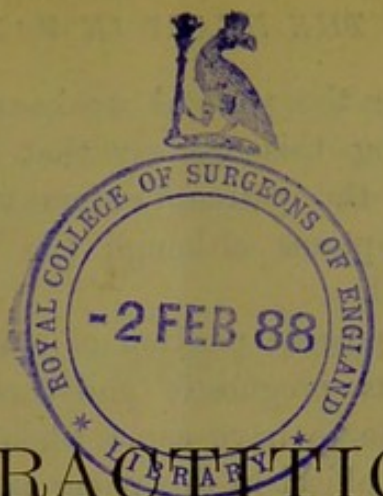
Failure of the heart in valvular disease / by J. Mitchell Bruce.



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FAILURE OF THE HEART IN VALVULAR DISEASE.

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It is only within the last few years that we have come to appreciate fully the significance of enlargement of the heart. Every thoughtful practitioner is now convinced, as the result of personal observation confirmed by high authority, that the subjects of valvular disease of the heart with enlargement may remain perfectly free from distress of every kind: that there are no symptoms attending *perfect compensation* of valvular lesions. We now understand that hypertrophy, whilst it is an indication of some disability or disease in connexion with the heart or vessels, is an indication also that the disability has been successfully removed, the disease practically cured by a process of natural recovery. We search anxiously for hypertrophy, we welcome its oncome, we work to establish and maintain it. On the same principle the appearance in such a case of symptoms of cardiac distress—of palpitation, dyspnœa, and dropsy—is now regarded as evidence of the occurrence of *failure of the heart*. We say that the "compensation has become imperfect," that the "hypertrophy has broken down." What were formerly described as the symptoms of valvular disease of the heart are now regarded as the symptoms of failure of the cardiac wall. By careful treatment we can

frequently remove the parietal weakness, and when we have succeeded in doing this we say that "compensation is re-established," and the patient enters upon a fresh period of freedom from symptoms although the valvular disease itself remains unchanged.

This being the present position of our knowledge of disease of the heart in its prognostic and therapeutical aspects, the time appears to me to have come when we can take another step forward. My object in this paper is to indicate one direction in which this advance may be attempted. The conviction has been forced upon me, not by theoretical considerations but by practical experience, that, after all, failure of the heart is only an effect behind which there lie a number of discoverable causes. If cardiac failure make its appearance in chronic quiescent valvular disease, it must have originated in some conditions or circumstances unfavourable to compensation; and prognosis and treatment to be sound and successful must be framed on a full knowledge of what these conditions or circumstances are. I do not refer to the necessity for a general acquaintance with the causes of broken compensation, but to the application of this knowledge *in each individual instance* of cardiac failure that comes before us. In my experience it is by no means common for the physician to enquire into the causes of cardiac failure in every case that seeks his advice. When a patient with palpitation, dyspnœa, cough, and threatening dropsy presents himself for treatment, I doubt whether we are always careful to put the question to ourselves —What has happened to this man that he should come to me with these symptoms after twenty years of freedom from suffering since the original rheumatic endocarditis? Time was when we were satisfied in such a case with the diagnosis—"mitral incompetence." We should now consider this diagnosis as insufficient, and would complete it by saying—"mitral incompetence with cardiac failure." I venture to say that this diagnosis is still short of the full truth, and that when we proceed to offer a prognosis based on such a conclusion only, and to apply treatment, we proceed on insufficient information. We must first determine the *cause of the failure*, why the heart has broken down,—whether from muscular strain, or nervous

exhaustion, or alcohol, or other discoverable cause. When we have settled this point to our satisfaction, and not till then, are we scientifically justified in offering a forecast or ordering a therapeutical course.

ÆTIOLOGY.

It is now a good many years since I began to make a careful note of the causes of failure of the heart in valvular disease. It has become a rule with me to ascertain in every instance, where full enquiry is possible, the whole circumstances under which my patient had been living when his heart symptoms made their appearance, and to discover which of these were faulty, or incompatible with healthy cardiac action carried on under the comparative disabilities of compensation. Until I have succeeded in this attempt in each case I have regarded my diagnosis as incomplete. The enquiry has proved a very interesting one, and instructive beyond expectation. I believe it has enabled me to be more accurate in prognosis, and it has certainly made me more confident if not more successful in treatment. I propose to enumerate some of the causes of broken compensation that have come under my observation, taking them in the order of the relative frequency of their occurrence. As we shall presently find, a combination of several causes is frequently at work, but first we will discuss them individually.

1. The most common and most readily appreciable of the conditions under which the compensated heart fails is *muscular overwork*. This statement applies especially to practice in hospitals, and amongst the poor in general. There is no difficulty in understanding the *modus operandi* of muscular failure or exhaustion in producing secondary dilatation of the heart. It is an obvious instance of the load rising perilously near the driving power of the muscle that has to move it to maintain the circulation. Every practitioner appreciates the importance of muscular strain as a factor in cardiac failure: he cautions his patient against it, and he commences treatment in urgent cases by ordering rest in bed.

2. The second place in relative frequency and importance in

the production of cardiac failure must be given to *nervous causes.* Of these the depressing emotions are the most serious —grief, distress, anxiety, fear, and hope deferred; but even excitement of a pleasing kind, such as joy and happy anticipation, may prove disastrous. One of the most frequent and trying of nervous conditions to the subject of chronic valvular disease is worry, in its many forms or phases. In other instances we find that the origin of broken compensation was severe and continued nervous strain or the burden of great responsibility —in the managers of large financial and mercantile establishments, in the successful lawyer, in the medical man in large and harassing practice. These and other nervous causes are so familiar to all who have seen many patients that I need not dwell upon them. The diagnosis in these cases is more obvious to the family practitioner than to the consultant: it involves a knowledge of the *man* as well as of the *disease* that we have to deal with. We shall presently find that they constitute a difficult class of cases as far as prognosis and treatment are concerned.

3. The next series of causes of cardiac failure to which I would draw attention are the different forms of *imperfect blood-supply* to the heart. As an active nervo-muscular organ, the heart requires to be abundantly fed with healthy blood by the coronary arteries, and any interference with the quantity or quality of the nutrient fluid tells with special readiness upon the parietal tissues in hypertrophy. Nutritive failure must be regarded as being present to some degree in every instance of rupture of compensation, even in those of nervous origin; but there are certain cases in which the blood or the blood-vessels are so manifestly at fault, and these only, that we should expect, and correctly, to find a coarse anatomical change in the muscular tissue—a granular or a fatty condition of the fibres. Two well-defined and distinct classes of cases present themselves to the practitioner in this connexion, and have to be carefully differentiated, particularly for the purpose of prognosis—the one an *impoverished condition of the blood*, the other a *diseased condition of the coronary arteries.*

(*a.*) *Impoverishment of the blood* is the key to a large number of cases of failure of the heart in valvular disease. Irrespective of the idiopathic anæmia of adolescence, the blood may be wasted

by hæmorrhages and discharges, starved by dyspepsia or actual want of nourishment. Compensation is a difficult—perhaps an impossible—process in the poor, under-fed, half-starved population of our large cities; or if compensation be established by treatment in hospital, it speedily breaks down. The coarse food of the poor generates flatulence and acidity, and the blood is poisoned with the products of dyspepsia.[1] These are amongst the cases of heart disease that frequent our out-patient rooms, or are repeatedly treated with less and less success on each occasion in our wards. The lot of the poverty-stricken cardiac patient is indeed a hopeless one.

Imperfect nutrition of the cardiac wall is at the bottom of failure in a class of patients at the opposite pole of the social sphere—the rich, the sedentary, the over-fed. Nothing is more common than to find the well-to-do subjects of valvular disease suffering from the symptoms of ventricular weakness. This is the effect of feeding their myocardium with blood that is over-rich, and laden with the products of disordered digestion, deranged liver-action, and imperfect elimination. The muscular tissue of the heart becomes fat and soft and pallid in such subjects; cardiac distress increases apace, and induces lassitude and indisposition to exercise; and the vicious circle widens and deepens by consequent increase of the assimilative and excretory derangements.

Under the same head I would mention cases of quiescent valvular diseases which have been treated with rest and nourishment to the extent of abuse. The unskilful practitioner, pursuing a dangerous routine in cardiac treatment, may have ordered for a dyspeptic patient, in whom he has unfortunately discovered a murmur, the strict avoidance of bodily exercise, an abundant supply of nutritious food and stimulants, and possibly tonics. The result is disastrous. The dyspeptic symptoms are aggravated; the compensation, previously sufficient, begins to yield; and the practitioner, who now feels confirmed in his diagnosis by the appearance of true cardiac symptoms, pursues his mistaken course with increased activity and more unhappy effect.

Yet another class of cases of the development of cardiac

[1] See an instructive case of this kind in Dr. King Chambers' book on *The Indigestions*, p. 21.

symptoms in valvular disease from impaired general nutrition requires to be noticed. I refer to those cases, which must be familiar to every practitioner of observation and experience, where persons with chronic valvular disease, after leading a life of activity and usefulness, have "retired" to enjoy "well-earned rest"—only, alas! to begin to suffer from symptoms of dilatation of the heart.

(*b.*) Disease of the *coronary arteries* I have mentioned as the second great cause of imperfect blood-supply. I have separated this local form of impaired cardiac nutrition from those forms just described under impoverishment of the blood, because I look upon it as perhaps the most hopeless of all the causes of failing compensation with which we have to reckon. I have lately met with several instances of this condition which have impressed me very strongly with its gravity. Two of the patients, who suffered from obscure symptoms and signs of aortic incompetence with failure, died suddenly. The *post-mortem* examination revealed almost complete obliteration of the mouths and first portions of the trunks of the coronary arteries from syphilitic disease, and extensive fatty degeneration of the ventricular walls. In a third case a powerful Guardsman broke down less suddenly with the symptoms of secondary dilatation of the heart—pulmonary complications and dropsy; and very similar lesions, also specific, were revealed at the necropsy. The aortic valves and arch were the seat of extensive syphilitic change; and the coronary arteries springing from it were involved in the morbid process, which had robbed them of the essential functions of nutrient vessels. I will return to these cases more particularly when I come to speak of prognosis.

4. According to my experience, one of the most anxious causes of failure of the heart in valvular lesions is the occurrence of *intercurrent disease*. This remark applies to all intercurrent disease of a serious kind, but there are two morbid conditions which I have learned to regard with special anxiety. These are rheumatism and lung disease. It will be well to devote a few words to each of these complications.

A cause of failure of compensation that we frequently meet with in young subjects—boys and girls from 12 to 16—of all ranks of life, is *rheumatism* of a subacute type. It would be

more correct to say that in many of these subjects the heart never perfectly recovers from the effects of the original rheumatism: compensation has not been thoroughly established when another rheumatic attack prostrates the patient, robbing him of muscular strength, of nervous energy, and of blood, and adding, it may be, to the primary damage by a fresh outburst of endo- or peri-carditis. The same happens, though less frequently, with adults. Many cases of failure or irritability of the heart, the origin of which is at first altogether obscure and puzzling, suddenly reveal their correct pathogeny by the appearance of pain in the joints.

Less frequent than rheumatism, but still far from uncommon, especially in adults, is the occurrence of acute *disease of the lungs.* In mitral disease, the most common form of chronic valvular lesion, the right ventricle mainly manages the compensation; and any considerable addition to the circulatory difficulties in the pulmonary circuit will seriously handicap the already labouring chamber. This is but another way of expressing the fact that in bronchitis and pneumonia our anxiety is always greatly increased by finding that our patient has a damaged heart, and for this reason chiefly—that we fear lest the heart, which we are careful to support in such a case by every available means, will break down under the excessive load that it has to drive. In the same connexion we can readily appreciate how *chronic* lung disease is also a standing menace to compensation. In chronic bronchitis, for instance, with local fibrosis, emphysema, and it may be bronchiectasis, affecting the subjects of mitral disease, the heart is very liable to fail under the influence of conditions that act primarily *on the lung.* In this way we may frequently account for the circumstance, which must have struck all of us, that certain of our chronic cardiac patients break down regularly with increased dyspnœa and the reappearance of dropsy at the advent of winter, and again possibly in the trying period of the early spring. In these patients it must be noted that the lung disease is not secondary to cardiac failure—a very likely diagnosis on the first occasion; but a perfectly independent disease, as a careful physical examination at repeated intervals and an intelligent consideration of the whole bearing of the case will serve to prove.

5. A highly instructive subject of study in connexion with failure of the heart in valvular disease are the causes *peculiar to women*. In one instance the occurrence of cardiac distress will be found to date from the first pregnancy. A second patient will have kept free from symptoms of heart disease until her confinement, whereupon pain, palpitation, and dyspnœa will have supervened. A third poor woman will resist these trying conditions, but will break down under the debilitating influence of protracted lactation. In a fourth instance the sexual climacteric will determine the onset of dilatation, especially if menorrhagia occur. In one sentence we may say that the risk of failure of the diseased heart is greatly increased in women on account of the difficulties and dangers attendant on menstruation, pregnancy, parturition, and the puerperal state, to which we must add abortion, lactation, and the period of sexual involution. These conditions manifestly act as complex causes. They disturb the nervous and circulatory systems, the blood, the digestion, the renal functions—indeed, they may be said to spare none of the physiological systems, ordinary or extraordinary, and through these they disorder the nervous and nutrient relations of the compensated heart.

6. A series of causes of failure of the heart for which we must never fail to enquire are the *cardiac poisons* in every-day use —tea, coffee, tobacco, and alcohol. The first three of these frequently occasion distressing sensations and palpitation during indulgence in them, or so shortly after as to connect them definitely with the attack, and their use is given up without much difficulty. It is different with alcohol. Its immediate effect is to increase the activity of the circulation and to relieve distress; whilst its abuse leads to serious impairment of cardiac nutrition. There are few more hopeless forms of cardiac dilatation than that which we encounter in the chronic drunkard—with debility, tremulousness, breathlessness, irregularity of pulse, dropsy, enlargement of the liver, broken sleep, and a peculiar chronic form of ill-developed delirium or slight dementia. Dr. Maguire has lately[1] drawn attention to the effect of alcohol in producing dilatation of the heart independently of valvular disease—an effect which we can easily understand when we

[1] *Lancet* (Report of Clinical Soc.), 1887, i. p. 1135.

consider the influence of the abuse of alcohol in undoing compensation.

7. The onset of symptoms in connexion with heart disease may sometimes be distinctly traced to *increase of the valvular lesion.* Occasionally the change may be moderately rapid—from fresh endocarditis. At other times it is sudden, and due to rupture of a diseased valve. I have met with a remarkable instance of this kind, in which a man with aortic incompetence, comparatively free from symptoms, one day suddenly became conscious during violent muscular effort of a loud murmur in his chest, and died in the course of a few months of steadily progressive dilatation of the heart. More commonly, however, the increase of the valvular lesion is slow and insidious, as in progressive mitral stenosis. Compensation may for a time keep pace with the lesion in the form of increasing hypertrophy, but it is clear that there is a limit to this process of adjustment; and after being repeatedly rescued from death by careful treatment, these patients must finally succumb to the very degree of anatomical change that characterizes their disease. It is obvious that the true nature of these cases when they come before us can be better appreciated by the regular medical attendant, who has had the patients under his observation for years, than it can possibly be by the consultant who probably sees them but on one occasion.

8. The last of the ordinary causes of failure of the heart in chronic valvular disease to which I would specially direct attention is *the limit of compensation.* In all or in most of our cardiac cases the time must come, sooner or later, when the full limit of compensation is reached. I do not refer to the limit of hypertrophy, for we know that enlargement may sometimes be enormous, until the heart reaches 40 oz.—double the weight it ordinarily attains in severe valvular disease. I speak now of the limit that is placed on life and health by the occurrence of secondary changes in the lungs, liver, kidneys, and indeed in the cardiac wall itself. Little by little, in all but the most favourable cases of valvular disease, the impaired circulation in the great viscera and nervous system, and the consequent functional disorders, sap the patient's nutrition. The very heart-substance itself, as was shown many years ago by Sir William Jenner, suffers from the

effects of coronary congestion, and becomes fibroid, œdematous, and feeble. Imperfect assimilation attends the digestive derangement consequent on portal congestion; imperfect oxygenation of the blood is the necessary result of the secondary pulmonary disorders; and the blood, thus impoverished and impure, is unfit to nourish the already enfeebled heart. This is the ordinary history of the cases of valvular disease of the heart that are so familiar to us in private and hospital practice under the name of "cardiac dropsy." Judicious treatment, applied in time and under favourable circumstances, may again and again secure for a time a fresh accommodation, a new adjustment of the physiological balance; but the end cannot be indefinitely averted—the limit of compensation is finally reached.

Combined or complex causes. I have discussed the foregoing causes of cardiac failure separately, as I proposed, but it is obvious that they are frequently associated. Over-work, underfeeding, and nervous depression go hand in hand; and we have seen that dyspepsia frequently complicates the ætiology under the same circumstances. The life, for instance, that many poor girls lead in London and other large cities brings into play, in such of them as are the subjects of chronic valvular disease, several of the causes of failure already mentioned. They work from morning till night in rooms which are often over-crowded, overheated, and badly ventilated. They are fed indifferently, and must take "what is set before them." They are badly paid for their work, and can have but few pleasures; and the social and moral atmosphere in which they live is often far from wholesome. Muscular exhaustion, dyspepsia, anæmia, menstrual irregularity, nervous depression alternating with morbid excitement, are the results of this life—a life that quickly tells upon the compensated heart.

Allied in a measure to this class of case is the failing heart of the *debauchee*—the intemperate, the dissipated. The causes of break-down here are even more complex. Irregular hours, disregard of proper diet, nervous excitement followed by intense depression, careless exposure to cold and wet, and, beyond these, indulgence in alcohol and tobacco—all combine to ruin compensation.

Prognosis.

The whole aspect of cardiac prognosis is manifestly changed by the preceding considerations. The first point that becomes clear to us is that there are two distinct and different directions in which prognosis has to be applied, namely, first as regards the valvular disease, and secondly as regards compensation. The prognosis of *valvular disease* has to be distinguished from the prognosis of *cardiac failure*. The former, the prognosis of "heart disease" as it is generally understood, is chiefly concerned with the particular valve affected: it teaches us, for instance, that aortic incompetence is a more serious lesion than aortic obstruction: it is mainly statistical in its method, and general in its application. The latter, the prognosis of cardiac failure, is concerned with the *cause* of failure: it teaches us, for instance, that aortic incompetence in a drunkard is a far more unfavourable condition than aortic incompetence in an overworked but temperate man: it is specific, personal, immediate in its application. I am afraid this distinction is too often forgotten. When his opinion is required of the prospect of a case of cardiac disease with symptoms of failure, the practitioner begins to prognose with a few words of general encouragement. He is most careful to dispel from the minds of the patient and the patient's friends the popular mistake that a diagnosis of heart disease is tantamount to a death-warrant. He says that "cases often recover and keep well": that rest and tonics will do much to restore the heart. But his reassurance does not take a proper form. Instead of saying, "These symptoms have come on because such and such a circumstance which I have discovered (nervous, toxic, nutritive, &c.) has interrupted the healthy action of the heart, and they may be expected to disappear if you are more careful and more fortunate in future," he proceeds to employ what I have called the statistical method —to apply to the case before him the results that have been arrived at in Assurance Societies with respect to the comparative value of life in the different forms of mitral and aortic disease. In following this plan the practitioner has made a great omission. He has thought too much of the "disease,"

too little of the *individual* before him. He has allowed himself to neglect in great measure the *elements of prognosis.* He has overlooked the guiding principle that to make a correct forecast we must know the *causes* that are at work. Before he can say what the probable length of life in a case of mitral disease will be, he must make himself acquainted with the conditions under which that life has been and is to be spent. I trust I have succeeded in making my opinion clear that successful prognosis in individual cases of disease of the heart (apart from the general rules laid down in connexion with the different forms of valvular lesion) depends on a *correct diagnosis of the cause of failure.*

If the cause can be accurately ascertained, prognosis ought not to be difficult in most cases. We find, let us suppose, that a patient with mitral incompetence has been grossly *overworked* before or at the time of the compensatory failure. We can discover no other cause for this the first occurrence of dropsy of the legs with dyspnœa and some bloody expectoration. In such a case we may promise with some confidence that a thorough rest and careful treatment otherwise will restore the man to his former health, and that if he can confine his work in future within reasonable limits he will keep indefinitely well.

We may sometimes venture to give a similar prognosis in the second class of cases to which I referred under ætiology—cases of cardiac failure from purely *nervous* causes—from grief, anxiety, worry, protracted distress, or unhealthy nervous excitement. But all of us who have had much practical acquaintance with this class of cases, whether in heart disease or in other connexions, know how cautiously we must forecast their future. We cannot compel a patient's feelings, however completely we may control his body. Rest in bed will not ensure rest from worry or other form of depressing emotion. The very fact of the appearance—perhaps the first discovery—of disease of the heart may drive such a patient into his grave. In every instance of heart disease one must be cautious in expressing one's opinion to the patient himself, but in such a case as this the rule is absolute—only a cheering prognosis will hold the patient together. What then shall be our forecast to the friends in a nervous case? We must try to ascertain the patient's dis-

position very closely. Was the nervous incident under which the compensation failed of the nature of a sudden shock ? or has his heart been broken by life-long cares in connexion with his family or his business ? We should take a very different view of the prognosis of a mitral lesion in these two instances. Equally cautious should we be in giving an assurance of recovery and return to indefinite compensation in a business man of fifty coming to us with a complaint of occasional attacks of what he calls "giddiness," but which we discover to be true faintness, with failing pulse, an irregular heart, an anxious countenance of unhealthy hue and dusky lips. These symptoms are often bad enough in themselves, but their seriousness is greatly increased by the fact that we cannot get the patient to submit to rational treatment.

The third group of cases which I discussed under ætiology are those in which compensation has broken down, and symptoms of so-called heart disease have made their appearance, in consequence of *interference with the blood-supply*. The prognosis in these subjects will be very different according as we refer the case to one or to another set of causes. If the nutrition of the heart have failed from *general* anæmia, the result for instance of menorrhagia, we may, as a rule, give an encouraging opinion, for with judicious treatment the blood will be restored and the muscle will regain its firmness and its power. Only the wasting drain must be checked ; for if hæmorrhage be excessive, or if it continue in spite of treatment, the patient may die, and then, as in a case recorded by Dr. Henry Green,[1] we shall find the heart in a state of acute fatty degeneration. All this we must take into account. In the same way we must reckon with anæmia from want or chronic dyspepsia, leucorrhœa, &c.

A class of cases of cardiac failure in heart disease which may bring us much satisfaction and no little credit professionally, are those in which the nutrition of the heart has become impaired in consequence of unsuccessful or *misapplied treatment*. Such are, for instance, the cases treated with injudicious rest, with excess of tonics, food, and stimulants—in a word, the cases treated in a routine fashion without rational principles as a guide. These are the instances in which we may be able to promise a fresh

[1] *Clin. Soc. Transactions*, vol. viii.

lease of life to a man condemned to death by a less skilful practitioner.

If, on the other hand, the nutrition of the heart have failed from purely *local* causes, from interference with the coronary circulation, we have to deal with one of the most severe, perhaps the most hopeless, class, as I have already said, of all these cases. When we meet with cases of this type, we must think well before we offer a prognosis. The burden of this paper is, I trust, a hopeful prognosis with successful treatment, but the very foundation of confident treatment consists in the recognition and elimination of cases beyond natural recovery or the succour of art. Let us see how we are to recognise these cases. First, we shall do wisely to suspect all cases of aortic incompetence that come to us with symptoms of failure indicative of active dilatation of the left ventricle and the left auriculo-ventricular opening, *i.e.* where a patient with aortic disease presents "mitral" symptoms — dyspnœa, pulmonary catarrh, hepatic enlargement, and dropsy. Secondly, aortic disease is also more unfavourable when non-rheumatic in origin: when due to degeneration, to syphilis, to injury. Degeneration is obviously bad. Syphilis involves both coronaries and cardiac tissue, though, on the other hand, if early recognised, it may yield marvellously to iodide of potassium. Injury is serious, because it may have torn the valve into shreds which beat against the sinuses of Valsalva and the mouths of the coronaries, and hopelessly ruin the local nutrition. Thirdly, aortic disease is very unfavourable when none of the ordinary causes of failure that we have discussed can be discovered. If an aortic case breaks down under favourable, reasonable, well-ordered conditions of life and work, local organic disease of an active, progressive, and serious kind is to be feared.

With regard to that important group of cases in which *intercurrent disease* handicaps the heart and destroys compensation, it is impossible to offer an encouraging prognosis in the two affections which I specially discussed—rheumatism and acute pulmonary disease. It is a common experience in practice that the form of subacute rheumatism which haunts the joints and heart in children and adolescents frequently proves fatal by exhausting the capacity of recovery. The heart is incessantly

worried by the rheumatic process, and frequently grows to an enormous size, with endo- and exo- and possibly myo-cardial inflammation and degeneration, until it reaches limits incompatible with recovery. It may be difficult to state the chances of recovery in these young persons, who sometimes improve very remarkably, and in whom the tendency to rheumatism will disappear if life be maintained for a few years. But I feel that I am right to caution the practitioner against making light of rheumatic attacks, however trifling they may appear, which persist or return at short intervals.

In acute pulmonary disease, especially bronchitis and pneumonia, the danger is great and urgent, and it comes from two sides: the two diseases mutually aggravate each other. We know that by far the most common cause of death in acute rheumatism with cardiac complication is acute lung disease. So in chronic quiescent heart disease, such as mitral obstruction, the supervention of bronchitis is very likely to be fatal. I have already said that this is a common type of disease in winter. The prognosis must then be stated to be in the first place grave, and in the second place dependent on the weather —a fine day or a fog in the month of November may mean life or death to such a cardiac patient. At the same time we must be careful not to be misled in these cases by the apparent completeness of the break-down. It is acute, but it is transient if the lung be relieved, and in a few days the right ventricle may regain its vigour.

The prognosis is frequently difficult to estimate in those cases which I mentioned in connexion with the *sexual function in women* —at puberty, at the climacteric, in pregnancy, after parturition, and from superlactation. Nervous and nutritive causes of much complexity are here at work, and we must use our best judgment in each individual instance to unravel them and attach to each its proper import. Menstrual cases are comparatively favourable, and so are cases of superlactation. But in pregnancy and *post partum* one has to forecast with great caution, as septic causes may be at work, and the disturbances of the nervous system and of general nutrition are also far more profound.

With regard to the prognosis of cardiac failure from *toxic* causes I need say but little—the success of the prognosis will

mainly depend on the correctness of the diagnosis. The habit of indulgence in tobacco and tea may not be difficult to break. Alcoholism is but exceptionally curable. Careful general consideration is demanded to estimate accurately the influence of gouty poison and of the products of dyspepsia on the prognosis of heart disease.

A cause of cardiac failure to which I have referred is the acute exacerbation, aggravation, or *increase of the lesion*, for instance by rupture. Such an event may instantly convert a favourable into an unfavourable case. Equally serious, though less striking, may be the effect of a fresh endocarditis in old-standing valvular disease. We shall do well to be cautious in expressing our opinion in such a case until we have learned how far compensation can be re-established under the new conditions.

We now come to another hopeless class of cases—those cases in which failure occurs from very chronicity—from compensation having reached its *limit*. The best evidence of this state of affairs is a clear history—or our personal knowledge—of repeated and ever less easily remediable failure of compensation: of recurrent dropsy and visceral involvement after the successful application of rational treatment. But the same tale is told by the result of a careful examination—by the anxious expression; by the complexion, which is sallow, livid, and jaundiced; by the suffused weary eyes; by the persistently dropsical legs; by the impaired resonance over the bases of the lungs, and other signs and symptoms of protracted pulmonary congestion; by the enlarged, indurated, tender liver; by the scanty, loaded, albuminous urine; by the feeble, helpless, wandering condition of the mind, which indicates passive cerebral congestion and poisoning by carbonic acid. These phenomena, in association with complete asystole of the heart, and a pulse that is scarcely perceptible, constitute the main signs of the approaching end of a long and brave struggle, in which the forces of compensation are being defeated by slow and steady sap instead of sudden assault. But let it be noted that even in these desperate-looking circumstances hope is not to be abandoned, unless it is certainly known that the previous treatment of the case has been conducted on thoroughly sound principles.

Those complex cases of failure which I referred to *city life* ought, if taken sufficiently early, to do well, at any rate for the time, and a favourable prognosis may be offered accordingly. It is different with the cardiac failure in the *dissipated.* Here the causes are practically irremediable, and the wretched subject is frequently beyond hope, even if his habits are controlled in hospital.

If we now review what has been said with respect to prognosis, we shall find that the forecast that has been made of a case of failure of the heart, as distinguished from valvular disease, may be either: (1) *favourable;* (2) *unfavourable*; or (3) *doubtful*, according to the conditions under which compensation has given way.

(1) Prognosis is comparatively favourable in cases due to—(*a*) *muscular overwork*, unless there be definite evidence of sudden aggravation of symptoms from strain, when the case may be most serious. (*b*) Failure of the cardiac wall from *impaired general nutrition* (poverty, anæmia, dyspepsia, &c.); but severe hæmorrhage may thus prove fatal. (*c*) *toxic causes*—except alcoholism. (*d*) Previously *misapplied treatment*—routine treatment, especially by rest and tonics irrespective of cause.

Briefly, the prognosis of cardiac failure may be said to be favourable when the cause is removable.

(2) Prognosis is comparatively unfavourable in cases of failure due to—(*a*) impaired *local* nutrition, *i.e.* vascular disease (disease of the root of the aorta and coronaries, syphilitic, degenerative, &c.) (*b*) *Intercurrent rheumatism*, in young subjects especially; and intercurrent *acute pulmonary disease.* (*c*) The *limit of compensation* having been reached.

Briefly, the prognosis of cardiac failure may be said to be unfavourable when the cause is irremovable.

(3) Prognosis is uncertain and obscure in cases of failure due to—(*a*) *Nervous* causes, which are often beyond our control. (*b*) *Aggravation* of the original valvular lesion—the prognosis depending on the extent of aggravation and on fresh compensation. (*c*) *Climacteric* causes; especially uncertain in pregnancy and *post partum.* (*d*) *Undiscoverable* causes.

Treatment.

Little space remains for the discussion of the question of the treatment of cardiac failure. But if the view of the subject that I have presented in this paper be correct, the general principles of treatment of heart disease can be briefly stated. Here, as elsewhere, sound treatment rests on correct pathology and mainly on correct ætiology.

The first principle I would formulate in these words: Do not treat cardiac disease without sufficient evidence that treatment directed to the heart is required. Let us be sure that the heart is failing before we proceed to apply our remedies. The day has gone by when men with any pretension to therapeutical judgment did not hesitate to "treat murmurs"—when they ordered digitalis and iron, let us say, for a mitral *bruit* which they happened to discover in a dyspeptic patient.

The second principle is equally clear. If the heart is really failing and treatment called for, *do not apply treatment in a routine fashion.* Cardiac failure is but an effect; rational treatment begins with attention to the cause. We must discover the cause of the break-down, and remove it if possible. Our means of treatment will vary widely in different cases. We have an extensive choice of remedies. We are not confined to a few routine methods or a few habitual drugs, but can turn to account every means hygienic and therapeutic that is suggested by faithful investigation and sound judgment. This is a very encouraging consideration. The poor overworked, half-starved labourer must be ordered rest and warmth and food in hospital. The sedentary free-liver requires the very opposite line of treatment—he must be sent to climb mountains or to hunt, to endure for a time the miseries of palpitation and breathlessness, if compensation is to be successfully restored. We treat intercurrent rheumatism with remedies, such as salicylates, calculated to quickly dispel it. We interdict the consumption of cardiac poisons. We remove the overworked, ill-fed, anæmic girl from business in town to the country.

It cannot be denied that it is difficult and laborious to carry out this system of arranging and ordering treatment—at least

when we first attempt it. It is so much shorter and simpler to follow what I may venture to call our present system—to sit down and write a matter-of-course prescription for digitalis, and give a few familiar cautions against exertion and excitement. The former plan is, as I have said of prognosis, personal, immediate, and specific, and it therefore involves the trouble of making ourselves acquainted with the whole conditions of the patient's life: the latter plan, being of general application, involves little or no personal enquiry. I will leave it to the reader to judge for himself which of the two plans of treatment he will prefer as being the thoroughly sound one. I can assure him that the rational plan is the more successful. Having raised and answered the question as to the cause of failure, he proceeds to order treatment. He has discovered that his patient suffers from menorrhagia dependent on polypus; he removes the polypus. He has found that what has been regarded as a secondary lung complication in the case is really an independent bronchial catarrh, which calls for special climatic treatment before the heart can be relieved; he sends the patient to winter on the Riviera. Or, again, his careful enquiries have elicited the all-important information that his patient is the victim of the alcoholic habit; whereupon he attempts, at least, the removal of the toxic cause of cardiac malnutrition. These are but a few illustrations of the application of what is in my opinion the second great principle to be observed in the treatment of disease of the heart. I trust they will suffice to indicate that it is thoroughly rational and successful in its working.

The third principle that ought to guide us, and the last that I shall mention here, is the complement of the second. It is this: If the cause of cardiac failure be undiscoverable, or irremovable, do not hesitate to treat the effects. The attempt to discover and remove the cause must not be carried too far As the end approaches in most instances of cardiac disease, when dilatation and dropsy and general visceral involvement have reached their limits, immediate relief of symptoms is our paramount duty. The same will be the case when symptoms are urgent and life is threatened at any stage, however early, in heart disease. In the face of urgent distress and impending

death, it would be unjustifiable, and in the highest degree unscientific, to begin to talk of causes. The effects—the pulmonary congestion, the hydrothorax, the choked condition of the right heart and veins—are threatening life, and must be unhesitatingly and immediately dealt with. Even in less urgent cases the same principle has so far to be pursued. The cause cannot always be immediately or completely removed. Nutrition cannot be restored all at once; toxic effects take time to pass off; intercurrent disease may have to run a course; and in the more complex cases of nervous and nutritive break-down it may take many months to restore healthy surroundings to the patient.

The therapeutic methods that are called for under these circumstances require only to be mentioned. We reduce the *load* that the enfeebled ventricle has to carry—directly by means of bleeding (venesection, cupping, leeching), indirectly by paracentesis, puncturing the legs, and evacuation of the bowels. We reduce the *resistance* ahead that the ventricle has got to overcome in systole, by means of nitrite of amyl, nitro-glycerine, alcohol, direct renal diuretics, and purgatives. We increase the nervo-muscular *force* of the heart, that is the driving power, by ordering ether, ammonia, and strychnine, and by allowing an abundant supply of well-digested and assimilated food. Pursuing one or all of these methods of treatment we may quickly succeed in restoring the balance of the circulation.

When we can act with more deliberation, we order specific cardiac and circulatory agents—digitalis, strophanthus, convallaria—which will increase the force of the ventricular contraction, will afford the auricles more time to fill and the ventricles more time to rest, and will promote diuresis. These drugs are invaluable remedies—so valuable indeed are they that their employment constantly suggests itself to the practitioner, not only in cardiac failure, but, as I have already observed, in cases of compensated valvular disease. Use then becomes abuse. In my opinion we shall never come to understand the proper employment of these powerful measures until we take the trouble to ascertain in *each individual case* the circumstances of the cardiac failure for which they are prescribed.