revealed a perfectly healthy state of all her organs, except the aorta, where there were a few scattered patches of recent atheroma. These, however, were too insignificant to account for such sudden death, and further investigation showed that both coronary arteries were contracted at the orifices, and that one was throughout its whole length abnormally small, probably from a congenital defect. It is well, therefore, to bear in mind that all attacks of an anginal type are best spoken of reservedly,

lest, as in this case, the outcome proves the cause to have been, though not organic disease of the valves or vessels, something more definite and dangerous than a mere nerve-storm.

In the treatment of aortic disease the primary object to achieve is the establishment and maintenance of sufficient compensation. In the case of our first patient, who suffered recently from rheumatic fever, the chances are that his compensation never became firmly established—that is to say, that the length of time over which he rested and the amount of care which was taken of him otherwise after his acute illness was not enough to build up his myocardium against the strain of his subsequent life. In addition to this, he has been careless of himself and has indulged in alcohol freely and unwisely. Consequently he comes to us with all the evidences of broken compensation; fortunately for him, it has not gone beyond moderate pulmonary congestion. Already since his admission the improvement in his condition is marked; the mitral murmurs which he then had have almost disappeared; the cardiac rhythm is regular; the locomotive pulsation in the vessels has diminished, and there is a distinct though feeble second aortic sound to be heard in the carotid arteries. There is now no cough and no physical signs in the lungs; the liver dullness has become normal; he is taking food well, and is sleeping comfortably in a normal position the night through. This amelioration has taken place under the influence of complete rest in bed, a simple and moderate diet, regulation of the bowels by means of salines and an occasional small dose of calomel. At first he took a mixture of sodium salicylate, digitalis, strychnine, and ammonium carbonate; this was followed later on by another, which he is still taking, containing dilute phosphoric acid, iron perchloride, nux vomica, and strophanthus.

The only point worthy of comment in the medicinal treatment of a case of this type is the employment of digitalis. The old tradition that digitalis is a dangerous drug in aortic disease clings to many of us so pertinaciously that we hesitate to avail ourselves of its valuable assistance. This is not, I venture to think, good practice. The true action of digitalis is that of a tonic, the best of all we possess, to the myocardium. When compensation is well maintained, its exhibition is never indicated, but whenever this fails, it should be called to the rescue, irrespective of the nature or situation of the valvular lesion. Not only so, but at the outset of its administration the drug may be given advantageously in bolder doses than are customarily employed. Fifteen minims of the tincture every four hours may be ordered with perfect safety, if we are careful to watch its effects and curtail the quantity when we get evidence of saturation. That evidence is forthcoming in an abnormally slow cardiac rhythm, diminished urinary secretion, and disturbance of the digestive organs. In many instances the good effects of the drug are evidenced only after these physiological effects are produced. No harm ever results if, at this stage, the drug is either temporarily withheld or its quantity diminished to 7 or 10 minims every eight hours. Strophanthus and convallaria are recommended as valuable alternatives which are better suited to aortic conditions; but though they are useful remedies, their good effect upon the heart falls far short of digitalis, and it is by no means uncommon to find a speedy improvement under the influence of digitalis, when the others have failed to bring about the least response.

Another point about the administration of digitalis which is not sufficiently recognized is the safety that may be secured by its combination with nitro-glycerine. In all cases where vascular tension is high the 1 per cent. solution of nitro-glycerine given along with digitalis prevents increase of peripheral resistance, and so robs the digitalis of that influence upon the arterioles on account of which its administration is supposed to be contraindicated.

In the case of our second patient, who has likewise improved decidedly since his admission to hospital, the treatment has been somewhat different. His chief complaint, when first brought here, was of pain in the chest on exertion; shortness of breath, which prevented his lying down in bed at night; and recurring anginal attacks of a severe type. This is a combination of symptoms with which we are very familiar here, and which is enough of itself to suggest aortic disease. You have satisfied your-

selves, I hope, that his case is one belonging to the degenerative type of this ailment, but, in addition to the knowledge you have gained of his present condition from the physical examination you have made, I must remind you that, when first seen, his pulse was one of very high tension. Here we found a leading indication for treatment, and, in accordance with our usual plan in cases of this description, he was at once put on a mixture of potassium iodide and nitro-glycerine. Both these drugs, and especially the latter, are valuable vaso-dilators, and, under their influence, this man rapidly experienced relief to his breathing and diminution in his precordial discomfort. He was kept closely confined to bed, his diet was simple and mostly sloppy, and every night for a week he was given one grain of calomel, followed in the morning by a moderate dose of ordinary white mixture. After ten days he was so much better that we were able to put him on iodide of iron, strychnine, and strophanthus, and to allow him a greater amount of freedom in regard both to diet and exertion. He has had no recurrence of his anginal attacks since a few days after his admission; he is now able to sleep comfortably in the ordinary recumbent position, and a moderate amount of walking exercise can be indulged in daily without producing either cardiac discomfort or undue physical fatigue. As regards the dosage of the iodide and nitro-glycerine in cases of this description, I am persuaded we often err in making the quantities too small—15 gr. of sodium iodide, protected by 2 minims of Fowler's solution, and combined with 2 minims of the 1 per cent. solution of nitro-glycerine may with perfect safety be given every four hours. Whenever the pulse becomes soft and begins to quicken, longer intervals should be allowed between the doses, and eventually the nitro-glycerine may be wholly withdrawn and the iodide continued in smaller amount, say 5 gr., along with strychnine and one or other of the milder preparations of iron. In regard to calomel, its value is not confined to its aperient effect; it is a useful intestinal antiseptic as well, and also assists in diminishing the tension in the arteries. Obviously the nature of these patients' occupation is inimical to the maintenance of their circulatory balance when they return to work. We always urge them to find a new field for their labours, in which exposure, muscular strain, and dietetic irregularity will enter less into their daily experience, but it is difficult for most men after middle life to embark upon a new calling, and many of our cases ultimately succumb to the circumstances of an environment which is unusually trying and hard even for those who are in the possession of full vigour and healthy organs.

A Clinical Lecture

UPON

THE SURGICAL ASPECTS OF HAEMOPHILIA,

WITH ESPECIAL REFERENCE TO

TWO CASES OF VOLKMANN'S CONTRACTURE
RESULTING FROM THIS DISEASE.

BY

ERNEST W. HEY GROVES, M.D., M.S., F.R.C.S.,

ASSISTANT SURGEON, BRISTOL GENERAL HOSPITAL.

I WISH to show you three patients to-day who illustrate very well some of the common surgical symptoms of haemophilia, and belong to two families which exhibit in a remarkable way the peculiar hereditary characteristics of this disease.

W. W. is a well-grown, intelligent lad, who has had almost every variety of bleeding. But first let me speak of his family, and for those to whom a tabular representation appeals more than a description I give a diagram of their genealogical tree (Table I).

This boy's great-grandmother had one daughter and one son. The son was a "bruiser and bleeder," and died of bleeding from the extraction of a tooth; the daughter, who was well and healthy, had six children. One son, who is now 36, has six healthy children; one son died at 17 from epistaxis, one son died at 6 from the bleeding of a finger crushed in a door; one daughter (Mrs. C.), is married, and has two children, a daughter of 3 who is normal, and a son of 5 who is a bleeder. The second