the right ganglion habenulæ to dip downwards into its substance at its posterior border; the right ganglion habenulæ presents all the appearances of an optic ganglion, being composed of a cortical layer of closely-set small cells, with a medullary mass of fine fibres. These fine fibres are not only connected with the nerve of the pineal eye, but also with the basal ganglion, or cerebrum, by

Fig. 9.

means of the fibres forming the tænia thalami, which reach the brain proper by way of the optic thalamus, or medullary stalk.

In addition to the large right ganglion habenulæ, there exists a small insignificant left ganglion habenulæ; this, also, is in connection with a much degenerated median eye, which is situated ventrally to the large, much more perfect eye commonly called the pineal eye.

Summary.—I will not, however, trespass upon your patience longer, as I have now put before you all the most important points upon which I base my theory of the manner in which the vertebrate nervous system has arisen, and have shown you how the single conception that the central nervous system has been formed by the growth of nervous material around the pre-existing inver-

Fig. 10.

tebrate alimentary canal explains the presence of the choroid plexuses, explains the shape of the central cavities of the brain and spinal cord, gives a meaning to the formation of the cerebral vesicles and of the neurenteric canal, gives an adequate reason for the discrepancy between the size of the brain and its case in the lowest vertebrates, and, finally, in the most natural manner accounts for the presence of the pineal eye.

AN ADDRESS ON CERTAIN POINTS IN THE PROGNOSIS AND TREATMENT OF DISEASES OF THE HEART.

Delivered before the Hunterian Society.

By P. H. PYE-SMITH, M.D., F.R.S.,
Physician to Guy's Hospital.

WHEN we say that a patient has "disease of the heart," we generally mean, and are understood to mean, that he has an organic lesion of the valves. If dilatation or hypertrophy of the cardiac cavities, with the effects they produce on the circula-

tion, are secondary to disease of the lungs or the kidneys, we speak of the patient suffering from chronic bronchitis or Bright's disease, as the case may be, with cardiac complications. If again we find that palpitation, irregular pulse, and other signs of disordered circulation are not accompanied by the physical signs which denote a valvular lesion, we seek for the explanation of the symptoms in derangements of the stomach, or nervous system, or in some injurious habit, such as excess in the use of alcohol, of tea, or of tobacco, and call the cardiac disorder a symptom.

Excluding these secondary and functional disorders, what proportion of cases of primary and organic disease of the heart are of valvular origin?

When Laennec introduced the practice of mediate auscultation, the answer would have been that the proportion was small. In the second volume of his great work, the chapter on Valvular Disease, its Anatomy and Physical Signs occupies only thirteen pages. It is preceded by six chapters on Hypertrophy and Dilatation of the Heart and five more on Atrophy, Softening, Indura-

tion, and Fatty Degeneration of the Cardiac Substance, and it is followed by chapters on Pericarditis of thirty-five, and on Aneurysm of forty-one, pages. Laennec's knowledge of cardiac pathology was, however, very imperfect, and scarcely went beyond that of Corvisart. He only recognised valvular disease of the obstructive kind, and supposed that all murmurs were direct, and caused by friction as the blood passed through a narrowed opening. When the existence of leakage, as well as obstruction, and of regurgitant, as well as direct murmurs, was established (chiefly by the labours of Hope and Williams and Corrigan in this country), it was for the first time understood how large a field was covered by the effects of mitral and aortic disease.¹

Of late years a reaction has taken place; the various forms of degeneration of the muscular substance have been studied with minute care, cases of primary hypertrophy and dilatation of the cardiac cavities have been recorded, and the principle of compensation for valvular effects has been worked out. We now understand how a serious mechanical defect in a valve may be made up for by increased vigour of the cardiac pulsations. "If the axe is blunt, thou must put to it more strength." If a pump leaks, we must work it the harder.

Thus the prognosis of cases of cardiac disease with loud murmurs has been regarded more favourably, and has been held to allow of admission in certain cases to the benefits of life insurance. On the other hand, the dangers of dilatation, hypertrophy, and muscular degeneration of the heart, independent of valvular lesions, have been again more fully appreciated than they were after the introduction of auscultation. At present, some physicians seem almost to teach that the existence of a murmur is an unimportant symptom, and that the whole prognosis depends upon our power of estimating the condition of the cardiac muscle and the amount of dilatation or hypertrophy of the cardiac cavities.

I believe that here, as so often, the reaction has gone beyond its due limits, and while fully recognising the importance of the state of the cavities and of their muscular walls, I still regard a murmur which points to an organic valvular lesion as the most important element in the prognosis of disease of the heart. Primary hypertrophy and dilatation are rare, and the great majority of cases of enlarged heart, both clinically during life and anatomically in the deadhouse, can be referred either to valvular disease, or to chronic pulmonary obstruction (I mean obstruction in the capillaries of the lungs), or to the obstruction in the systemic arteries and capillaries which accompanies the chronic form of Bright's disease.

We will deal first with functional disorders; next with that of primary lesions of the cardiac walls and cavities; and thirdly, with that of the more common and more important cases which depend on lesions of the valves.

Here, as everywhere, prognosis and treatment must almost exclusively depend upon accurate diagnosis, and the first step is to determine whether disorders of the circulation are functional or symptomatic of some organic lesion.

I. FUNCTIONAL DISORDERS OF THE HEART.

Idiopathic Tachycardia.—The interval between the cardiac beats is first diminished, but afterwards the pause between the two sounds and the duration of the first are also shortened. In other words, the *pulsus frequens* is almost invariably a *pulsus celer*. We all know how readily the frequency of the cardiac beats may be increased in a healthy subject; we have only to run a hundred yards to increase, and perhaps to double, the number of the pulse. We know how mental emotions produce the same effect, and have learned to make allowance for the rapid pulse of the candidate for life insurance or of a patient who comes to us for the first time. It is remarkable that we have no corresponding means of slowing a man's pulse below its natural frequency when he is lying down, tranquil in mind and body.

This physiological tachycardia quickly disappears when the

bodily or mental excitement which causes it is past. A permanently rapid pulse—that is, one above 80 in the minute—should in an adult be always regarded as a pathological condition. Apart from cases of pyrexia and of valvular disease, such frequency of pulse should direct our attention to the lungs, for it is often an early sign of phthisis. It may also, as Dr. Spender observed, be noticed during the initial stages of osteo-arthritis. Dr. Wilks has recorded several instances in which extremely rapid pulse occurred at the onset of scarlatinal nephritis.

All the conditions which increase the frequency of the cardiac action in a healthy person act with increased effect where there is already disease of the heart. Hence the importance of guarding our patients from any of the causes, whether bodily or mental, which accelerate the pulse. On the other hand it is a matter of common experience that by practice (that is, mere repetition) or by training (that is, repetition scientifically adjusted) the effect of excipients upon the heart may be much diminished.

When a frequent pulse is an accidental or temporary condition, the rapid cardiac action is associated with increased force of the beats, in other words, there is palpitation as well as tachycardia, and the thumping of the heart subsides as its rapidity decreases. A very rapid pulse without palpitation is more likely to depend upon mitral disease or phthisis than to be physiological. Persistent rapidity of the heart does not appear to be a sign of hysteria, for it is always produced by severe bodily pain or by mental anxiety.

The prognosis of cases of primary idiopathic tachycardia appears to be unfavourable. The condition, however, is a rare one, and it is impossible to be sure of our diagnosis until a *post-mortem* examination has established the absence of primary organic disease. Judging from the cases published by Sir Thomas Watson, Dr. Walshe, Dr. Bristowe, and Dr. Broadbent, the number of the pulse may continue at 200 for a long period of time and under excitement may run up to as many as 300 in a minute.

When death has occurred it has been sudden and the heart has been found dilated, without other lesion. In the treatment of such cases Dr. Walshe has found digitalis useless, and Dr. Wilks says the same of stimulants.

The Tachycardia of Exophthalmic Goitre.—Closely allied with these idiopathic cases of rapid action of the heart is the tachycardia of exophthalmic goitre. When not complicated, as it sometimes is, with mitral disease, its prognosis is, on the whole, good as regards the patient's life, although we have little or no means of relieving it by treatment. Some statistics have been published by my colleague, Dr. Hale White, which show that a fatal event is not altogether infrequent.

"Irritable Heart."—The condition described by Da Costa under this name was a rapid and often irregular pulse, with palpitation, in young and otherwise healthy soldiers whose circulation has been overstrained by marching, or by drill. I have seen what is probably a similar condition in young men who have devoted themselves to athletics to a point beyond their strength. The prognosis in such cases is favourable, and the only treatment necessary is physiological rest to the organ, with as much nourishing but unstimulating food as can be assimilated.

Irregular and Intermittent Pulses.—Functional irregularity of the pulse, when permanent and unconnected with cardiac or renal disease, is most often due to flatulent dyspepsia, to chronic alcoholic excess, or to the abuse of tea drinking or tobacco smoking.

With regard to intermittence of the pulse, I would quote the words of Heberden, who wrote in 1782: "Some books speak of intermitting pulses as dangerous signs, but I think without reason, for such trivial causes will occasion them, that they are not worth regarding in any illness unless found with other bad signs of more moment." This has been the experience of all physicians since. Often intermittence is a senile symptom associated with atheromatous arteries, sometimes beginning before the 60th year, and continuing for twenty years. Not only does it appear not to interfere with health, but the patient is often unconscious of the fact. In a patient considerably over 80, I lately noticed the pulse intermit not only for a single, but for two, three, or four beats, and yet neither his own feelings nor the appearance of the face showed the slightest disturbance.

While, however, intermission without other disturbance of the pulse is of slight prognostic importance, when combined with any other form of irregularity it often accompanies the gravest disturbance of the heart, the kidneys, or the brain.

The treatment of an irregular pulse must always depend upon our recognition of its cause. When due to mitral disease, digi-

¹ May I here be allowed to urge the extreme inconvenience which is due to the poverty of our nomenclature of the cardiac valves? The mitral and the tricuspid alone have distinctive names. When we speak of "aortic disease" no one knows whether we refer to a lesion of the aortic valves or to one of the aortic trunk. "Pulmonary disease" and "pulmonary obstruction" are terms still more widely misleading. We have well-established names which refer to the aortic and pulmonary valves—semilunar and sigmoid. Is it hopeless to adopt the latter as a distinctive name of the aortic valves and their diseases, or to use the term *levoisigmoid* for the aortic and *dextroisigmoid* for the pulmonary valves?

tal is an invaluable remedy, upon which increasing experience makes us rely the more; when irregularity is the result of dyspepsia, this drug is, I think, of little service, and more benefit is gained from soda, bismuth, and laxatives, and above all from insisting upon diminution in the amount of food, on prolonged mastication, and on avoidance of those articles which the patient's own experience teaches him will produce flatulence.

The Pulse of High Tension.—The prognosis of a pulse of high tension depends upon its cause. This, as is well known, is most frequently granular degeneration of the kidneys; but although a sign of a serious and practically incurable disease, it is nevertheless not a bad sign; that is to say, if a patient has chronic Bright's disease, it is better that his pulse should be persistent, small, firm, and resisting than rapid, compressible, and irregular. The former condition shows that the hypertrophied left ventricle is keeping up the circulation in spite of increased peripheral resistance, and the chief dangers arise from the high arterial tension leading to rupture and hæmorrhage; but the latter condition shows that the ventricle has become dilated, that it is unequal to its task, that the arterial blood pressure is lowered, and the condition of the circulation approximating to that of chronic mitral disease. In these cases there often supervenes dropsy of mechanical rather than renal origin, as shown by its sparing the face and scrotum and settling in the legs. In such cases apoplexy is, I think, rare, and death often occurs with congested lungs and failing heart.

In the rare but interesting cases in which there is habitually high arterial tension with no renal disease, the treatment advised is saline purgatives with restricted diet. Some of them are believed to be due to gout or to plumbism, but it seems probable that in these the early stage of interstitial nephritis is already present.

II.—PRIMARY LESIONS OF THE CARDIAC WALLS AND CAVITIES.

Passing now to the organic affections of the heart which are unconnected with valvular lesions, we must remember the difficulty of determining the relative amount of hypertrophy and dilatation of the heart, and the impossibility of being sure that it is secondary until we have anatomical proof of the fact.

Hypertrophy from Overstrain.—The cases most frequently met with appear to be those of moderate primary hypertrophy, with accelerated pulse and palpitation, which sometimes follow over-exertion or overtraining in young and otherwise healthy men, or in boys who have not yet reached their full strength.

Closely allied to these are the cases of cardiac hypertrophy in children who have been set to work above their years, which has been described by Sée and other French physicians as *mégalo-cardie des croissants*.

A third clinical group is that of cardiac hypertrophy as the result of the irritable heart described by Da Costa. The same condition has been recognised by Fraentzel in the German army, and by Dr. Myers and others in our own. In all these cases physiological rest to the overtaxed organ is the proper treatment, and probably in the majority of cases it is sufficient. Digitalis, steel, and strychnine are usually indicated, and their use is followed by satisfactory results. There are, however, cases in which, either from the morbid process having gone too far or from the pathology being different, no treatment is of avail, and the case goes on to a fatal termination, often by sudden syncope. Such results have been recorded by Sir Thomas Watson and by Dr. Bristowe. In both their cases the heart was found dilated after death with the valvular apparatus normal. The probability is that, as in cases of secondary affection of the cardiac chambers from valvular or renal disease, there is always at first some hypertrophy in response to increased stimulus. Sooner or later, however, this physiological compensation fails, and dilatation begins.

Rapid Dilatation without Hypertrophy.—Another group of cases of primary enlargement of the heart contains those of rapid dilatation with little or no hypertrophy, which are found to be sometimes the result of rheumatic fever when there has been no implication of the valves or the pericardium. They may also occur as a sequel of other acute febrile disorders, and Dr. Goodhart reported, in the *Guy's Hospital Reports* for 1879, several cases of sudden death from acute dilatation of the heart after scarlet fever, evidently due, not to an increase of intracardial pressure, but to diminished power of resistance of a damaged muscle. Others were, however, probably due to increased blood pressure from concomitant acute nephritis, and would therefore be com-

parable to the cases of rapid pulse with dyspnoea and palpitation coming on in acute scarlatinal nephritis, which were described by Dr. Wilks in a paper before the Clinical Society in 1870.

The only febrile disease in which I have lost a patient from this condition is diphtheria, and then the fatal syncope has been associated with post-diphtherial paralysis; but I have frequently noticed a rapid pulse, after an attack of enteric fever, with palpitation persisting for some time, and giving just cause for anxiety lest fatal syncope should occur. In such post-febrile conditions, although digitalis is a valuable drug, I believe that alcohol, and particularly wine, is a still more important remedy.

Fibroid Degeneration.—Much has been written of fibroid degeneration of the heart, and considerable light has been thrown upon its pathology, particularly as the result of thrombosis and embolism. The late Dr. Fagge contributed a paper on this subject to the *Pathological Transactions* in 1874; it was dealt with by Dr. Quain in his Lumleian Lectures, and by Dr. Charlewood Turner in a valuable contribution to the *Transactions* of the International Congress which met in London in 1881. It is a condition which sometimes leads to sudden death, as in a remarkable case published by Dr. Whipple in the *Pathological Transactions* for 1870; but we have no means of recognising it during life, and from a therapeutic point of view the only point of interest is the possibility of some cases of fibroid disease with pseudo-hypertrophy, dilatation, or cardiac aneurysm depending upon the presence of a syphilitic gumma with interstitial myocarditis. The existence of such cases is not only established by anatomical evidence, but by the occasionally marked benefit of an anti-syphilitic course of treatment. Such have been published by the late Dr. Fagge and by Dr. Balfour of Edinburgh.

Fatty Degeneration.—With respect to fatty degeneration of the heart, it is difficult to speak with confidence as to prognosis or treatment because of the imperfection of our methods of recognising its existence. It seems natural to assume that the mere excess of the normal subserous fat, which we call fatty overgrowth of the heart, must be a matter of less consequence than fatty degeneration of the muscular fibres of a vital organ; but it is doubtful whether this point of view is correct. When fatty overgrowth is excessive, it almost always encroaches upon the muscular fibre, so that the walls of the heart become much attenuated, and sometimes one may see the subserous fat reaching to the endocardium. Apart from this diminution of the muscular tissue of the heart, the mere presence of a mass of inert material within the pericardium will probably hamper the cardiac movements. Intramuscular fatty metamorphosis is not a common event either in cases of obesity or in those of valvular disease. We meet with it most constantly as the result of prolonged and excessive anæmia in the worst cases of chlorosis, in Hodgkin's disease, in leukaemia, and in the idiopathic anæmia of Addison. We also see it as the well-marked result of poisoning by phosphorus. That it leads to the feeble pulse, the syncope, and the liability to sudden death of these disorders there can be little doubt; but either as a primary condition or as an important complication of cardiac dilatation and hypertrophy, it is doubtful whether fatty degeneration plays so important a part as has been supposed.

(To be continued.)

PHTHISIS IN HOLLAND.—Between 1879 and 1888, the number of persons who died of phthisis in Holland is said to have been not less than 247,687, being at the rate of 5.24 per mille of the whole population. During the same period the proportion of deaths from all other diseases of the respiratory organs was only 2.58 per mille.

SUICIDE AMONG SCHOOL CHILDREN IN GERMANY.—Some statistics as to the alarming number of suicides among the pupils of middle class schools in Germany were recently given in the *BRITISH MEDICAL JOURNAL*. The Prussian Government has, it is stated, ordered an official inquiry to be made into the causes of the evil, with a view to its prevention.

INFIRMARY MEDICAL SUPERINTENDENTS' SOCIETY.—The usual monthly meeting of this Society was held at Lambeth Infirmary on November 29th, when a series of interesting cases was shown to the members by the Medical Superintendent, Dr. R. H. Lloyd. This included a case of tetanus, of some days' duration, occurring in a boy aged 10, and a favourable prognosis was given. The clinical meeting was followed by a discussion on various matters connected with infirmary administration.