weeks or even months of careful observation to find this optimum, and much perseverance will generally be required. In this respect help may frequently be obtained from the patient himself, who is often able to say what quantity suits him best; in other words, his own sensations are often a good guide. Having discovered this dose, in the great majority of cases it will be necessary for the patient to continue taking the drug for the remainder of his life. Apparently the cumulative action is not nearly of such serious account as has been supposed. Still the pulse ought to be watched day by day. By this mode of treatment you will save many lives and also bring comparative comfort to people who would otherwise be the subjects of much misery. When auricular fibrillation is not accompanied by a rapid pulse, no marked result follows the administration of digitalis; the result is scarcely greater than in cases without auricular fibrillation. The improvement is slow, and it is often difficult to be certain how far the improvement may be due to the rest in bed and other measures adopted, or to the drug.

If the case is not one of auricular fibrillation you will know that—apart from those exceptional cases in which auricular fibrillation occurs during a course of administration of the drug—as a rule, though, I am glad to say, not in all cases, the beneficial results will be only moderate or slight, or none at all. If improvement does occur, it can usually be maintained without continuing the drug. And, lastly, in regard to the administration of digitalis, it is possible to do harm in cases of diminished conduction.

Apparently, no preparations of the digitalis series of drugs are superior to the tincture of digitalis for ordinary treatment. Strophanthus and squills should be regarded as substitutes for digitalis. They may be tried when a patient is found to be peculiarly intolerant to digitalis.

In regard to the so-called alleged active principles of digitalis, it cannot be too strongly insisted that there is no pharmacological or clinical evidence that they are either pure substances or that different samples of them are uniform in potency. And therefore there is no reason to believe that they are more reliable than, and possibly they are not so reliable as, different samples of the tincture.

In the most acute cases of cardiac failure, when it may be desirable to elicit a more rapid effect than is possible by the administration of tincture of digitalis or one of its allies by the mouth, strophanthin may be administered intravenously, either two or three doses of $\frac{1}{100}$ of a grain at an interval of two hours, or a single dose of $\frac{1}{100}$ of a grain. Marked improvement sometimes follows in from four to eight hours. The action is in every way similar to that obtained by tincture of digitalis by the mouth, except that the same result follows in from four to eight hours instead of in about a week. Whenever necessary, we can continue the administration of tincture of digitalis by the mouth.

I have come to the end of the time at my disposal. My remarks have, on account of time, necessarily been incomplete. But I have tried to solve in some measure the problem of the significance of cardiac irregularity and the problem of the difference in the results following the administration of digitalis.

Many problems still remain to be attacked and solved. But, in conclusion, I venture to reassert the belief that the introduction of graphic methods in the study of heart disorders has been attended with brilliant results, and will compare favourably with any in medicine or surgery which have been introduced in recent years for the benefit of suffering humanity.

MR. P. H. ADAMS, B.M., F.R.C.S., has been elected to the Margaret Ogilvie Readership in Ophthalmology at the University of Oxford.

THE Metropolitan Asylums Board has arranged, for the benefit of candidates for the Diploma in Public Health, a three months' course of lectures and demonstrations in hospital administration—at the Eastern Hospital, Homerton, by Dr. E. W. Goodall, at 2.30 p.m. on Tuesdays and Fridays, beginning April 4th; at the North-Western Hospital, Hampstead, by Dr. J. MacCombie, at 5 p.m. on Mondays and Thursdays, beginning April 7th; and at the Grove Hospital, Tooting, by Dr. J. Beggs, at 5 p.m. on Wednesdays and Fridays, beginning on April 25th. The fee for the course is three guineas.

An Address

ON

CERTAIN PHYSICAL SIGNS OF MYOCARDIAL INVOLVEMENT.

DELIVERED AT THE OPENING OF THE NORTH-EAST
LONDON POST-GRADUATE COLLEGE,

BY

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LADIES AND GENTLEMEN,—In my address to you to-day I am going to speak chiefly of acute and subacute affections of the heart. This, as you are well aware, is a wide subject, and involves many problems of complexity, and some which will long remain unsolved.

But in one direction the veil of obscurity is rising a little, and it is to the new facts which have been laid bare and their possible significance that I wish to direct your attention at the present time. I have no doubt that it has been repeatedly impressed upon your minds that, in acute and subacute affections of the heart, the damage is not limited to the membranes which cover the organ both within and without. It is needless to remind you that the myocardium also suffers. Yet in practice our attention tends to direct itself especially to that organ or that portion of an organ which is responsible for evident physical signs. Our conception of a given patient's condition is profoundly affected by the appearance of a pericardial friction rub or endocardial murmur. It is but natural, perhaps, under the circumstances, that our thoughts should focus upon pericardium or heart valve, as the case may be. We are impressed by what we hear through the stethoscope, and our diagnosis, "acute pericarditis" or "acute endocarditis," clearly reveals the trend of these thoughts. It is imperative that the myocardium should not be lost sight of; this warning has been repeatedly uttered. The valve and the pericardial sac are relatively but trivial appendages; the simple loss of one or other, though it may increase the burden of work to be borne by the organ as a whole, is not irremediable. Neither valve nor sac is indispensable; efficient or tolerably efficient work can be undertaken and continued for long, if not normal, periods of time by a heart deprived of one of these structures. That fact is taught quite clearly by clinical experience.

The myocardium is the vital part of the heart; its condition should be now, and in the future inevitably will be, the first consideration in all patients in whom the working power of the heart is to be estimated or aided.

The importance of the myocardium, be it in acute or chronic heart affections, is paramount, whenever we regard our patient from the purely cardiac standpoint. And when I speak of the myocardium, I am thinking not only of the contractile tissue of the ventricle, but also of that of the auricles.

These preliminary remarks bring me to my main topic. It is of great importance that any physical sign which speaks of events which are happening in the muscle of the heart should receive the closest scrutiny, and that we should endeavour to ascertain its exact significance. Certain of these signs it is my purpose to discuss.

The greater part of the myocardium may be termed *silent*, in precisely the same sense that certain of the areas of the brain are so termed. Conspicuous lesions of the ventricular wall may be present and give rise to neither sign nor symptom. Large areas of muscle may be degenerate, and the symptoms and signs may be few and obscure. But as there are certain tracts in the central nervous system which, when affected, produce manifest disturbances, so, as recent work has shown us, there are tracts in the heart which display their injuries in a conspicuous manner. The auriculo-ventricular bundle, as you are aware, is a fine neuro-muscular strand which connects to the musculature of the auricle; in its downward course it divides into two main branches, the right and left divisions of the bundle, each of which is connected to the ventricle by an intricate arborization. This system

formed by the main bundle, the two main divisions and their terminal arborizations, is concerned in maintaining the co-ordinate beating of the cardiac chambers. When the auricle contracts an impulse travels through the main conducting tract, passes into its two chief divisions, and is distributed in the two ventricles. The impulse awakens a ventricular response. The impulse occupies time in passing from auricle to ventricle, and, as an indication of the time taken, we use the interval which elapses between the onsets of contraction of these two chambers (*As-Vs* interval). The *As-Vs* interval, as it is termed, thus becomes an index of the functional integrity of the tract as a whole. It may be estimated clinically with considerable exactitude, for we have means of recording the contraction in auricle and ventricle. The two methods of obtaining an index are the polygraphic and the electrocardiographic. The *As-Vs* interval as expressed by these two methods varies normally within certain limits. In polygraphic curves the *a-c* interval (representing, *a* the contraction of the auricle, and *c* the impulse in the arteries of the neck) varies from one-tenth to one-fifth of a second. In electrocardiographic curves the *P-R* interval (representing, *P* the first auricular electric effect, *R* the first ventricular electric effect) varies from 0.12 to, at the most, 0.20 second. Increase in the *As-Vs* interval or a failure of the ventricle to respond to the auricular impulse tells us clinically of deficient action of the auriculo-ventricular tract. These phenomena are spoken of collectively as "heart-block."

Now, chronic heart-block has received much study, and is at the present time one of the most fully understood subjects of modern medicine. Acute and subacute heart-block, though much more frequent, has received far less attention. My remarks will be confined to these last types, and I shall illustrate them freely by brief reports of actual cases.

Acute and Subacute Heart-block in Rheumatic Cases.

We may commence by studying infections of rheumatic origin, for they are of the greatest importance and throw considerable light upon the pathology of chronic rheumatic heart disease. I will cite a characteristic case.

A young girl, who had enjoyed fair health, was admitted under my care at the City of London Hospital, giving a history of palpitation, shortness of breath, and pain in the chest and knees of five weeks' duration. She was very seriously ill, having evident signs of pericarditis and cardiac embarrassment accompanied by commencing signs of oedema of the lungs.

I need not detail the physical signs, but, as a *post-mortem* examination subsequently proved, she was suffering from inflammatory lesions of the pericardium and of all the valves, with the exception of the pulmonary, and that these lesions were clearly of rheumatic origin. Four days after admission irregularity of the heart was observed, and this was proved to be due to heart-block (prolonged *a-c* interval and dropped beats); the heart-block continued until death supervened a fortnight later. A microscopic examination of the heart, undertaken by Dr. Butterfield, revealed perivascular inflammatory lesions of rheumatic type in the myocardium, which, while diffusely scattered in auricle and ventricle, were conspicuous in the region of the central fibrous body, where they had invaded the auriculo-ventricular bundle.

This case, which has been fully described, with the curves,² may be taken as our starting point. It represents one of the most severe forms of acute rheumatic heart disease, invading all the layers of that organ and quickly terminating the life of the patient.

It serves as an extreme type of the cases which I shall present to your notice. The appearance of heart-block at an early period of observation was the most emphatic sign which we obtained of myocardial involvement. I do not say it was the only sign, for in this particular instance we should have been led to suspect it from the profoundness of the symptoms, the prostration, the extreme breathlessness and restlessness of our patient. But it is an important case, because we had the rare opportunity of correlating our physical findings, including the graphic records, with a very complete examination of the heart.

A case, which was not dissimilar in many respects, has been reported by Gerhardt.⁷ It was a case of acute rheumatic heart affection in which all layers of the organ were involved. But there was this important difference: whereas our case died of the rheumatic affection, Gerhardt's case succumbed, while recovering, from a superadded infection—namely, typhoid. In Gerhardt's

case perivascular inflammatory lesions were also found, but while the ventricular muscle did not escape, the bundle showed the chief inflammatory lesion. In this case, too, heart-block was the most positive sign of the myocardial mischief.

My purpose in citing these two cases will be clear. It is to impress the simple fact that the development of heart-block during the course of an acute rheumatic illness is an evidence of myocardial involvement, not necessarily limited to the auriculo-ventricular bundle, but almost certainly spreading to a greater or lesser extent beyond it. The sensitive tract, when implicated, is responsible for signs which, relative to the extent of the lesion, are far more conspicuous than those yielded by implication of the general mass of the ventricular muscle; and I am obliged to cite examples of severe trouble because they alone come to autopsy. The majority of the rheumatic illnesses end in recovery, and this is one of the chief difficulties in studying them. We have largely to judge of the events which have occurred in milder attacks from our findings in those which are severe.

I have notes of another patient, a lad of 14 years, who, having suffered on three occasions from rheumatic fever, was admitted to hospital with frank signs of acute pericarditis. He was for the time being very seriously ill. A large effusion developed which subsided but slowly. Within a few days of admission heart-block was noticed (prolonged *a-c* interval and dropped beats), and this was maintained for a number of days (Fig. 1). Eventually,

Fig. 1.—Polygraphic curves taken from a boy suffering from pericarditis of rheumatic origin. It shows partial heart-block; prolongation of the *a-c* interval and two dropped beats are present.

and as he recovered, the irregularity disappeared, and the *a-c* interval became normal. His heart permanently crippled, he left hospital.

We should stop to ponder the extent of the damage which the myocardium suffers in these severe cases which recover. That it is implicated is certain, that there is partial recovery is equally certain, but that a permanent legacy remains few can doubt.

We have not the opportunities of following the development of a chronic rheumatic heart fully through all its stages in one case. We can but sample our cases here and there, following the disease as a whole in different patients and piecing the history together. Ever and anon a patient succumbs; it may be at an early, it may be at a later period in the history of the affection. We seek what we can find in his remains, but often the morbid process has advanced too far for our needs.

We know the chronic malady to be slow in its completion. Is its progression uniform or does it come in successive and well-defined stages? I believe that we must accept the last view, from the evidence which we possess, and that, while we must recognize that the steps are neither equal in extent nor uniform in their incidence, it is by oft-repeated damage that those incurable rheumatic heart diseases are produced, the unfortunate sufferers from which swell the ranks of our out-patient departments and wards.

We must look for the steps, and the observation of transient heart-block will, I believe, often help us to discover them. This brings us to considerations which I am anxious to emphasize, for the cases already described are those in which none could overlook the signs of infection of the heart. In a very large percentage of the cases of gross heart affection, which from their nature and antecedents may be classed as rheumatic heart disease, no history of grave illness can be obtained. How many young girls come to us with advanced mitral stenosis and degenerate heart muscle and relate the story of a single and mild attack of chorea or rheumatic fever! Yet mitral stenosis does not develop during an attack of either; the advanced lesion is one which has evidently taken a long while in the making. So also has the degenerate muscle

which so often accompanies the valve lesion. The process occurs insidiously, and it is often most difficult to trace its cause.

Now heart-block is not an uncommon accompaniment of acute or subacute rheumatism, and, as in the cases of Cowan, McLeod and Paterson³ and of Magnus Alsleben,¹¹ it may be practically the only sign of invasion of the heart. It is generally transient; it is mild in degree, consisting often of a simple prolongation of the *AS-Vs* interval. Such simple prolongation of the interval will only become manifest when sought deliberately; it is probably a relatively common event in rheumatic cases, being as a rule overlooked.

But it is seen not only during the actual course of the joint invasion, but also from time to time in rheumatic subjects, apart from such disturbance. I may instance a patient who illustrates this contention.

A young woman of 21 was admitted to hospital for an operation in relief of exophthalmic goitre. The eyes had been prominent for two years; palpitation and the enlarged thyroid had appeared six months previous to admission. She was a rheumatic subject, having had an illness, in which several joints were painful and swollen, at the age of 16, and having suffered from frequent attacks of joint pain in the interval. Her heart exhibited the excited action common in her disease; the ventricular rate was 135 per minute on admission; a little enlargement to the left and a systolic murmur were noted. The greater part of the thyroid was removed at the operation, and the progress of the patient was uninterrupted for the space of six days; on the seventh day the heart became slow (40 to 50 per minute), and occasionally irregular, and this proved to be the result of partial heart-block. There was no fever at the time or subsequently. The further course is important. On the first day of the appearance of the irregularity nearly every second auricular impulse failed to reach the ventricle; on the second day the alternate impulses failed to yield response in the majority of instances (Fig. 2); on the third day the failure of response was much less frequent; on the fourth day the failure of response was not seen, but the *AS-Vs* interval, as portrayed in

Fig. 2.

Fig. 3.

Figs. 2 and 3—Polygraphic curves from a case of exophthalmic goitre. The first curve was taken near the beginning and the second curve near the end of a transient attack of partial heart-block. The patient was a rheumatic subject. Fig. 2 shows very frequent dropped beats. Fig. 3 shows simple prolongation of the α -c interval.

both polygraphic curves and electro-cardiograms, was unduly prolonged (*a-c* and *P-R* intervals from 0.28 to 0.30 second). On the seventh day the interval was still prolonged (Fig. 3), but on the eighth day the prolongation had vanished, and was not again seen, though the patient was repeatedly re-examined.

The events which were passing in this heart can be readily interpreted; we can imagine the origin of an inflammatory process, situated in the myocardium and involving the junctional fibres; an exudation of leucocytes attended by engorgement of the vessels and tumefaction; later its subsidence, the absorption of the fluids, and the

relief of the bundle tension. But what we cannot estimate in this patient is the degree of permanent damage left in the bundle and in the muscle of the heart. That in this or similar cases there is permanent damage most, I think, will agree; and what I suggest to you is that it is precisely in attacks such as these that the foundation of gross rheumatic heart disease is laid. I am inclined to regard an attack of this sort as one of a number of steps which, if they succeed each other, lead to permanent crippling of the heart as a working organ. In the patient in question the step would be an early one, and would not necessarily be repeated. I use it as an illustration from this standpoint, and also because had the attack occurred while the patient was at home it would almost certainly have escaped detection; it occurred without symptoms, and its course was afebrile. How often the susceptible experience such attacks is, of course, unknown at the present time; observation of them is often accidental, and their study demands very numerous fruitless examinations. They are certainly far more common than we have had reason to suppose. I might instance a very similar attack, also afebrile, in a girl of 13 years, who had rheumatic fever a year before I saw her. She sought advice for pains in the neck. The heart was normal in size, but there were early signs of mitral stenosis. The heart-block (Fig. 4) lasted for approximately four weeks, and then subsided entirely.

We may pass to later chapters in the same story. If chronic myocardial disease comes in the manner suggested we may expect to see evidences of the same process in cases in which the heart mischief is already far advanced. These cases are under more constant supervision, and our observations are consequently more numerous. I may cite the essential features of two illustrative cases.

A man of 47 years was admitted to hospital with signs of cardiac failure. He had been the subject of rheumatic fever at the age of 30, and was laid up with pleurisy at the age of 40. For twelve months prior to admission he had noticed shortness of breath and palpitation, following exertion and meals especially. For six weeks there had been pain and swelling in the upper abdomen, and the breathlessness had increased; a little dropsy had been present in the legs for a fortnight. Upon examination he was found to be breathless and cyanosed. The heart's impulse was diffuse; the limits of dullness were increased, lying $1\frac{1}{2}$ and 5 inches to right and left of the mid-sternal line. A faint systolic murmur was audible at the apex. The remaining sounds were natural. The pulse was rather feeble, but it beat regularly at 96 per minute. The liver edge was readily felt two fingerbreadths below the costal margin; the legs pitted considerably with pressure, and so also did the sacrum. The urine contained albumen.

During his stay in hospital he developed a febrile attack of approximately five days' duration, the temperature varying, rising in the evenings to 100° or 101.5°, and falling halfway, or actually to normal, in the early hours of the mornings. With the appearance of the fever, heart-block presented itself in the form of "dropped beats" and prolongation of the *As-Vs* interval. The heart-block was maintained over the febrile period, though it gradually diminished in degree. When the temperature resumed and maintained its normal level, the heart-block subsided and was not again noted (see chart and Figs. 5 and 6). During the attack the remaining physical signs of the heart remained unchanged. The patient left hospital with but little improvement in general condition.

The next case is of interest from several points of view.

Fig. 4.—An electro-cardiogram from a young girl who had early mitral stenosis of rheumatic origin. Prolongation of the *P-R* interval and frequent dropped beats are seen. The heart-block was temporary.

In the first place the heart-block consisted of a simple and temporary prolongation of the *As-Vs* interval; there were no "dropped beats." In the second place, there were other signs of myocardial involvement.

A chart showing a transient attack of fever in a patient suffering from cardiac failure. The chart shows also the days upon which the heart-block was found, and the relation of this disturbance to the febrile attack.

Fig. 5.—A polygraphic curve taken from a case of cardiac failure of rheumatic origin during a febrile attack. It shows frequent dropped beats.

Fig. 6.—A curve showing the recovery. The pulse is now regular, and the *a-c* interval is $\frac{1}{2}$ second in duration.

A girl of 13 years was admitted to hospital on July 31st, 1911, complaining of breathlessness on exertion, anaemia, and palpitation, which had been noticed since an attack of rheumatic fever a year before. She had obvious signs of mitral stenosis but there was not much cardiac enlargement. The limits of cardiac dullness lay 1 and $4\frac{1}{2}$ inches to right and left of the mid-sternal line. There were no signs of venous or liver engorgement and no dropsy, but the temperature was raised to 101° . During the next three days the fever subsided. For twelve days it remained normal, and during this period the *As-Vs* interval was found to be normal on three occasions (Fig. 7). Then for a period of five weeks the temperature fluctuated, rising frequently to 99° , 100° , or 101° , sometimes falling to the base line and resting there for a few days, or at the longest a week. Coincidentally the pulse-rate rose, and the child was restive and more breathless. The heart increased in size, until at the end of the febrile period the limits of dullness lay $1\frac{1}{2}$ and 7 inches to right and left of the mid-line. Early in the attack the *As-Vs* interval became slightly but distinctly prolonged (Fig. 8). The course was towards recovery; heart-block was present for a fortnight after the fall of temperature, but eventually cleared up entirely; the cardiac dullness decreased a little in area, but the original area was not regained. The remaining physical signs were not materially affected. The child showed no improvement in general health from first to last, but left the hospital rather worse than better for her stay.

Thus although the heart-block may be transient, as is customarily the case in these patients where its onset is observed, yet the myocardium may, as this case fully illustrates, suffer permanent injury.

It may be asked why the heart-block is so often transient;

it is usually temporary because the injury in the acute or subacute stage exceeds that of the period of recovery which follows; the inflammatory products are absorbed. That damage to the bundle should be permanently manifested

Fig. 7.

Fig. 8.

Figs. 7 and 8.—Two polygraphic curves from a child who suffered from rheumatic heart disease, including mitral stenosis. Fig. 7 was taken before and Fig. 8 during a period of pyrexia. Fig. 7 shows a normal *a-c* interval. Fig. 8 shows prolongation of this interval. There was subsequent recovery from the heart-block.

in some instances naturally suggests itself. I can call to mind no case in which the onset of heart-block has been witnessed and in which it has remained, but that the

bundle is frequently and permanently damaged by rheumatism is fully evidenced by the now well-known fact that permanent increase of the *As-Vs* interval or higher grades of block are by no means infrequent in chronic rheumatic heart disease.

I will leave the case for my contention that heart-block is a valuable sign of myocardial invasion in rheumatic heart disease at this point. I could cite or quote more instances of a similar nature to those which I have related briefly to you, but little could be gained by so doing at present. We require more necropsies than we have at present in confirmation of our clinical conclusions. We require, especially, autopsies in cases from which graphic records have been taken. The field of work is not unpromising, and in this connexion I may quote the words of a pure pathologist who has given special attention to the microscopic lesions of rheumatic heart disease. Aschoff¹ writes: "We do find not infrequently, especially in cases of rheumatic myocarditis, actual destruction of the smaller and larger branches of the conducting system, the already described specific rheumatic nodular structures having a special tendency to develop beneath the endocardium and occasionally right in the connective tissue sheaths of the system."

Heart-block in other Acute Infections.

Amongst the first cases of clinical heart-block which were recorded is the instance reported by Mackenzie.¹⁰ It occurred as a sequel to a severe febrile attack of influenzal origin. Wenckebach¹³ has recorded a similar case; the heart-block was noted during the stage of recovery. In rheumatic infections it appears coincidentally with the pyrexia, where this is present, as we have seen. Heart-block has also been recorded in pneumonia; in Magnus-Alsleben's case¹¹ it first appeared while the temperature was falling. I have seen a similar case.

The patient was a carman, aged 27, and the pneumonia was severe. The temperature on admission was 104° ; the pulse 160. Respirations were very frequent (56 per minute). The lower lobe of the right lung was involved primarily, and the patient was cyanosed and delirious. On the eighth day there were signs of fresh consolidation in the left lung, and the crisis did not come till the tenth day. The delirium continued for three days

* When the patient is going to die the heart-block remains; I have a good deal of evidence which suggests that many of the patients who enter hospital for what is termed "broken compensation" are in reality suffering from a new, and perhaps fatal, invasion of the myocardium.

after the crisis. Heart-block (frequent dropped beats and a long *a-c* interval) appeared two days after the crisis, and persisted, though subsiding gradually, for ten days (Fig. 9). In this case and in Magnus-Alsleben's, both of which recovered, the question of the myocardial infection was complicated, because the patients were upon small doses of digitalis; but

Fig. 9.—A curve showing frequent dropped beats. It was taken from a patient admitted for acute pneumonia, and represents the mechanism of the heart shortly after the crisis.

Fig. 10.—From the same case a fortnight later. At this time the heart-block had disappeared. The patient was upon the same doses of digitalis when the two curves were taken.

that the heart-block in my own case was not attributable to this source seemed clear, as the drug (digitalis *m v*, t.d.s.) was withdrawn on the day when the block appeared, and its readministration as soon as the heart-block subsided failed to reintroduce it (Fig. 10).

I have also seen heart-block (dropped beats and long *As-l's* interval) of a few days' duration as the sole sign of myocardial involvement in a case of infection of the bladder by a coliform organism (Fig. 11); and a case in which there was a mixed invasion of the endocardium by pneumococci and coli has been reported by Cowan, Fleming and Kennedy.⁴ The last-mentioned case came to autopsy; while the muscle of the ventricle showed both recent interstitial change and patchy degeneration of the fibres, the heart-block which had been present was accounted for by an inflammatory lesion of the bundle.

A significant instance of acute heart-block as a sign of myocardial disease is found in diphtheria. Three cases (Magnus-Alsleben,¹¹ Fleming and Kennedy,⁶ and Dunn⁵) of the disordered mechanism have been reported in diphtheria; in each the illness terminated the life of the patient. In a fourth but less certain instance (Price and Mackenzie¹²) the issue was the same. The microscopic examination was undertaken in three cases. In Magnus-Alsleben's case the bundle showed parenchymatous degeneration, and is alone described. In Fleming and Kennedy's case a conspicuous inflammatory invasion of the auricular and ventricular muscle which involved the bundle was seen. In Price and Mackenzie's case the lymphatic infiltration of the myocardium was extreme and the bundle itself was free of it.

While heart-block is necessarily, first and foremost, a sign of bundle involvement, it does not signify that demonstrable lesions are always to be found, though in the majority of cases they can be; to us as clinicians the demonstration of functional disability is of equal if not

without the exposure of the remaining muscle. It may be that under some circumstances the last-named is less susceptible, but the fact remains that the damage is rarely confined to the special tissues. In a measure, therefore, they serve as a gauge for the rest of the myocardium.* Heart-block in acute conditions may not be an infallible sign of general myocardial disease; few signs are infallible, but it is certain that it often speaks of invasion when other signs are absent.

Damage of a Bundle Branch as a Temporary Phenomenon.

I have described to you a number of cases, rheumatic and otherwise, in which heart-block appeared as a transient physical sign, and have sought to convince you of its value as an evidence of myocardial invasion. Now, heart-block is the result of functional deficiency of the auriculo-ventricular junctional system where it exists as a single strand of tissue. You will remember that before this system connects to the ventricle it breaks into two main branches. Functional impairment, though complete, in one of these branches, does not hinder the passage of the auricular impulse to the ventricle, for it is able to pass along the other. Damage to a bundle branch does not originate heart-block. Nevertheless, a lesion of this character, though it does not disturb the rate or regularity of the ventricular

beating, can be detected by modern means. The branches of the bundle can no longer be classed with what I have termed the silent mass of myocardium. The physical sign has been but recently described, and it is electro-cardiographic. A lesion of a bundle branch produces conspicuous distortion of the electric curves, distortion of a definite and known type which can be recognized clinically. The physical sign has been repeatedly found in human hearts since its discovery, but in all the cases in which it has been recognized (my own series includes 15 cases) it has been a permanent feature of the curves. I may briefly describe a case which has recently come under my observation, and in which it was transient:

Fig. 12.—Electro-cardiograms from a case of aortic disease, taken on August 18th, 1912, and shortly after the admission of the patient in a slight febrile attack. The heart was beating regularly. Each auricular contraction *P* was succeeded by a ventricular contraction *R', S', T'*, of anomalous form. The type of ventricular curve in the three leads is one which indicates functional impairment of the right branch of the auriculo-ventricular bundle.

A bookbinder, aged 32, who had enjoyed good health until

six years previous to his admission to hospital, complained of shortness of breath while walking. This had been present for six years; for several months precordial pain of an aching character was

Fig. 11.—An electro-cardiogram from a boy who was admitted to hospital for subacute cystitis, due to a coliform organism. Prolongation of the *P-R* interval and a single dropped beat are seen. The heart-block was transient.

greater import. But, as I have attempted to show, we are justified in utilizing it as a sign upon a broader basis in acute infections. The bundle belongs to the myocardium, and an invasion of the bundle, especially when, as is usual, it happens through vascular channels, can hardly occur

experienced. These were his chief symptoms. He had been

The case reported by James,⁸ in which a deep ulcer invaded the bundle, and that reported by Jellinek and Cooper,⁹ in which a localized area of anaemic necrosis, presumably of gonorrhoeal origin, affected the upper part of the ventricular septum, do not materially affect the argument.

feeling more seedy than usual for some days, sought advice, and was admitted. He was found to have considerable enlargement of the heart, and free aortic regurgitation was present.

Fig. 13.—Electro-cardiograms from the same case taken on the following day. The heart was beating regularly as before, but the auricular contractions P, which themselves remain unaltered in form, are now followed by the physiological type of ventricular curve, indicating in this case the subsidence of the conduction defect in the right branch of the bundle.

The pulse beat perfectly regularly at 64 per minute. There were no signs of venous stasis or of dropsy at his admission, but his temperature was 99°. On the second day it had risen to 100.5°, from which point it gradually fell to normal during the next few days. From this time it remained normal. Electro-cardiograms were taken for the first time on the third day during the pyrexial period; they were of very abnormal type (Fig. 12), and of the form which we now recognize as being characteristic of damage to the right bundle branch. On the succeeding day I had occasion to examine him again, and was surprised to find a complete alteration of the electro-cardiographic pictures. They had reverted to the physiological type (Fig. 13), and this

type was maintained during his stay in hospital.

The change may be interpreted in precisely the same manner that we have interpreted transient heart-block. It is probable that in this case, too, an invasion of the myocardium—which, from the temperature chart, we should be inclined to regard as infective—happened some little while before admission. The damage of the bundle branch was the sole sign detected, and this sign, like the heart-block of our other cases, was transient. It vanished, and the subsidence of the fever was not long delayed.

This is an isolated instance. I have little doubt that the observation will be often repeated, but as to the actual frequency of the phenomena I have but little knowledge. I have described the sign to you, although its value in general practice is negligible, for it serves to impress the lesson that important events occur in the hearts of our patients which we cannot detect by ordinary means; in the investigation of disease the sign is certainly of importance.

In conclusion, let me remind you that in a very large percentage of chronic heart cases we are unable to trace the cause of the disease; chronic heart disease is not always referable to definite illness from which our patients are known to have suffered; oftentimes the reverse is the case and the disease develops silently. If we can discover any indications which help us to recognize the presence of active processes in these patients from time to time—and such I believe are the physical signs which I have described—our knowledge of the disease moves forward by a distinct step.

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OWING to the greater demands made upon the British Fire Prevention Committee for testing facilities, it has been decided to enlarge the testing station and to add to the plant. The main building is being rearranged so that the principal rooms will be available for the committee's technical and historical collections. It is anticipated that the alterations will be completed early in April, when the testing operations of the new session will be commenced with several tests on fire-resisting doors, various forms of glazing, and some new extinguishers; certain further tests with concrete floors, etc., and with partitioning materials, will also be carried out.

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MANIFESTATIONS OF A HEALTHY HEART.

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THE novel thesis set forth in Dr. James Mackenzie's article published in your issue of December 21st last, to the effect that irregularities of pulse frequency and functional murmurs are manifestations of a healthy heart, is one which demands careful consideration. I, for my part, beg to submit that those phenomena are partial expressions of a syndrome of which the fundamental conditions have to be sought elsewhere than in the heart. They are, in fact, signs of a consequential impairment of the heart muscle and of the arterial tunica media. They occur in association with a dicrotic pulse characterized by feeble development, or complete absence, of the predicrotic and post-dicrotic waves. The arteries, in fact, do not receive their normal fill of blood, and are practically empty during an undue proportion of the cardiac cycle. Such phenomena point to two defects: an inactive tunica media and consequent deficiency of recoil and of conductivity, and an actual defect of myocardial energy the importance of which is relatively aggravated by the fact of the central organ being called on to perform a considerable part of the work which should be carried out by the arterial heart. Under some circumstances, such as moderate exertion, or even menstruation during a period of invalid repose, the impoverished tunica media seems to pass temporarily out of action. It is easy to understand how great is the danger of heart strain if any effort be persevered in while that condition lasts. The syndrome in question is one of atony of the whole cardio-vascular system.

An almost universal symptom of that condition is an undue degree of postural tachycardia. The pulse is abnormally accelerated by the act of rising from the recumbent or sitting posture, and that acceleration will, in some cases, be indefinitely maintained until a position of repose is resumed.

The murmurs, also, which are so often to be heard in these cases, are so largely influenced by posture that in many instances they subside on the erect position being assumed, and are, in any case, loudest while the patient is in the recumbent position—that is, as Dr. Foxwell pointed out some years ago, while a distended right auricular appendix is pressing on the root of the pulmonary artery. It is difficult to determine whether the systolic apex murmur, which is sometimes to be heard, should be attributed to papillary weakness, to asthenic dilatation of the auriculo-ventricular ring, or to conduction. Dilatation of the pupil is a common feature of these cases, especially in the young. Irregularity of the pulse, if not actually present, is liable to be induced by any additional call on myocardial energy, or by vaso-dilatation however induced; though in the less severe cases the balance may sometimes be redressed by the stimulus of moderate exercise. In some, as has been stated by Dr. Mackenzie, the irregularity follows the respiratory rhythm; that is, the pulse falters synchronously with the descent of the diaphragm. In the majority of cases there is some degree of extension of the area of cardiac dullness. In childhood and adolescence the blood pressure is low.

As regards the etiology of these cases, careful investigation, especially when it can be carried back to the early stage of life, lights on some form of autotoxis. The most common is that of gastro-intestinal origin. In other subjects a rheumatic or gouty taint seems to bear a causative relation to the syndrome. Not a few subjects bear the rachitic stamp in the form of beaded ribs, retraction of the lower part of the sternum, or a tendency to pigeon-breast. But whatever the cause the result is a general impairment of power and efficiency, and a consequently diminished ability to bear strain, whether mental or physical. Such are the children and adolescents who become the subjects of the strain usually attributed to athleticism. Sound and healthy lads do not strain their hearts in ordinary and legitimate forms of game and sport, but those under consideration, short of wind, subject to palpitation and tumultuous action on exertion, and lacking in staying power, albeit full of pluck and will-energy, may meet misfortune in a football match, a paper-chase, a boat-race, or a game of rackets—I quote cases within