though it has not perhaps been kept distinctly in view by subsequent observers. This is, that the subjection of the frame to any undue amount of heat so enfeebles its powers of resistance, that the same dose of poison produces a much more deleterious effect than it otherwise would. This occurs in two ways: first, by the tone of the capillaries and small vessels becoming so enfeebled that they no longer are able to retain the blood within their channels, so that extravasations occur, giving rise to purple blotches on the skin, bloody urine, and various internal congestions. I have already argued, in my Lumleian Lectures and work on *Functional Nervous Disorders*, that such may be the effect of heat and of all influences that impair nervous power. Secondly, the same influences probably allow a much larger quantity of morbific matter to be generated than would be in circumstances more conducive to vigour. This is well expressed in paragraph 12 of Sydenham's *Epistolary Dissertation*, vol. ii, p. 61, which I must ask leave to quote: "If this view be true, a blind man may see that the treatment of the first days is all important as regards the event. Inflame the spirits by cordials and bed-clothes, and their power of assimilation, already too great, becomes greater still. Add to this, that the blood and the other humours heated by such processes yield all the easier to the violent impression of the morbific particles. Hence arise more pustules than were wanted, and danger to the patient's life. Contrariwise, the opposite regimen and the free fresh air, soften down the violence of the fevered and acrid particles, and confirm and condense the humours, by strengthening them against the morbific spirits, so as to withstand their attack; whence no more variolous matter is secreted than is proportionate to the genius of the disease."

It is really remarkable to notice how Sydenham's good sense and observation guided him, in the treatment of variola, to practise contrary to what theory indicated. I must quote another passage for the sake of the bearing it has on our original subject; which my readers, I fear, will think I have forgotten. "Those who disapprove of this method, will object that by a narcotic so largely and so frequently given the peccant matter will become fixed and salivation be checked. To this I reply, that it is true that the salivation may be diminished; still it will never wholly cease. Nay more, some time after the anodyne has been given, it will break out afresh. From this accrues a double advantage. The patient, having been restored by the anodyne, is all the stronger for the expectoration. The excreted saliva, although scantier, is all the better excocted. In the next place, the decrease of the ptyalism is well made up for by the increased swelling of the hands and face. This is all the surer and all the freer for the repetition of the narcotics, especially on those days when the swelling most regularly takes place..... In respect to these, I confidently assert that no competent judge can deny that the absence of these swellings, on the days on which they are due, is a worse omen than the interruption of the ptyalism. For my own practice, I would rather risk a check to the ptyalism than a check to the swelling; and I think that this is so thoroughly required by the disease, that the practitioner who debars his patient of such an auxiliary has but little observed the complaint." (*Epistolary Dissertation*, vol. ii, p. 74, par. 40.)

From this passage, it is clear that Sydenham was not afraid of arresting elimination by a pretty free use of opium; and that he set more value on the due swelling of the hands and feet than on the salivary excretion, though the latter is surely more eliminant than the former. Paregorics he gave, as he tells us, once or oftener in the day, from the sixth to the seventeenth day of the disease. Another means which Sydenham used to check "the inordinate assimilation of variolous matter", was the free addition of some spirits of vitriol (sulphuric acid) to some thin drink. This "goes far to help the patient out of danger." Sulphuric acid certainly acts as an astringent to the skin, and, as most of us believe, to the bowels. My own experience convinces me that it is not eliminant.

[*To be continued.*]

ON PROGNOSIS IN HEART-DISEASE.

By W. H. Broadbent, M.D., Assistant-Physician to St. Mary's and the Fever Hospitals; Lecturer on Physiology at St. Mary's Hospital Medical School.

[*Continued from page* 598.]

Hypertrophy and dilatation, then, are looked upon as caused by the valvular lesion, and as indicating its extent. The greater the degree of these structural changes, as ascertained by increased area of deep cardiac dulness, by the situation and character of the impulse and apex-beat, and by the modification of the heart-sounds, the greater the mechanical difficulty resulting from the altered valves, and the more grave the prognosis.

I must guard myself from being understood to assert without qualification the converse of this—that the less hypertrophy and dilatation, the smaller the valvular injury. Both propositions, indeed, would be rendered more exact, if preceded by the words, "in the absence of symptoms".

The prognostic meaning of hypertrophy and dilatation is recognised by all writers alike; but there is considerable diversity of opinion as to the relations between these changes and the valvular disease on the one hand, and subsequent effects on the circulation on the other. The idea which naturally presents itself, that the mechanical obstacle arising from the state of the valves is the cause at once of the hypertrophy and dilatation and of the derangement of the circulation, is, it seems, too simple. According to most authors, the symptoms and the ultimate fatal termination are due, not to the valvular disease itself, but to the hypertrophy and dilatation with which it is associated. Again: these structural alterations are considered by many writers as not altogether or even mainly the result of the valvular alterations, but of various accidental conditions, such as carditis, innutrition, etc., which are present in some cases and absent in others; the changes in the walls and cavities of the heart occurring or not accordingly.

It can never be altogether useless to ascertain the real relation between pathological sequences, or the connexion between pathological conditions and symptoms; and I propose to examine the two points referred to, taking first that which requires something in addition to the mechanical difficulty occasioned by the valvular lesion, as needed to give rise to changes in the muscular walls of the heart. It is stated thus by Dr. Walshe: "No direct ratio holds between the amount of hypertrophy and the amount of valvular obstruction, showing that there is something beyond the mechanical difficulty." Dr. Stokes, again, asking why in one case the cavities are unchanged, in another dilated, suggests as the cause carditis, imperfect arterialisation of the blood, obstruction of the coronary arteries, etc. It is not

denied, of course, that carditis, degeneration, and the other affections named, may give rise to dilatation and hypertrophy; they may do this when the valvular apparatus is perfect, and, *à fortiori*, when this is deranged; but it is certain that they are not essential, and that the changes in the valves alone are sufficient. The muscular structure is often found perfectly free from any evidence of innutrition, or degeneration, or inflammation, when the heart is considerably dilated and hypertrophied, and no assignable cause can be found but the valvular lesion. My own conviction is, not only that they are sufficient, but that they are the predominating cause; the other assigned causes being only occasionally and accidentally operative. How otherwise does it happen that hypertrophy and dilatation are so common in valvular disease, so exceptional independently of this, unless, indeed, a mechanical difficulty of another kind, such as pulmonary or renal or arterial disease, exists? As to the want of correspondence between the amount of valvular alteration and of muscular change, it must be remembered that before death we have absolutely no means of directly estimating the former; and that, in a large proportion of the cases referred to by Dr. Stokes, the occurrence of dilatation, or its absence, would be explained by the existence of extensive or slight mechanical difficulty. Even the fact, unquestionably true, stated by Dr. Walshe, "that no direct ratio holds between the amount of hypertrophy and the amount of valvular change," as ascertained by *post mortem* examination, is capable of explanation—first, by the circumstance that different affections of the valves have inherently and mechanically different degrees of tendency to the production of this result. Again, comparing only similar affections, *time* is an important element in the development of hypertrophy; and it is often very difficult to assign a date to a given morbid condition. Thus, in two cases of apparently equal obstruction, one will be accompanied by great hypertrophy, because the patient did not at once succumb to it; the other with comparatively little, because the difficulty increased more rapidly than the heart's power, and death occurred quickly. Again: the time of life at which the lesion of the valves takes place forms an important consideration. The cases of enormous hypertrophy and dilatation we meet with arise almost exclusively in early life; later, the heart no longer undergoes these great changes, but loses, as I should put it, its adaptive capability; and thus an obstruction, which at fifteen or twenty would be survived with enormous hypertrophy, would at forty or fifty prove fatal with much less change. Again: the variable amount of work the heart has been called upon to perform by the different habits of patients, and especially the period after the occurrence of the valvular change at which active exertion was undertaken allowing or not the heart to adapt itself gradually to this change, will have great influence on the condition of its walls and cavities. It will be seen from these considerations, that an unvarying direct ratio between the valvular and structural changes is not to be looked for, and that its absence furnishes no valid objection to their standing in the relation of cause and effect.

This question will again be referred to when the second of the two points, which is closely associated with it, is considered. This is, that the symptoms and ultimate fatal termination of heart-disease are due rather to the hypertrophy and dilatation than to the valvular affection; or that, at any rate, these structural alterations add a new source of danger. As to the exact share of each, opinions differ. Dr. Chambers, in his "Clinical Lecture on Heart-disease," says: "The importance of valvular lesions consists in their liability to cause enlargement of the heart;" and again: "If the muscular structure remains healthy, injured valves do not appear capable of causing death." Dr. Walshe considers that "dilatation renders any valvular condition more dangerous," and that "hypertrophy has the same kind of effect to a less degree;" and, if I have rightly gathered his meaning, it is to the effect that hypertrophy and dilatation are capable of giving rise to certain results; when these conditions are found associated with valvular disease, any such results are still to be considered as due to them, the residual phenomena only to the valvular lesion. Dr. Stokes, without making any such definite statements as these quoted from Dr. Chambers and Dr. Walshe, holds similar views. He says: "It is in the vital and anatomical condition of the muscular fibres that we find the key to cardiac pathology; for, no matter what the affection may be, its *symptoms mainly depend* on the strength or weakness, the irribility or paralysis, the anatomical health or disease, of the cardiac muscles. It was long ago observed by Laennec, that valvular diseases had but little influence on the health when the muscular condition of the heart remained sound; and every day's experience confirms this observation." Dr. Latham, speaking of hypertrophy as existing with valvular disease in a case untroubled by cardiac symptoms, says: "Hypertrophy is here the safety of the patient. Take away the hypertrophy and leave the injured valve, and the patient would be in a far worse state." "Hypertrophy" he elsewhere pronounces to be a "tremendous evil"; but it tends to counteract another tremendous evil, valvular disease. Dilatation supervening on disease of the valves is a "virtual obstruction" added to a "positive obstruction". Dr. Fuller, in speaking of the prognosis of endocarditis, seems, on the whole, to regard the valvular lesion as causing the structural changes, and also the symptoms; but he does not consistently hold to this view. Thus, at page 108, he says: "The more serious the mischief, the greater the obstruction to the blood's current, the quicker will hypertrophy and dilatation result, and," he continues, "the sooner will the effects of these lesions be perceived." Curiously enough, he later inverts the conditions, and, in the prognosis of valvular disease, speaks of it as "more rapidly fatal when it occurs in a heart which is already dilated and hypertrophied, than when it is set up in a healthy heart;" which is true, no doubt; but such a case must be of rare occurrence, and the peculiar sequence excessively difficult to make out.

Dr. Markham says: "Hypertrophy and dilatation of the heart, and diseases of its valves and orifices, are pathological states of the heart so closely blended together, that in practice it is scarcely possible to describe the consequences which ensue to the system from the one apart from the other."

There are here at least three perfectly distinct ways of regarding the connexion between hypertrophy and dilatation and subsequent symptoms—as the main cause of these symptoms, as aiding in their production by aggravating the ill consequences of valvular derangement, and as tending to neutralise these ill consequences. The point in which all agree is, that valvular disease accompanied by these structural changes is more serious than when it is not thus accompanied; but this fact is capable of the double interpretation, either that the valvular affection is rendered more serious by their presence, or that they are present because it is more serious. Which of the two interpretations is the more correct, will be best determined by considering what

would be the consequence of the valvular without the structural changes and the modifications these will produce. That in practice, as Dr. Markham says, it is scarcely possible to distinguish between the effects of the one and the other, is sufficiently evident from the diversity of opinion shown to exist.

The statement of Dr. Chambers, however, that "injured valves do not appear to be capable of causing death, if the muscular structure remain healthy", admits of direct refutation. It is disproved by the single fact, that accidental rupture of a valve may at once give rise to fatal syncope. If this do not occur, it may be followed by a rapid development of cardiac symptoms, dyspnœa, pulmonary obstruction, dropsy; and the more rapidly death ensues, the less of structural change will be found. Sometimes, also, endocarditis produces such extensive mischief in the valves, that it proves speedily fatal without the production of any considerable degree of hypertrophy or dilatation.

I may here remark that the observation of Laennec quoted by Dr. Stokes, to the effect "that valvular disease has but little influence on the health when the muscular condition of the heart remains sound", which, at first sight, might seem almost identical with what is said by Dr. Chambers, is perfectly distinct, and is indeed a mere statement of fact; but the explanation of this is that, when the muscular condition remains sound (understanding by this free from hypertrophy or dilatation), the valvular affection is slight. If Dr. Stokes had said, in the passage given above, that the prognosis turned upon the condition of the cardiac muscles, instead of that the symptoms depended upon this, no exception could have been taken to the statement; and I cannot withhold an expression of my admiration of the lucid and forcible way in which he sets forth the signs of danger in heart-disease, or of the clearness and precision with which Dr. Chambers gives the elements of prognosis and the indications for treatment.

As was said a moment ago, it will best be seen whether hypertrophy and dilatation are to be considered as the cause of the symptoms, and whether, as Dr. Walshe has it, they add to the danger of valvular disease, when we examine the effects the valvular lesions would necessarily produce, independently of the changes in the walls and cavities, and the modifications of these effects to which the structural changes would give rise. This will also furnish an opportunity for tracing the mode of their causation.

First, then, as to aortic constriction. If this exist to a degree sufficient to give rise to mechanical obstruction, since the same force will not propel the same amount of blood in the same time through a narrower orifice, there must be either increase in the propulsive power of the heart or diminished rate of circulation. The increased power is gained by hypertrophy; and, unless this go beyond the degree necessary to overcome the obstruction, which is extremely improbable, it cannot be considered as the cause of subsequent symptoms or as adding to the danger of the valvular disease.

A direct aortic murmur, accompanied by hypertrophy, is attended with greater danger than one not thus accompanied; not because a new element of danger is superadded by the hypertrophy, but because the valvular lesion in the one case has occasioned a mechanical difficulty sufficiently great to call for hypertrophy and not in the other.

Again, in aortic regurgitation, since a certain proportion of the blood sent into the aorta at each systole returns into the ventricle, there must be, in order to maintain the same rate of circulation, either an increase in the number of heart-beats per minute or an increase in the quantity of blood expelled by each. The increased capacity of the dilated left ventricle brings about the latter result, and thus compensates for the regurgitation. The greater the amount of regurgitation, therefore, the greater the necessity for dilatation.

The dilatation of the left ventricle known to accompany aortic regurgitation, is accounted for by the fact that during diastole the ventricle is exposed to a double distending force—the entry of blood from the auricle and pulmonary veins and the backward rush of blood from the aorta. When this is the case in any considerable degree, dilatation is inevitable; and no structural change in the muscular fibres is needed for its production. Unless constriction co-exist, there is no direct provocation to hypertrophy in increased resistance to the exit of blood; but there will be a call for increased exercise of force in the additional quantity of blood to be lifted at each systole, and a degree of hypertrophy results from this.

The dilatation of aortic regurgitation is a totally distinct thing from the dilatation which is sometimes met with as the result of structural weakness or degeneracy of the muscular walls of the heart. The two have only one condition in common—increased capacity of the ventricle. In the one the walls yield because they are inherently weak, and the effect on the circulation is not materially different from the effect this weakness would have if the dilatation did not follow—a tendency to stagnation from want of *vis a tergo*. In the other, the walls yield, not because they are weak, not even because they are weak relatively to increased demand for force, but because they are exposed to special abnormal causes of dilatation. If the valvular lesion could be removed, leaving the hypertrophied and dilated ventricle, the effect on the circulation would be not stagnation but acceleration.

Dilatation with hypertrophy cannot, then, in aortic regurgitation, be considered as aggravating the evils of the valvular lesion. On the contrary, it tends to compensate and neutralise them, and is distinctly conservative. Nor can it be said to give rise to the symptoms; and, indeed, in my opinion, the symptoms ascribed to dilatation are in all cases due rather to the cause of this condition, whether this is in the valves or in the muscular walls. The fact that dilatation is sometimes excessive and out of proportion to the hypertrophy, does not invalidate what has been said as to the primarily conservative character of this change. The increase of capacity is a necessity in order to make up for the regurgitation. When this goes beyond the required point, or hypertrophy fails to keep pace with it, there is a default in the compensatory arrangement from want of strength in the heart's walls; and the predominance of dilatation marks the degree of this failure.

Dilatation itself directly contributes an element of danger only when it gives rise to regurgitation (a valvular derangement, it may be remarked); indirectly it does so as necessitating increase of force on account of the increase of surface in the ventricle.

When, as is often the case, aortic regurgitant disease is set up late in life from degenerative change in the valves, the muscular structure is frequently also weakened, and dilatation from this cause may be superadded to the dilatation produced mechanically; but it is the weakness, the want of power, which is the source of danger, rather than the change of form.

[*To be continued.*]