2. That watery or rice-watery purging in cholera epidemics is not always followed by the state of asphyxial collapse, recovery taking place in some cases without its occurrence.

3. That collapse, so far as the asphyxial symptoms are concerned, depends largely and primarily on blood changes consequent on the loss of water and salts mainly. Before going further I shall attempt an epitome of Dr. George Johnson's theory, in order that my objections to it may be more clear.

1. There is no ratio, or an inverse one, between vomiting and purging and collapse. In the worst and most malignant cases, the patient may die of collapse without either purging or vomiting having taken place. The symptoms of collapse are not such as an excessive drain of fluid is likely to produce.

2. The cholera-poison in the blood eliminates itself by purging and vomiting, and produces asphyxia directly from its irritating properties upon the smaller branches of the pulmonary arteries.

3. Consequently, astringents, by retaining the poison in the system, aggravate the disease instead of curing it.

The corner-stone of Dr. Johnson's theory is the assumed existence of a class of cases, "*the worst and most malignant*," in which the asphyxial symptoms of collapse are primary and unpreceded by vomiting and diarrhœa. Though there are numerous authorities in support of this view, I have seen two epidemics of the gravest character in Mauritius, and frequently had cases of cholera, during several years in Indian hospital practice, without ever having the fortune to find or verify one of these cases of dry cholera, and have hence grown to be very sceptical of their existence.

The physician, at a time of intense pressure, is apt to admit, without much investigation, the first statement made; and many patients, such as soldiers or sailors, are anxious to hide the self-neglect, against which they have been forewarned.

This point merits close attention in future epidemics; every alleged case should be sifted retrospectively, and *post mortem* appearances should be carefully described.

Terms suggestive of arithmetical precision are misleading in a discussion involving the intricate complexity of pathological reactions. The varied states of the recipient body are conditions of the cause, equally with the morbid poison introduced. We know too little of either sets of factors to establish ratios; but if there were an inverse ratio between watery purging and collapse, the latter should occur rather soon than late, before than after elimination; yet it will be conceded that the first evident effect of the cholera-poison is most generally diarrhœa, fæcal (which, I presume, is the equivalent of bilious in Dr. Johnson's terminology) at first, later, clear or rice-watery.

The term serous, often applied to the latter, is as misleading as collapse; the one suggesting loss of the albuminous parts of the blood, just as collapse leads the mind rather to syncope than to asphyxia. Yet in cholera the loss of albuminous parts of the blood is hardly appreciable, the loss being mainly of water and salts; to this doubtless is due the frequent rapid recoveries, so different from those after hæmorrhages and other exhausting profluvia, in which the blood loses its more highly organised parts.

I believe it to be matter of fact, that asphyxial symptoms are not known to coexist with or to succeed purely fæcal diarrhœa; though, when this is excessive, syncopal symptoms may ensue, producing one kind of collapse. Asphyxial symptoms, however, only follow in cases of watery or rice-watery stools, where the watery and saline parts of the blood are diminished to such a degree as to affect the normal reactions between the blood and the air in the lungs, from which time there is diminished attraction of blood to the pulmonary capillaries, and what blood does pass through the lungs is imperfectly arterialised, the systemic arterial vessels are imperfectly filled, while the venous system and right side of the heart are filled to repletion, and the full symptoms of cholera collapse are established.

Though we cannot by any artificial experiments deprive the blood of its watery parts and salts, and hence have been driven to estimate the consequences by the analogies of blood-letting, etc., we have occasional proofs of asphyxial symptoms following copious watery purging, which has originated merely in errors of diet, or abuse of purgatives, where there has been no suspicion possible of cholera-poison. As an instance of this, I give a case in the *ipsissima verba* of a weekly report of April 30th, 1864.

"Corporal M. has had several attacks of what appears very like cholera in all its symptoms, purging of watery fluid, severe cramps of legs and arms, and a leaden look, with the cold sweats of collapse. He generally makes a rapid recovery; on this occasion, I tested the clear straw-coloured fluid passed, expecting to find it rich in albumen, but neither boiling nor the addition of nitric acid caused any precipitate.

"This may account for the slight ulterior effects of attacks apparently so severe as his have been."

Though this is a mere sketch of the case, I may say that he resembled in all essentials the asphyxial type of collapse, with death-like look, and sodden, shrivelled fingers.

This was the eighth attack, more or less severe, on record. In three of the previous attacks a red herring, a Christmas dinner, and a seidlitz powder, were the causes assigned; on this occasion, as on several others, he was unable to or unwilling to assign any cause for the attack.

I have little doubt that similar cases will be remembered by others. Their occurrence is wholly opposed to the view of the asphyxial condition being due to a spasm of the smaller branches of the pulmonary arteries, arising from the direct irritant effect of a specific poison, and lends support to the view that the central fact in cholera is a material change in the constitution of the blood, mainly a loss of water and salts, rendering it unfit to undergo the usual chemical change in the lungs (with its train of consequences on the circulation), and that this loss is mainly produced by profuse watery discharges into the gastro-intestinal canal, as evidenced by the order in time of the successive phenomena.

If the account I have given be accurate, there is good ground shown for preferring a stimulant opiate, and astringent treatment, to an eliminative one, during the short interval, when, and when only, treatment has any well proved value.

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* p. 661 *of* vol. i *for* 1866.]

In affections of the mitral valve, the effects of the derangement no longer fall upon the left ventricle, but on the auricle, lungs, and eventually on the right ventricle. When there is regurgitation through the orifice, it would seem, at first sight, that a certain increase of capacity would be needed to make up for this loss, and part of the force of the left ventricle will be wasted or misdirected in driving blood back-

wards as well as forwards. Primarily, however, there exists no mechanical cause of dilatation, and the provocation to hypertrophy is but slight. Even this will be wanting in obstructive disease. Accordingly, in mitral regurgitation, we do not find any considerable degree of dilatation or hypertrophy in the left ventricle, and in mitral constriction there may be contraction. A moment's consideration will show that increased capacity and strength of this cavity would by no means have the same effect in mitral as in aortic regurgitation. In the latter condition, so long as the mitral valve is competent, the result is that a larger volume of blood is thrown into the aorta at each systole; in the former, the increased amount of blood contained by the dilated ventricle would be divided between the aorta and the auricle, and a part of the increased force would be expended on the lungs. The auricle, however, becomes dilated and hypertrophied, as a result of the backward pressure, and this extends backwards through the lungs, and is felt by the right side of the heart, which becomes hypertrophied, often to a remarkable degree. It is difficult to understand how the pulmonary vessels and capillaries can resist the tension to which they are exposed; but it cannot be doubted that the right ventricle, by the additional force it exercises, aids in supplying the left ventricle with blood. This I look upon as a compensatory action of considerable importance. The increased pressure will send the blood more rapidly through a constricted orifice, and will tend to diminish the amount of regurgitation when the valve is incompetent. The thin walls of the right ventricle, however, readily yield to a distending force; and a frequent consequence of this is regurgitation through the tricuspid orifice, a provision which postpones the occurrence of pulmonary apoplexy.

I think it will be evident from the considerations adduced, that the relations between valvular and associated structural alterations is one of cause and effect; and I am convinced that both will be better understood, and their bearing on prognosis better appreciated, if, instead of looking upon their combinations as valvular disease of altogether uncertain amount, complicated by hypertrophy and dilatation of independent and accidental origin, or the converse, the hypertrophy and dilatation are regarded as the direct results of the valvular affection, and as measuring the degree of mechanical difficulty occasioned by the obstruction or incompetence.

It further seems to me clear that, on the whole, these changes, dilatation as well as hypertrophy, tend to neutralise the mechanical obstruction resulting from the imperfect action of the altered valves, and are thus distinctly conservative.

A valvular murmur, then, accompanied by hypertrophy, or dilatation, or both, is still attended with greater danger than a similar murmur not so accompanied; not, however, because the hypertrophy and dilatation add new elements of danger, but because the valvular change causing the murmur gives rise also to mechanical difficulty of serious character in the one case, and not in the other.

If, in addition to the disease in the valves, there be degeneration of the muscular substance of the heart, the dilatation may be taken as expressing the relation between the mechanical difficulty and the power of the heart to cope with it.

At the point which we have now reached, we may consider the prognosis of those cases in which valvular murmur exists, but without any apparent effects, the health and strength remaining good; and it must be remembered that, in heart-disease especially, prognosis includes not only the signs of approaching evil, but the probabilities of continued immunity.

When with the valvular murmur there is no change in the form or volume of the heart, or derangement of its action, it may be concluded that the valvular mischief is slight and unimportant. If, further, it be known to be of old standing, and to have been caused by acute endocarditis, the probabilities are, that it will not shorten life, will give rise to no symptoms, and have no ill effect whatever on the health. If, on the other hand, the murmur be recent, and have come on late in life, the same hope cannot be held out; it may indicate incipient degenerative change, and the progress of this change will determine the future of the case. Careful observation of the murmur from time to time, and of the state of the heart's walls and cavities, will be required in order to arrive at a safe prognosis.

When the murmur is accompanied by evidence of structural alteration, a cause for this exists in mechanical difficulty occasioned by the change in the valve. The seat and character of this difficulty are to be taken into account, and the order of relative gravity of the different valvular affections must be borne in mind; but we look chiefly to the condition of the walls and cavities. If the amount of change be only moderate, and especially if hypertrophy predominate over dilatation, the patient, subject to the conditions mentioned above respecting the stationary or progressive character of the disease of the valve, may have an indefinite term of life before him, untroubled by cardiac symptoms. But the existence of the hypertrophy shows the valvular lesion to be such as to interfere with the circulation; and it must be remembered that the compensatory arrangement may be easily disturbed. Precautions, therefore, must be taken against occurrences which would throw increased labour on the heart, such as overwork, exposure to cold, etc.; and, if complications arise, the extent of the hypertrophy or dilatation will form an important element in the estimation of the degree of danger, indicating, as it does, a pre-existing injurious tendency.

In addition to the structural condition of the walls and valves of the heart spoken of, there may be indications of its functional condition (the subject being still in the enjoyment of good health), which may be reassuring, or the reverse. It will confirm other favourable signs, if the heart's action be equal, regular, tranquil, and of moderate strength, and not readily excited to palpitation. If, on the other hand, slight causes be sufficient to give rise to hurried and violent action, or if habitually there be any considerable departure from the normal force or regularity, these are further symptoms of the serious character of the affection.

The prognosis becomes more grave with increased amount of structural change, and more especially as dilatation is associated with, or predominates over, hypertrophy; but these conditions, even in an extreme degree, by no means threaten a speedy dissolution, and are not inconsistent with a prolonged and comfortable existence. There is, however, an increased liability to complications from comparatively slight causes; and sooner or later a time arrives when the heart is no longer equal to the work imposed on it, and symptoms of embarrassed circulation arise. These then form the next element of prognosis, and, when well marked, give more definite, though more unfavourable, indications. The period of time at which they follow the occurrence of the valvular lesion is a most important point to be ascertained. The more quickly they supervene, the more serious their significance. When they appear early, they show that the mechanical difficulty caused by the valvular incompetence has been too great for the compensating tendency of the struc-

tural changes, which thus cease to indicate its amount. They are dwelt upon with great force by Dr. Stokes, more particularly as he considers "that the number of cases in which we are warranted in making a special diagnosis of valvular disease is small." We judge of the functional as well as structural condition of the heart by its action as seen, felt, or heard, through the walls of the chest, both habitually and as influenced by various circumstances. We learn the state of the systemic circulation by the pulse, and by examining the veins and capillary circulation; and of the pulmonary circulation, by the degree of dyspnœa, and the readiness with which this is induced. But there may also be present some of the train of cardiac symptoms, uneasy sensation, oppression, or actual pain referred to the heart itself, low spirits and irritability of temper, pulmonary embarrassment in various degrees, from mere shortness of breath up to the terrible paroxysmal dyspnœa termed cardiac asthma, or there may be incipient dropsy. The complications which immediately threaten life are reserved for later consideration.

The heart and pulse are to be observed mainly with a view to the indications of sustained or failing power in the heart, and of sufficient or insufficient supply of blood in the arteries. Speaking generally, if the heart evince vigour without excitement, and a strong heart-beat be not contradicted by a weak pulse-wave, the effect on the circulation has not reached a point attended with immediate danger. But each of the different valvular diseases gives rise to a characteristic modification of the pulse, which must always be taken into consideration. Aortic constriction tends to render the pulse small, with a prolonged wave. Aortic regurgitation is associated with the well known visible and audible collapsing pulse. Mitral regurgitation gives rise to irregularity in force and rhythm both in the action of the heart and in the pulse at the wrist. The degree in which these peculiarities are manifested, especially when traced by the sphygmograph, may assist in estimating the amount of change in the valve; if they be not borne in mind, none but fallacious inferences would be drawn from the pulse in prognosis. When, in aortic obstruction, it becomes fluttering and irregular, and when the characteristic collapsing pulse of regurgitation ceases to be evident and is replaced by a pulse weak and frequent, the heart is failing, and unfavourable symptoms, if not present, may be expected. In mitral regurgitation, there may be extreme irregularity of the pulse; and there may be occasional contractions of the heart, which, from momentary weakness or from want of a sufficient amount of blood in the ventricles, do not reach the wrist, thus causing one kind of intermission. But so long as the arteries are, on the whole, well filled by the systole, the heart retains a degree of vigour, and, in the absence of other unfavourable indications, may be expected to carry on the circulation indefinitely. It is, indeed, astonishing how little trouble may accompany mitral regurgitation, with enlargement of the right ventricle, and an irregular and intermitting pulse; and how long life may last. When it becomes weak, frequent, and fluttering, even though more regular, or when it is altogether uncertain, the imminence of dangerous consequences is great.

The occasional abortive systole referred to is commonly, if not always, the result of varying pressure on a dilated heart in the movements of respiration. In emphysema and bronchitis, the pulse is always weaker during the laboured inspiration, and stronger during the forcible expiration, as pressure is applied to, or withdrawn from, the heart; and a sustained powerful inspiration will in any person cause the pulse to be for the moment slow and feeble; but, when the heart is dilated and weak, it feels the effects even of ordinary respiratory movements, and frequently to such an extent that a systole corresponding with commencing inspiration, or with the respiratory pause, is so far neutralised by the removal of pressure as to fail in propelling the blood into the remote arteries; or, as it has seemed to me, the expansion of the chest has acted rather on the thin-walled left auricle, preventing the blood from entering the ventricle.

To return to our subject: whatever the structural condition of the heart and its habitual action may be, if it be liable to excitement from slight and varied causes, such as a little exertion, change of posture, moderate emotion, the taking of food or stimulants, this is an unfavourable sign.

Fulness of the veins indicates obstruction to the entry of blood into the right side of the heart; and this again usually implies obstruction to the pulmonary circulation. Pulsation in the large veins of the neck is the most reliable sign of tricuspid regurgitation—the last in the chain of consequences tending to dropsy.

In the state of the capillaries, we find most unmistakable evidence of an obstructed or stagnating circulation. The face congested in different degrees, the cheeks of a deep or dark red, or approaching to purple in hue, with blueness of the lips and lividity of the nose; or the face may have a dusky pallor, the nose being cold and livid. The extremities, again, may be cold and purple; the colour returning slowly after pressure. It is not necessary to go fully into the signs of sluggish capillary circulation, or to point out their significance. They belong in their marked form to a late stage of the disease, and their prognostic import is not to be mistaken.

The symptoms of secondary respiratory embarrassment are of every degree of intensity, and belong to every stage of valvular affections. Shortness of breath on exertion, and especially in walking up-hill or against the wind, is common to all heart-disease; and, except when experienced in an extreme degree, is not a very serious indication. It is simply an exaggeration of what occurs to every one taking violent exercise. The simultaneous pressure on the veins by all the muscles of the body at first brings the blood to the right side of the heart faster than it can be sent through the lungs and aërated—whence the panting. If the exertion be begun gradually, or be persevered with in spite of the dyspnœa, the circulation is equalised, and we "regain our wind". In valvular disease, this takes place with greater difficulty; but commonly, except in some cases, by starting gently and not giving in to the early dyspnœa, a sufferer from this may be equal to considerable exertion or sometimes even to severe manual labour. Shortness of breath to this degree is one of the symptoms often complained of for years without marked increase; and sufferers from it may have become so habituated to it, as to be unconscious of a condition of dyspnœa which at once attracts the attention of the physician. Frequently, however, "shortness of breath" and "want of breath", as it is often expressed by patients, becomes a most distressing symptom. The sufferer may have to stop and pant a score of times in walking one hundred yards; he may be unable to lie down; and may have paroxysms of breathlessness without assignable cause. In this case, the derangement of the pulmonary circulation is greater and danger imminent. Habitual cough, persistent bronchitis with watery expectoration, occasional œdema of the lungs, are signs that the obstruction is having its effect on the pulmonary

structures, and premonition of what may be expected on the slightest occasion.

Commencing dropsy, which will most commonly be associated with, or preceded by, evidences of pulmonary obstruction, is a serious symptom, but of very different import in different cases. This, however, will be more fully entered into later.

It is not necessary to dwell on other symptoms, such as sleeplessness, or sleep broken by dreams, low spirits, anxiety, irritability, apprehension, which, while adding to the patient's sufferings and helping to wear out his strength, teach nothing in regard to prognosis not already known by surer signs. A careful study of the countenance cannot, however, be too strongly recommended. By certain indefinable tokens, with which we soon become familiar, we may often go in advance of more positive indications.

As to complications other than those in the lungs, whether secondary to the impeded circulation or originating in the organ affected, in the liver, gastro-intestinal tract, or kidneys, each must be set down as an unfavourable note. Most serious is renal disease, which at the same time deteriorates the blood, and imposes on the heart additional mechanical labour by obstruction in the systemic capillaries. The conjunction of cardiac and renal affections is most ominous.

If the existence of adhesion of the pericardium can be satisfactorily made out, it must be looked upon as adding to the danger of the case; but this will probably be indicated by symptoms of cardiac difficulty.

[*To be continued.*]

Reports of Societies.

OBSTETRICAL SOCIETY OF LONDON.

WEDNESDAY, JUNE 6TH, 1866.

ROBERT BARNES, M.D., President, in the Chair.

SEVEN gentlemen were elected Fellows of the above Society.

Specimen. Dr. GREENHALGH exhibited several specimens of medicated cotton wool, which had been recently made at his suggestion by Messrs. Bell and Co., of Oxford Street. He used them chiefly for application to the neck of the uterus and vagina. Being prepared with glycerine, they could be used much stronger, and applied for a longer period, and are much cleaner than the greasy pessaries and suppositories in ordinary use. The specimens exhibited consisted of cotton wool with iodine and iodide of potassium; with atropine; with morphia; with iron and morphia; with tannin; and with matico. He had pursued the following mode of application: a portion, about the size of a half-crown piece, secured by a piece of thread, is applied through the speculum to the affected part, over which a larger piece of cotton wool, similarly secured, and freely saturated in glycerine, is to be placed, and retained *in situ* from twelve to twenty-four hours, when it can be withdrawn by the threads either by the patient or practitioner.

Dr. WYNN WILLIAMS exhibited a specimen of a large abdominal cyst, which he had removed from an unmarried female, 40 years of age. The case had been supposed to be one of ovarian disease, and a month previous to the operation seventeen quarts of fluid had been withdrawn by tapping. Death unfortunately took place twenty-four hours after the removal of the mass; and a *post mortem* examination showed that the tumour had no pedicle, and was unconnected with the uterus or its appendages. The specimen being of an exceedingly interesting nature, it was referred to a committee for further investigation, and a report thereon.

Dr. SANSOM exhibited an Uterine and Vaginal Douche.

Dr. GRAILY HEWITT exhibited a specimen of Uterus during Menstruation; also a coloured drawing of the specimen while in a recent state. The specimen exhibited very perfectly the condition of the uterus during menstruation. One ovary contained a Graafian follicle quite recently ruptured. The subject of the case was a girl, aged 15, who died forty-eight hours after being severely burnt.

Dr. J. BRAXTON HICKS exhibited some Sticks of Anhydrous Sulphate of Zinc, which he recommended to the notice of the Society, having himself found them very useful and safe in the treatment of those conditions of the canal of the cervix uteri requiring styptics: such as produced cervical leucorrhœa, menorrhagia, etc. He considered that they were much more efficacious than fluid injections, because the stick could be allowed to remain in the canal, whereby a much more prolonged contact was obtained. They were made for him by Johnson and Sons, Basinghall Street, City.

MENSTRUATION IN PREGNANCY. BY GRAILY HEWITT, M.D.

The following case was related illustrative of the occurrence of menstruation in pregnancy, and as a contribution to the knowledge of this subject. A. B., aged upwards of 30, had several pregnancies. The last child was born June 23rd, 1865; suckled one month. The catamenia appeared from Sept. 15th to 25th; in October they were absent; on Nov. 7th she had a discharge of blood, with slight watery discharge, alternating for a week. Dec. 7th, she was "poorly," as usual, for six days. January 8th, 1866, she felt quickening. March 1st, pregnancy was distinctly diagnosticated. Delivery of a female child, apparently about a fortnight short of full time, took place on May 17th. The author considered it probable in this case that there was a twin conception, one ovum perishing and giving rise to the flooding observed in November. It might be that some other cases of apparent menstruation in pregnancy have a similar source; but in regard to the majority of the cases of menstruation in pregnancy, and excluding cases of irregular hæmorrhage, he believed the source of the blood to be the decidua vera, as in ordinary menstruations, the unusual condition in such cases being the absence of adhesion of the two membranes, the decidua vera and decidua reflexa. The decidual chamber may, in other words, persist to a later period than usual, in which case there is no difficulty in accounting for the exudation of blood from within it, and its appearance externally.

ON ANÆSTHESIA BY MIXED VAPOURS. BY ROBERT ELLIS, ESQ.

In opening this subject Mr. ELLIS said it would be taken for granted that the administration of mixed anæsthetic vapours possessed certain advantages over that of pure chloroform, counteracting the depression produced by the latter agent, and giving great security to the anæsthetic art. But the difficulty consisted in the due application of these vapours, and up to this time the anæsthetic fluids had been simply mixed together, and their resulting vapours administered. It was then shown that the whole theory of anæsthetic mixtures, and especially of those recommended by the Chloroform Committee, was based on an error; this being the idea that the vapours of each fluid would rise from the mixture in