it is associated, the patients seem conscious that the pain, even when slight, is of serious import. The emotional side of heart disease is a subject upon which much might be said. Cardiac pain may be regarded as the outcry of a weak or overtaxed heart. It expresses the struggle of an over-distended heart to empty itself. Consequently dilatation will be the factor common to them all. The struggle will be greatest and the resulting pain most severe in those parts of the heart where the muscles are biggest. The right auricle may be very greatly distended with little more than discomfort. Pain will be more marked where the right ventricle is concerned, but all the most severe attacks of pain are associated with the left heart, and especially with the left ventricle. One other factor must be borne in mind. It is this, that the call upon the heart and the consequent distension must be more or less sudden—gradual distension may occur without pain, and reach even a higher degree; but sudden distension leads to an attack of pain. This, of course, is a matter of daily experience in left ventricle affections. The first attack of angina may follow exertion, as in the case I described above. Afterwards an attack may be produced by very slight effort, even that of walking a short distance fast or uphill.

The nature and cause of angina has been much discussed, but I think all we really know about it may be summed up in the term "cardiac colic." I do not know of anything observed in angina which cannot be seen in other forms of colic. The pallor, faintness or collapse, the small arteries, dilated pupils, and various conditions supposed to stand in some causal relation with angina may all be equally seen in biliary, renal, and intestinal colic.

The pain of a distended urinary bladder or of a pregnant uterus at the commencement of parturition is strictly comparable with the pain of angina. In all cases alike it is the struggle of the muscular organ to get rid of its contents, and the spasmodic contraction of the muscles is attended with the paroxysms of pain. As soon as the organ has emptied itself the pain ceases. If it cannot get rid of its contents, the paroxysms become less and less severe as the muscles become feeble, and finally a condition of paralytic distension develops in which the pains cease. So it is also in the heart, the most violent attacks of angina occur generally when the heart muscle is still fairly strong. As the heart fails the attacks may become less severe, and when extreme dilatation occurs and the other signs of cardiac failure appear, the tendency to severe angina disappears.

The term "cardiac colic" removes angina from the little niche in which it stood all by itself, and places it in the general group of colic about which we know a good deal, though much remains still to be learnt.

REFERENCE.

¹ Arch. f. Ger. Physiol., 1904, lvii, 607.

A Lecture

ON

CONGENITAL HEART DISEASE.

DELIVERED AT THE HOSPITAL FOR SICK CHILDREN,
GREAT ORMOND STREET,

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LADIES AND GENTLEMEN,—There is much that might be said upon this subject which would interest the pathologist and embryologist, but the time at our disposal compels me to restrict myself to the more purely practical side. We can readily group the morbid changes in congenital heart disease under two headings:

1. Those due to malformation.
2. Those due to intrauterine endocarditis.

The first group can then for convenience sake be classified into three subgroups:

Subgroup I.—Arrest of development in early fetal life: Such results follow as the formation of hearts with two chambers only, a ventricle and an auricle; or possibly there may be two auricles and one ventricle. These conditions are incompatible with life.

Subgroup II.—Arrest later in fetal life: In such cases the auricles and ventricles though developed are imperfect, and the aorta and pulmonary artery may be partially developed or the large vessels displaced.

Subgroup III.—Defects at the end of fetal life; for instance, premature closure of the foramen ovale, or premature obliteration of the ductus arteriosus.

The second group of cases—those resulting from intrauterine inflammation—are of more interest, and in this group are to be found remarkable examples of fetal endocarditis, probably rheumatic in origin, of which the following case from the Museum Catalogue of University College Hospital is a striking instance:

In a child who lived for thirty days there was discovered the day after birth a loud systolic murmur which was heard all over the precordium and at the back. It was a small, quiet, and pale infant, but, except for occasional blueness of the lower eyelids, showed no cyanosis. The mother had suffered twice from rheumatic fever, and was attacked a third time during her pregnancy. Her three other children were healthy. The necropsy showed that the interventricular septum was deficient at the upper and back part, and to the margins of the aperture the cusps of the mitral and one cusp of the tricuspid valve were adherent. Both valves were beaded by vegetations. The foramen ovale was patent, as also the ductus arteriosus. The other valves were natural.

Another striking example which came under my own observation was that of a child born cyanosed and in an almost asphyxiated condition. Death followed on the third day. During pregnancy the mother had suffered from a severe attack of rheumatic fever. The necropsy showed extensive and recent mitral endocarditis.

The diagnosis of this form of heart disease has been made even in intrauterine life, as in those cases reported by Peters and others in which a murmur has been detected in the fetal heart.

In the second group, then, the important lesions are connected with fetal endocarditis and consequent anomalies of the valves and cardiac septa.

The relation of congenital heart disease to a family history of rheumatism is a point of some interest, and I have come upon several examples of this occurrence.

CASE I.—In 1905, a boy, aged 2, was brought here with congenital heart disease, and a history of rheumatic arthritis in pregnancy was given by the mother.

CASE II.—E. D., aged 9 months, suffered from congenital heart disease, the mother from rheumatic arthritis, and the maternal grandmother died from heart disease consequent on rheumatic fever.

CASE III.—An infant of 2 years with congenital heart disease, whose maternal grandmother suffered from severe rheumatic fever, and his uncle also from rheumatic fever.

CASE IV.—H. S., aged 3 years. Congenital morbus cordis. Maternal grandfather died of rheumatic fever with heart disease. The mother of the child had five married sisters, each with a child who was cyanosed on exertion and short of breath.

CASE V.—G. R., aged 2 months. Congenital heart disease. His mother had repeatedly suffered from swollen joints.

These cases, with the two I have alluded to more fully, will serve as a reminder of the importance of the rheumatic infection in intrauterine life. Congenital syphilis is also a cause of congenital cardiac disease, although, as it so happens, I have rarely met with a convincing example of its occurrence.

The most important clinical cases in this group are those in which there is narrowing of the pulmonary artery. This is the form of congenital heart disease in which life is more likely to be prolonged to adult age than in any other.

There is frequently associated with this stenosis a patent interventricular septum. This opening is, as a rule, a small one at the upper part of the ventricular septum, in the membranous undefended space. In addition, the foramen ovale may be patent and the ductus arteriosus incompletely closed.

Stenosis of the aortic or mitral valves are also met with occasionally, and the tricuspid valve may be damaged, but such conditions are far less common than the pulmonary lesion.

The symptoms of congenital heart disease, where they are well marked, are remarkable ones, but in infancy they are sometimes somewhat indefinite. The most frequent of all manifestations, cyanosis, usually attracts attention at birth, but it would be an error to suppose that infants

by any means always show the cyanosis. Prominent and important though the symptom is, I think it is a little overrated, and if you rely upon it too much you will certainly be deceived. These infants may be brought because they are listless, small, and puny, and fail to make progress. Unless, then, a routine examination of the chest is made, the true explanation of these symptoms may not be discovered.

Here is a case in point. This female child of 18 months, who is not cyanosed, but looks pale and delicate, was brought to the out-patient department at 9 months for wasting and difficulties with the chest. Examination of the chest discovered a loud systolic murmur with its maximum intensity over the pulmonary artery and audible over the entire precordial area. In this case there is very considerable enlargement of the spleen, but the character of the murmur and condition of the heart are not explicable as a hæmic condition.

The cyanosis in congenital heart disease implicates the skin and the mucous membrane, and when it is extreme reaches a mulberry hue. The explanation of its occurrence has not been agreed upon. Some have attached much importance to the mixture of venous and arterial blood, others to deficient aëration of the blood, and of late a good deal of attention has been directed to the increase in the number of the red blood corpuscles. This is not a condition peculiar to this form of heart disease. A cyanotic condition, for example, is met with in some adults who have no morbus cordis, but an enlarged spleen. It is, however, very striking in some of these cardiac cases, and was commented upon originally, I believe, by Toiniessen. Baunholtz's case gave a count of 9,447,000 per c.mm., a specific gravity of 1071, and a percentage of hæmoglobin of 160. There is an increase in the number of red blood corpuscles, also a concentration of the blood itself, which tends to make its passage in the vessels more difficult.

Symptoms of great importance are laboured respiration, paroxysms of disordered breathing, with unconsciousness and epileptiform attacks. These attacks are sometimes prolonged and most dangerous. Their interest too is very great, for it is not at all easy to understand what is happening when you find a child lying, sometimes for hours, unconscious with extreme tachycardia. In rare cases angina pectoris has been recorded. You will find occasionally that the child is brought to you because the mother has been struck by the rapid tumultuous action of the heart.

Clubbing of the fingers, toes, and nose are also important results of congenital heart disease. This is a later symptom, and generally very obvious in those children who are greatly cyanosed. Lastly, cold extremities and a subnormal temperature are frequently observed.

Examination of the chest will generally settle the question of the diagnosis. The heart is usually found to be slightly enlarged, and there may be sometimes bulging of the precordial region. On auscultation a loud systolic murmur is audible over the precordium, with its point of maximum intensity over the pulmonary artery immediately to the left of the sternum. This bruit is harsh, and over it there is frequently a thrill to be felt by the hand. The pulse on slight exertion may become feeble and irregular. This child illustrates a characteristic case of this type:

H. P., aged 5, has never been strong from birth, and at 2 years of age a doctor discovered a malformation of the heart. He is cyanosed, and the fingers and nose are clubbed. The right side of the heart is hypertrophied, and in the second left intercostal space, close to the sternum, there is a loud systolic murmur, which I attribute to the narrowing of the pulmonary artery. Over that murmur there is a definite systolic thrill. There is also behind the sternum, at the level of the fourth intercostal space, a loud superficial systolic murmur, which I believe to be evidence of a patent interventricular septum. This boy has found that when he runs he suffers from pain over the heart, but his most alarming symptoms were fainting attacks, accompanied by severe pain in the head and abdomen. His disposition is very excitable. He is also small, and obviously delicate.

Not all basal congenital bruits are loud; they are sometimes soft and whiffing, and then should the child cry they are easily overlooked. Cases more puzzling still are those in which during the first few weeks there is no audible bruit, although there are the symptoms of congenital heart

disease, and which later develop a loud bruit, with a diminution in the urgency of the symptoms.

The bruits of congenital heart disease, again, are not always basal or heard at their loudest over the base of the heart to the left of the sternum. This case illustrates the occurrence of an apical murmur of congenital origin.

L. M., aged 2 years and 4 months, was discovered at birth, by the doctor in attendance, to have a damaged heart. Since then he has been subject to attacks of bronchitis and pneumonia. His aspect is pale rather than cyanotic, but the superficial veins are more obvious than usual, and the nose slightly clubbed. There is a double murmur over the cardiac impulse, in part systolic and in part presystolic. I believe there is a congenital mitral lesion. On the maternal side there is a strong rheumatic history.

In some cases a diastolic murmur is discovered, and again there may be a continuous humming bruit, lasting through the whole cardiac cycle, rising and falling with the systole and diastole. The maximum intensity of this bruit is in the second left intercostal space, and in that position also the pulmonary second sound may be curiously loud and low-pitched.

The interpretation of such a bruit as this is that the ductus arteriosus which, under normal circumstances, closes in the first fortnight of life, has remained patent.

Thus, this boy, aged 6 years, was brought to me in May with physical signs pointing to multiple bronchiectasis at the base of the left lung, and this curious condition of the heart. If the hand is placed over the upper part of the sternum a well-marked systolic thrill is felt, and on listening in the second left intercostal space a loud murmur is audible, occupying the whole cardiac cycle and rising in intensity with each systole of the heart; there is also a loud low-pitched pulmonary second sound. This boy is not cyanosed, but he feels the cold and becomes blue on exposure to cold. The fingers are not clubbed. He is small and undergrown. The condition of the lung has much improved with treatment, but there is of course the evidence of the bronchiectasis still present, and I fancy these are the result of a congenital condition also.

The explanation of the cardiac signs I believe to be this: The long, loud murmur occupying the whole cardiac cycle is the result of a patent ductus arteriosus, and the curiously loud, low-pitched second sound may be due to some dilatations of this patent ductus, the second sound having the character of the aortic second sound in cases of aneurysm of the aorta.

The only other condition in which I have met with a peculiar murmur like this has been in a man who had an aneurysm of the aorta which had ruptured into the pulmonary artery.

In 1904 I had a little girl under my care who had suffered severely from an attack of influenza, which had produced dilatation of the heart with dangerous attacks of syncope. This child, I found, had, in addition to an apical systolic bruit, which I ascribed to the influenzal dilatation, a murmur similar to the one in the case of this boy—namely, a loud murmur, rising and falling with systole, occupying the entire cardiac cycle, and at its maximum in the second left intercostal space. The pulmonary second sound was loud and clanging. There was no clubbing or cyanosis. This child, after a prolonged illness, recovered; the apical murmur disappeared, but the congenital bruit remained unaltered.

The degree of cardiac hypertrophy in congenital heart disease varies very considerably, and if one had to formulate any general rule it would, I think, be that the enlargement is remarkably slight, although in exceptional cases the cardiac dullness may be extraordinarily increased.

Arrest of physical development is a prominent feature in some cases, and two little girls recently under my care illustrated this admirably. One of them in particular resembled a doll in the smallness of her features, bones, and muscles. In other cases, as Dr. A. E. Garrod has pointed out, the malformation of the heart is associated with the Mongolian type of imbecility. This is a very serious matter, for such children are not able to earn a living.

The older children, as well as infants, feel the cold very much, and prefer to sit hugging the fire rather than warm themselves by exertion, which makes them short of breath. One such patient had a weakness for hot beer which his father, a publican, prescribed for him on his own responsibility.

The lives of these children are very precarious. The majority die when under 2 years of age. And sometimes death occurs suddenly, an occurrence so distressing that it should always be borne in mind. Bronchitis may result from slight exposure to cold, and an attack of gastro-enteritis or of measles or other infectious disease may prove rapidly fatal. Puberty throws a strain upon the older children, and I think the well-known danger of tuberculosis is more apt to attack these children after puberty than before. It has been my experience to find that between the ages of 2 and 12 years an attack of endocarditis has been more frequent than tuberculosis, and in several cases the type of endocarditis has been malignant.

Tuberculosis of the lung is not always fatal in these children, and the following case is one of singular interest as an illustration:

A boy of 4 years came here in 1901 exceedingly cyanotic and with a troublesome cough. I found every evidence of a classical pulmonary stenosis and recent tuberculosis of the upper lobe of the left lung. The disease slowly progressed until a cavity formed. Then the process arrested, and two years afterwards there were very obvious signs of a quiescent cavity in this lobe. Three months later he was brought again with high fever, dyspnoea, and intense cyanosis. A diagnosis of acute pulmonary tuberculosis was made. After a brief illness the child died, and at the necropsy a small quiescent cavity was discovered in the left lung, and the upper lobe was shrunken, but there was no trace of recent tuberculosis. On the contrary, the cause of death was malignant endocarditis grafted on the pulmonary stenosis.

Here there is proof that tuberculosis of the lung may be recovered from even when it attacks a small child suffering from severe congenital heart disease. Had we treated this child according to the recent method of vaccination, what a good case we should have made for that method of treatment!

The development of endocarditis is a complication which it is not by any means always easy to detect. Yet its occurrence is most serious, for it is liable to be malignant in type. The difficulty of the diagnosis will be readily understood when it is remembered that there is already a loud murmur present due to the malformation. In some cases the endocarditis is evidently the result of acquired rheumatism, for the endocarditis is associated with pericarditis and arthritis, and even nodules. In other cases the illness is more obscure, and the diagnosis may rest upon irregular fever, precordial pain, and a persistently excited action of the heart. Should a bruit develop under observation, this will prove the most reliable direct evidence in favour of acquired endocarditis.

It is not surprising that acute rheumatism is liable to occur in a certain number of children afflicted with congenital heart disease, for it is recognized that a certain number of such cases are the result of intrauterine rheumatic endocarditis, and the tendency to fresh attacks of rheumatism exemplifies the ordinary behaviour of the disease.

Sudden death, pulmonary tuberculosis, acute endocarditis or intercurrent disorders may thus terminate life. Yet another danger to life is gradual failure of compensation comparable to the failure so often seen in acquired heart disease; this is less common than perhaps might have been expected.

A fatal cerebral thrombosis occasionally ends life abruptly, as the next case exemplifies.

An infant of 9 months was brought here with obvious signs and symptoms of congenital heart disease. There was nothing unusual about this case, although it was severe and the condition critical. Three days later, while washing the child, the mother noticed that the right side of the face, the arm, and the leg were paralysed, and when I saw it there was a complete right hemiplegia. The quiet onset, feeble action of the heart, and general weakness of the infant, led me to the diagnosis of a thrombosis. The child lingered for a day or two, and at the necropsy a thrombosis with softening in the cortical area on the left side of the brain was discovered.

I think it may be said that the diagnosis of congenital heart disease is, as a rule, an easy one, and that when difficulties arise they depend either upon the absence of cyanosis or of a reliable history. Occasionally fibrosis of the upper lobe of the left lung causes retraction of the lung from the pulmonary conus and the development of a loud bruit systolic in time, which may be mistaken for a congenital malformation. When both congenital and acquired disease are present the twofold nature of the lesion may be overlooked. Some cretins are remarkably

cyanosed, the extremities showing the coloration in particular, and on more than one occasion I have heard considerable doubt expressed as to whether cretinism could really explain this phenomenon. In such cases treatment with thyroid extract has settled the question, and the cyanosis has rapidly disappeared with the improvement in the cretinous symptoms.

Many pulmonary conditions may produce cyanosis, but the diagnosis is generally obvious.

The differential diagnosis of the various forms of malformation is unsatisfactory. Some of the details have been already given, and are sufficient to act as a general guide.

PROGNOSIS.

The general prognosis, since it includes every sort of malformation compatible with live birth, is grave. The first principle is to reckon symptoms as more important than physical signs. A small opening in the foramen ovale or in the septum between the ventricles may give rise to no symptoms at all. On the other hand, such symptoms as paroxysms of dyspnoea or convulsions, or a persistent low temperature, are very ill omened. Another important point in the general prognosis is the social status of the patient. A child who can be given all the advantages of a warm climate and can escape in after-years the not unmixed blessing of having to earn a living, who can be well clothed and be educated by tutors, stands a far better chance than the child who sells matches in the streets, with icy-cold extremities and no proper meals.

The occurrence of an attack of acute rheumatism is an exceedingly serious matter, and liable to end in a malignant endocarditis. Repeated bronchitis, pneumonia, tuberculosis, and all acute maladies, including influenza, may entirely alter the prognosis in a case apparently favourable.

When the condition of the heart itself is taken into account, the prognosis is better in lesions of the ordinary type than when the lesion is an unusual one. It is not indeed possible, even with the ordinary systolic bruit and thrills, to be absolutely certain of the nature of the malformation, but in general it means a pulmonary stenosis, and this, if moderate in degree, is compatible with a life reaching to adult years.

Laurence Humphry points out that the prognosis is better when with pulmonary stenosis there is an opening in the ventricular septum, for this opening eases the pressure in the right ventricle.

A systolic murmur heard at its maximum intensity about the middle of the precordial area, and not giving rise to a thrill or to hypertrophy of the right ventricle, suggests this particular lesion of a patent septum.

The extent of the cardiac dullness to the right of the sternum is also some guide in prognosis, for when the increase is considerable, either the lesion is considerable or the strain on the right side for the extent of lesion is great.

The degree of cyanosis cannot be relied upon in the question of prognosis. The most cyanosed cases that I have met with have been children over 8 years of age. On the other hand, it happens by no means uncommonly that infants with congenital heart disease, and little or no cyanosis, die quite suddenly. The only warning that may be given in such cases is a refusal to take food and a general surface coldness. Again, it does not follow that because there is only a slight degree of cyanosis, which has only been observed when the child began to walk, there may not also be a rapid development of serious symptoms and death.

Cyanosis, in its most marked degrees, is associated with such lesions as pulmonary stenosis and patent septa; and these are recognized as the less severe types of malformation.

The treatment is palliative. These children must be kept warm, and very carefully clothed with this in view. A warm equable climate is much to be desired. If they can digest fat it is advisable to give them an excess, and keep them plump. No overcoat fits so well and acts so effectually as an undercoat of adipose tissue. Whenever possible they should be educated, and a light employment chosen for them. The amount of exercise must be adapted to each particular case, and will always need caution. Of the ordinary cardiac stimulants strychnine is more useful than digitalis. Cod-liver oil, iron, and malt are often well borne.

For the fainting attacks it is well for the parents to keep at hand a rapidly-acting diffusible stimulant, such as is prescribed for patients who live in dread of an attack of angina pectoris. Sal volatile, ether, and spirits of chloroform makes a useful combination. A warm mustard bath is valuable for an infant in this state of collapse, and oxygen is also of assistance. When the heart failure is more gradual and a result of overdistension of the right side of the heart the application of leeches over the liver brings relief.

AORTIC INSUFFICIENCY.

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DR. MITCHELL BRUCE'S clinical lecture, published in the *BRITISH MEDICAL JOURNAL* of January 6th last, will amply repay careful study. There is one point, however, about which, as he admits, "there is a division of opinion," since it is "maintained by some authorities on diseases of the heart that digitalis should not be given in aortic incompetence," but he justifies the use of digitalis, because his patient, who had "failure of the heart in association with aortic incompetence, was beneficially, as well as safely, treated with . . . digitalis."

I am in thorough accord with so distinguished an authority when he makes his deduction from clinical experience. The theories which are evolved from laboratory experiment are not always justified or proved by bedside observations. The records of a physiological observation and the evidence of a sphygmograph or cardiograph may not repeat themselves, as we expect they should, when we arrive at the bedside.

There would appear to be a tendency in laboratory experiment to record results with a somewhat biased mind. A sphygmographic curve intended to show the effect of this or that drug may mislead, and I think often has done so. I have worked with this instrument for a number of years, but I cannot satisfy myself as to the clinical accuracy of its statements. Hence the more patients I see the more I am convinced that clinical knowledge and experience constitute the most valuable form of medical observation. I would, therefore, meet Dr. Mitchell Bruce on the same clinical grounds, by recording my experience of the use of digitalis in aortic regurgitation.

It is not a little singular that some of the most eminent authorities on cardiac affections, who advocate the use of digitalis in this grave valvular affection, do so with qualifications and reserve. They, with some notable exceptions, appear to be somewhat lukewarm in its praises, or at least to resort to some saving clause, as though they feared that their writings and opinions should be falsified by the test of accumulated experience. Dr. Oliver, when advocating the use of digitalis in this disease, admits that the cases must be carefully selected. Dr. Barrs says that digitalis may aggravate symptoms, and in some cases, although much temporary relief has been afforded, sudden death had followed its use. But his opinion is decidedly in favour of the drug when there are signs of ventricular failure. Sir William Broadbent, as becomes a physician with his profound clinical knowledge, is cautious when speaking of the use of this drug. He points out the advantage of digitalis when dropsy supervenes, but shows that if its exhibition be persisted in after this, it is not uncommon for patients to die suddenly, sometimes in bed, more frequently when they have begun to get up. He says, "There are grounds for suspicion that digitalis contributes to this. In the absence of mitral symptoms, it is rarely that digitalis is called for in aortic incompetence, or is of service, and it may undoubtedly do harm."

Probably the controversy may never be settled in our time. Digitalis will have its advocates and its opponents. Many of the former will say that the latter are influenced by the teaching of pharmacology; and the latter will retort that the former are imbued with the experiments in physiology and physics. It may be as well, therefore, if short records of a few cases which have come under my notice should be published:

1. Kate F., aged 35, sempstress, was under my care as an out-patient for fourteen years. She suffered from aortic regur-

gitation of rheumatic origin. It is true that the first sound over the aortic area was attended by a slight murmur indicating that there was some obstruction co-existing with incompetence. During the whole of the long period she was under my care she never had digitalis in any form. The treatment consisted of periodic rests in bed, a careful dieting in which animal food was as predominant as her means would allow, and for medicine the tincture of nux vomica at intervals. This line of treatment was resorted to when attacks of dyspnoea and other signs of cardiac failure appeared from time to time; but she was able to earn her living by fairly hard work, though accompanied by anxiety. Then she was transferred to another physician during one of her bad periods, and was given digitalis. After the ninth dose (1/4 x of the tincture) she suddenly died.

2. Clara G., aged 31, had acute rheumatism when she was 17 years old. A lady of independent means, she could hence rest when necessary, and follow medical advice without anxiety as to her livelihood. She consulted me seventeen years before her death, complaining of dyspnoea on slight exertion. I found that she had aortic regurgitation, pure and simple. I saw her from time to time and from year to year, at intervals of about three months, and never during that long period did she take digitalis in any form whilst she was under my care. Shortly before her death she went to reside in Surrey, where she again suffered from an attack of breathlessness, and requested her local medical adviser to write to me, which he did, giving me an excellent account of her symptoms and his treatment. His remedies contained digitalis in one form or another, and I wrote to him to express my opinion that this drug was harmful in her case. She died suddenly on the fifth day after digitalis was first prescribed.

3. A young practitioner, having charge of a ward in a poor-house infirmary, came to me in distress, bringing the following story: Three old men were under his care suffering from aortic disease, regurgitation being the principal or dangerous condition. As they were all showing signs of heart failure and had oedema of the lower extremities, my friend ordered each to take a mixture containing digitalis three times a day. One man died on the third day thereafter, another on the fifth, and the other on the seventh day—all somewhat suddenly.

4. A gentleman, aged about 60 years, was desirous of effecting a "short term" (a few months) assurance of his life. I found on examination that he had a blowing diastolic murmur over the aortic area and a reflux pulse. Seeing that I hesitated over his proposal, he volunteered this statement: "I suppose you are frightened at my heart. I know it is wrong, but it is no worse now than it has been for some years, and I have just been to my doctor, who has given me this prescription." It contained digitalis, whereupon his proposal was rejected. Within a few weeks—not more than three—a notice of his death claim was received.

5. Miss N., aged 17, consulted me in 1888 on account of shortness of breath. She had had two attacks of rheumatic fever since she was 14 years old. I found that she had all the signs and symptoms of aortic regurgitation, but the mitral valve was "still holding." I prescribed a diet and regimen similar to that mentioned in the first case, and gave her my opinion as to the inadvisability of her taking digitalis. She has obeyed this injunction to the letter. She married, against my advice, in 1892, being then aged 21 years, and she has now three healthy children. She called to see me in February last in apparent good health but with obvious signs of aortic regurgitation.

6. At a clinical lecture some few years ago I exhibited six patients, all adults. Three were suffering from aortic regurgitant disease accompanied also by mitral insufficiency, whilst the other three had only aortic disease (regurgitation). Owing to some mistake on the part of the hall porter the patients, all six, were allowed to climb a deep staircase to the theatre, although orders had been given that they should be taken up by lift. The three patients who had the most extensive valvular lesions—aortic regurgitation and mitral regurgitation—performed the journey with ease; the other three, who had aortic regurgitation only, were so distressed on their arrival at the demonstration that they could not be examined for quite an hour; and, indeed, I deemed it wise to retain two of them in the wards over night.

These last cases may be somewhat away from the subject I am discussing, but I quote them as illustrating the advantages which will accrue to a patient with aortic insufficiency when he shall also have mitral insufficiency coexisting. I could relate other cases in which sudden death has occurred within short periods of the administration of digitalis in aortic insufficiency pure and simple, but these will suffice.

And herein lies one of the points of my contention. Not only would it appear that a patient suffering from aortic regurgitation has less distress if he also has mitral regurgitation, but he is also capable of greater exertion. There is a safety-valve action through the mitral orifice comparable to the tricuspid safety valve on the right side during prolonged muscular strain. It is true that the one is a pathological condition and the other a physiological