later show evidence of the disease. Ophthalmoplegia or ptosis may be present, the pupils show something abnormal, either dilated or contracted, possibly fixed and insensitive to light. But above all the patient is unusually lethargic and drowsy, but answers questions rationally when roused.

It was fortunate that I saw these cases at Birmingham, for afterwards I was called to cases at various military hospitals and in the neighbourhood of Exeter. In every case I have seen the early diagnosis has been very difficult, although later the symptoms were quite diagnostic.

When I was in France in July, 1918, the disease was attracting a good deal of interest, as it was said that a special organism had been found which had reproduced the disease in monkeys. I have not, however, noticed that these observations have been confirmed. Often when I have seen one case, I have seen one shortly after, either in the same town, or in a neighbouring district; it has not, however, been possible to trace any connexion between the cases. I saw two cases at Portsmouth within a few weeks of one another, and about six or eight weeks ago I saw a case at Chagford, one at South Molton, and one at Aylesbeare in one week, but there seemed to be no connexion whatever between the cases, and I know nothing as to the cause of the disease.

Convalescent Dépôts in France.

In France I was much interested in the convalescent dépôts for tired and convalescent soldiers. I visited four or five, and spent the whole afternoon at No. 10, at Ecourt, near Boulogne, studying the organization. They were of two grades. In Grade I a certain amount of treatment and the special exercises for heart cases, etc., were carried out. In Grade II no treatment was given. No. 10 Convalescent Dépôt at Ecourt was Grade II. Here there were between 1,900 and 2,000 men, all employed. Gardening was a great feature, and here I saw some of the best flowers and finest vegetables I have ever seen. At this dépôt all the men, except those with special employment, took part in parades twice a day, about 500 in each. The men remained there from seven to fourteen days, and a few for a month, before returning to their duties. I watched the 2 p.m. parades.

(a) Five hundred paraded for games; watching them afterwards, I saw some excellent boxing, hockey, and football. All of the men were engaged in these games; it was not a question of a few players and the rest looking on.

(b) Five hundred men had a route march of one and a half hours.

(c) Five hundred men paraded for bathing.

Each parade had its own band, which played at the head of a parade, and, although the personnel was always changing, the bands were very good.

Prizes were given for company matches and competitions, and the prize team walked off parade first.

The whole organization was arranged to keep up the mentality of the men, and I never saw men more keen or in such good condition; the contrast with the men staying about in our small convalescent homes at home was remarkable.

These convalescent dépôts impressed me enormously. As a profession we hardly realize how much the mentality of a patient helps or retards his cure. Hurst and others have pointed out how much the suggestion unintentionally conveyed by the questions of the medical attendant may have to do with producing the symptoms in hysteria. He has also shown how many of the symptoms observed in organic disease, especially of the nervous system, are functional, being grafted on to the true organic symptoms. Every one recognizes what an important part suggestion plays in the cure of the functional conditions, but until I studied these most admirable convalescent dépôts, where fagged out soldiers and convalescents were, so to speak, resuscitated in two or three weeks and made fit to return to duty, I had never realized how necessary it is to keep up the mentality of a sick man, and to keep him employed, interested, and amused if he is to be restored to health as quickly as possible.

Army Sanitation.

One of my most pleasant experiences was when I joined the medical head quarters of the Fourth Army in France, and found there our old friend, Major Leonard Tosswill of Exeter, occupying the important position of Chief Sanitary Officer of that army. We English are inclined to grumble, and to decry and underestimate our achievements, but we

may well be proud of the achievements of our Royal Army Medical Corps in the face of all their difficulties in the field; difficulties of which their stay-at-home critics have not the faintest conception; and most especially may we be proud of the sanitation of our armies under the direction and supervision of men like Major Tosswill. With him I visited the St. Requier School of Sanitation for the Fourth Army, where forty men at a time were put through a course of sanitation, and taught to make all kinds of sanitary appliances out of old biscuit and petrol tins. From these they will produce pipes to be used as chimneys, stoves without solder, field kitchen ranges and de-lousing pits. They learn to make bricks by pressure, and how to construct latrines, etc.; how to purify water; and the part which flies and lice exercise in the production of disease, and how to destroy them.

With Major Tosswill I visited Amiens, then a deserted desolate bombarded city, for the sanitation of which he was responsible. With him I saw the way in which the horse manure was prevented from breeding flies, and visited several of the baths established for the men at the front and for providing them with lice-free fresh clothes. In fact, he showed me the numerous details which went to make up the more or less perfect sanitation of our army, which, considering the difficulties of a fighting army constantly on the move, were really admirable. The question arises, What is to become of the well trained sanitarians of St. Requier, and of the schools of sanitation belonging to the other armies? What is to be done with the splendidly trained sanitary officers who have all this war experience? In the future are the small towns in England, at the cost of a few pounds, going to have public disinfecting chambers on the principle of the de-lousing pits, and will the huts recently used throughout France as hospital wards be erected at little cost as isolation hospitals? Or are we going to forget all that the war has taught us, as in so many directions we seem already to have forgotten? Time will show. But it is to the younger men of the nation who have gone through this war that we must look if all this experience is not to be lost.

In conclusion, I should like to say how pleased I have been to be associated with the regular R.A.M.C., some of whom have done scientific work of great value, while many of the higher officers in the service are wonderful organizers who have done grand work throughout the war. With them have been associated some of the best men in England, who, like myself, have been temporary officers, and I am pleased to think that, while our medical knowledge has been greatly advanced, our sick and wounded have been cared for as they never have been in any previous war.

Observations

ON

MITRAL STENOSIS IN SOLDIERS.

BY

THOMAS F. COTTON, M.D.

(A Report to the Medical Research Committee.)

WHEN the authorities at the War Office decided to establish a special hospital at Hampstead, and later at Colchester, for the treatment of soldiers invalided with affections of the cardio-vascular system, an unrivalled opportunity was given for the study of heart disease in these men. During the past three years there have been under observation more than 800 patients with clear signs of structural heart disease. The majority of these were young men, with little or no disability before enlistment; they were passed as fit for military service, the greater number had seen active service abroad, and in only a very few were there signs of heart failure while in hospital.

In the wards of the public hospitals there are always patients with advanced heart disease; clinical observations made by able observers, and accurate pathological studies by careful laboratory workers have led to a sound understanding of diagnosis and treatment in these cases. At no time in the past has it been possible to observe in the wards of a hospital such a large number of patients with early organic heart affections. The opportunity seemed a favourable one to study the symptoms and

signs in these men, with the view of explaining the significance of the symptoms and the relation they bear to the signs of organic disease of the heart during the early stages of its development. With this purpose in mind I have chosen mitral stenosis in soldiers as a subject for investigation. My reason for doing so is, that this type of valvular disease, whether early or developed, always means a structural lesion of the heart, and it is a valvular defect which develops slowly, and so allows one to correlate in a definite manner symptoms and structural disease.

During the past winter I had under observation at the Sobraon Military Hospital, Colchester, 75 patients with signs of mitral stenosis. I have analysed the histories in 50 of these and determined the exercise tolerance in all. It is on the results obtained from the clinical observations in these men, their symptoms and signs, and physical endurance, that I am led to draw certain conclusions, which in my opinion offer a satisfactory explanation for the physical condition of the soldier with mitral stenosis as we have seen him in the army or at the pension board.

Their average age was 29 years, and the average period of military service three years and one month, including one year and nine months' foreign service. The average duration of symptoms in 40 was one year and three months, and of these a few had been in perfect health one month before admission to hospital. Of 8 others with symptoms before enlistment, one had been unfit for three years, one for eight years, and six since childhood. The civil occupation had been heavy in 22, moderately heavy in 15, and light or sedentary in 12; 34 had played games, such as football, tennis, and cricket, up to the date of enlistment; 11, from lack of interest or physical incapacity, played no games. In the army, the military service had been heavy in 25, and light in 19. The onset of symptoms occurred after an acute infection in 26, or 52 per cent.; after a severe physical effort in 8, or 16 per cent.; and after gassing in 4. In 10 the symptoms developed gradually, and no cause could be found to explain them. A history of one or more attacks of rheumatic fever was present in 22, and of these 13 dated their symptoms from this infection. There was no enlargement of the heart in 30; in 15 the cardiac impulse was palpable just outside the nipple line, and in 5 there was moderate enlargement.

The exercise tolerance was determined in 17 by submitting them to graded exercises and observing their response to these exercises. The exercises were those used at the hospital for sorting and treating D.A.H. cases. In all (75) the capacity to bear a physical strain was determined by giving them a simple exercise test, consisting of a brisk walk up and down twenty steps twice. Symptoms of distress were noted, when present, and the pulse rate was taken before the effort, immediately after, and at the end of two minutes. The exercise tolerance in 50 was gauged by the degree of distress, the rise of the pulse rate, and the length of time required for the heart to return to the pre-exercise rate. In 25 the exercise tolerance was determined by the symptoms alone after the exercise test; the pulse rate was recorded with the Mackenzie polygraph for five minutes before the exercise, with the patient sitting, immediately after, and at the end of two minutes.

Of the group of 50, 25 presented early signs of mitral stenosis, and 25 signs of developed stenosis of this valve. Of the early cases, 15 had good exercise tolerance and 10 poor exercise tolerance; of the later cases, 14 had good exercise tolerance and 11 poor exercise tolerance. The average pulse rate in those with early signs with good exercise tolerance was 90 before exercise, 142 immediately after, and 84 at the end of two minutes. In those with poor exercise tolerance the average pulse rates were 99, 162, 114. Of those with developed signs of mitral stenosis with good exercise tolerance, the average pulse rates were 93, 153, 82; of those with poor exercise tolerance, the rates were 86, 166, 100.

It will be seen from these figures that in the early cases with poor exercise tolerance the pre-exercise rate is slightly higher than in those with good exercise tolerance, an increase of 9; that the rate after exercise is distinctly higher, an increase of 20; and that at the end of two minutes the heart has not returned to the pre-exercise rate—114 as compared with 99 before exercise, a difference of 15, or, when compared with the pulse rate at the end of two minutes, in those with good exercise tolerance, a difference of 30.

In the other group, with developed signs of stenosis, if a similar comparison is made, it will be seen that in those with poor exercise tolerance the pre-exercise rate is slightly lower, a difference of 7, the rate immediately after exercise is higher by 13, and at the end of two minutes the rate is 100 as compared with 86 before exercise, or 82 at the end of two minutes in those with good exercise tolerance.

If these figures are represented diagrammatically—(Diagram I)—it will be seen that in those with poor exercise tolerance the pulse rate reaches a higher

level immediately after exercise than in those with good exercise tolerance, that this level is approximately the same whether the signs of mitral stenosis are early or developed, and that the pulse rate does not return to the pre-exercise rate at the end of two minutes. In those with good exercise tolerance the pulse rate is higher when the signs are developed, and after two minutes has returned to the pre-exercise rate. It may, then, be concluded from these figures that the increase in pulse rate is a useful sign in estimating exercise tolerance in patients with mitral stenosis, but it is not a guide in distinguishing between early and developed signs of mitral stenosis.

In the average the exercise tolerance in patients with early mitral stenosis is better than in the D.A.H. cases. The exercise tolerance was determined in 75 with mitral stenosis, and of these, 48, or 64 per cent., had good exercise tolerance, and 27, or 36 per cent., had poor exercise tolerance. In 50 with D.A.H., 24, or 48 per cent., had good exercise tolerance and 26, or 52 per cent., had poor exercise tolerance. The difference in the exercise tolerance in the cases tested is probably due to the fact that those with mitral stenosis were sometimes invalided because they had signs of structural heart disease, while those with D.A.H. were more often admitted into hospital because of symptoms after effort.

Forty-two men were admitted into hospital with unmistakable signs of mitral stenosis. They had no symptoms before enlistment; a large majority played games and worked hard; they experienced no difficulty with the military training, and during two years of active service 25 carried on as fit men. Their physical endurance equalled that of healthy men. Some, after three years' military service, with two years in France, had no symptoms three months before admission to hospital. The pathological changes in and about the mitral valve, leading to a narrowing of the auriculo-ventricular orifice, develop slowly. The signs by which mitral stenosis is diagnosed are signs of organic valvular disease. It is inconceivable that symptoms of such recent origin coincided with the development of disease in this valve. It is certain that mitral stenosis existed in many if not all these men throughout the whole period of their military service. From the histories obtained in these patients, and from the signs which they presented while under observation in hospital, I am led to conclude that mitral stenosis is compatible with a state in which there are no symptoms, and that this type of valvular disease is consistent with good exercise tolerance.

A patient with mitral stenosis and definite signs of cardiac failure will always show a poor exercise tolerance, and symptoms are always complained of. Breathlessness after effort is a conspicuous feature during this stage of the disease. Palpitation, giddiness, precordial pain, exhaustion and lassitude are other symptoms. But cardiac failure is not the only cause of such symptoms in mitral stenosis. There is in patients with heart failure a breathlessness due to deficient aeration of the lungs, associated with signs of venous engorgement and pulmonary congestion, enlargement of the liver and dropsy. There is another type of respiratory distress which is caused by an acidosis or diminished alkalinity of the blood; it is paroxysmal in character, not necessarily accompanied by cyanosis, and is seen in patients with cardio-renal disease. The patient with early pulmonary tuberculosis is unduly breathless on exertion. Breathlessness may be observed in healthy men after a severe effort, or in unfit men in whom there are no signs of structural disease after moderate exercise. Clearly heart failure is not the only cause of breathlessness. Pain that is referred to the heart, and anginal in its distribution, is a symptom of heart failure, and may be associated with signs of heart failure. But precordial pain is not always substernal; all of the distinctive qualities of angina pectoris may be absent. Patients without signs of structural heart disease may complain after exertion or at rest of a sharp cutting or even boring pain felt in the region of the heart. Pain of this sort is a frequent complaint in the D.A.H. patient in whom there are no signs of cardiac failure. Palpitation, giddiness, and exhaustion are symptoms often observed in the D.A.H. case. The point which I desire to make is that symptoms associated with heart failure may be observed in patients presenting no signs of structural disease of the heart, such as cardiac enlargement, valvular disease, or venous engorgement.

DIAGRAMS SHOWING PULSE RATES BEFORE EXERCISE; IMMEDIATELY AFTER; AND AT THE END OF TWO MINUTES.

I. Continuous line = early signs of mitral stenosis; broken line = developed signs of mitral stenosis. II. Tolerance determined by symptoms and pulse rate. III. Tolerance determined by symptoms alone. IV. Diagrams II and III combined. In each diagram the two low summits are those with good exercise tolerance; the two high summits those with poor tolerance. (In II, III, and IV, continuous line = mitral stenosis; broken line = D.A.H.)

Now a patient with mitral stenosis and no signs of cardiac failure may have a poor exercise tolerance, with such symptoms as breathlessness, palpitation, giddiness, and precordial pain after effort. If a comparison is made of the histories of soldiers with mitral stenosis, and no signs of venous engorgement, and no material enlargement of the heart, with an equal number of unselected D.A.H. cases, it will be found that the symptoms in the two groups are similar in every respect, and indistinguishable the one from the other. Breathlessness is present in all, and is provoked by an effort which would not be complained of in a healthy subject. Sometimes it is felt at night—sudden in its onset and of short duration. Palpitation is complained of by many. It is felt after an effort, and is then associated with an over-acting heart. Emotional stimuli produce it, and sometimes it is a distressing symptom in bed at night. Pain in the region of the heart is a common complaint; it occurs after an effort, and it may be felt at rest. It is not always limited to the precordium. Sometimes it is confined to a small area about the left costal border, or near the angle of the scapula. It is usually described as a sharp, cutting, stabbing pain, not to be distinguished from a stitch. Rarely there is skin hyperaesthesia over the heart and tenderness of the left pectoral muscle. Giddiness after effort or on change of posture is a frequent complaint. Exhaustion after an effort, a feeling of lassitude in the morning, tired out before the day's work has begun, mental depression or irritability of temper, flushing of the face, cold or clammy hands, sometimes tremulous and sweating axillae—these symptoms complete the picture of the effort syndrome in patients with D.A.H. or early mitral stenosis.

They date their symptoms from the same causes. A history of symptoms occurring after an acute infection, after gas poisoning, severe physical strain or shell shock, is as frequent in the patient with early mitral stenosis as in the D.A.H. case. With the exception of rheumatic fever, which is higher in mitral stenosis, the incidence of infections is the same in the two groups. In both the symptoms may be sufficiently severe to incapacitate them for work. They may both have an exercise tolerance so poor that the slightest effort causes distress. In some moderate exercise is tolerated, in others the physical capacity for work is reduced only slightly less than that of a healthy individual. Further, these symptoms may entirely disappear and a normal exercise tolerance may return, with the signs of mitral stenosis remaining unaltered by the improved condition of the patient.

In the presence of signs of mitral stenosis it is natural to ascribe the symptoms observed after effort to cardiac weakness. The breathlessness, the precordial pain, the over-acting heart, are all symptoms which have long been associated with organic heart disease. But it is known that such symptoms may be absent in patients with mitral stenosis, and we have abundant evidence that these symptoms may be present in the absence of any evidence of structural heart disease. Can we not say that the symptoms in patients with early mitral stenosis and D.A.H. are due to the same cause, and that in the state of our present knowledge there are no symptoms which we can definitely attribute to this type of valvular disease during the early stages of its development? One test of this is a comparison of the pulse rates after exercise.

The pulse rates of 41 patients with early mitral stenosis were taken before and after a simple test exercise, and symptoms of distress, if any, were observed and noted. The physical exertion required of these men was a brisk walk up and down twenty steps twice. The pulse rates were counted, with the patient standing, before the exercise, immediately after, and at the end of two minutes. The exercise tolerance was determined more by the appearance of the patients than by the pulse rate. Where there was any doubt in estimating the degree of fitness, a high pulse rate after the exercise and a slow return to normal was considered a sign of poor exercise tolerance. A moderate increase in rate, with a quick return to the pre-exercise rate, was looked upon as a sign of good exercise tolerance. A group of 25 unselected D.A.H. cases were submitted to the same exercise, and their tolerance was determined in the same way.

Of those with mitral stenosis, 24 had good exercise tolerance, and the pulse rates averaged 91 before exercise, 147 immediately after, and 83 at the end of two minutes; 17 had poor exercise tolerance, and the pulse rates were 90 before, 165 immediately after, and 105 at the end of two minutes; 13 of the D.A.H. cases had good exercise tolerance, and the pulse rates were 96, 146, 91; 12 had poor exercise tolerance, and the figures were 112, 159, 106.

When the exercise tolerance is good, in those with mitral stenosis, the increase in pulse rate is 56, as compared with 50 in those with D.A.H. When the exercise tolerance is poor in the former, the increase is 75, as compared with 47 in the latter. This wide difference in the increase in the pulse rate, when the exercise tolerance is poor, may be explained by the high pre-exercise rate in the D.A.H. patients—112 as compared with 90, a difference of 22.

In 25 patients with mitral stenosis, and an equal number with D.A.H., the exercise tolerance was determined by the symptoms alone after the test exercise. The pulse rates were

recorded with a Mackenzie polygraph, and the pre-exercise rate was the average for five minutes, with the patient sitting. The pulse rates in 19 with mitral stenosis, and good exercise tolerance, were 78, 123, 81, an increase of 45; in 6 with poor exercise tolerance they were 101, 144, 98, an increase of 43. In those with D.A.H., 11 had good exercise tolerance, and the rates were 93, 126, 97; 14 had poor exercise tolerance, and the rates were 106, 161, 115, an increase of 55.

The pulse rates in those with good exercise tolerance are approximately the same after exercise; the higher pre-exercise rate in those with D.A.H. will explain the greater increase in those with mitral stenosis.

When the exercise tolerance is poor the pulse rate is higher in the D.A.H. cases; but only 6 with mitral stenosis were tested, as compared to 14 with D.A.H., so that I do not attach much importance to these figures.

Now if the pulse rates are compared in all, those in whom the tolerance was tested by the symptoms and pulse rates, and by the symptoms alone, we will be dealing with a larger group of men, and a fairer comparison can be made.

The pulse rates of 43, with mitral stenosis and good exercise tolerance, were 85, 136, 82, an increase of 51; in 24, with D.A.H. and good tolerance, 94, 137, 95, an increase of 43. The rates after exercise are the same, and the difference in the increase is equal to the difference in the pre-exercise rates. In 22, with mitral stenosis and a poor exercise tolerance, the rates were 95, 140, 104, an increase of 65; in 26, with D.A.H., 108, 160, 111, an increase of 52. The rate after exercise is the same, and the difference in the increase equals the difference in the pre-exercise rate.

These figures will be more easily read if presented in a diagrammatic form. In the diagrams the dotted lines represent the pulse rates in the D.A.H. cases before exercise, immediately after, and at the end of two minutes, and the unbroken lines those with mitral stenosis. The figures at the side are pulse rates per minute.

In Diagram II are represented the pulse rates when the exercise tolerance was determined by the symptoms and the pulse rate. In those with good exercise tolerance the summits of the curves reach approximately the same point; in those with poor exercise tolerance they are at a higher level, with the mitral stenosis peak a little above the D.A.H. peak. In Diagram III the tolerance was determined by the symptoms alone. In those with good exercise tolerance the summits of the curves reach about the same height, and at a lower level than in those with poor tolerance. The mitral stenosis lines are both lower than the D.A.H. lines, and considerably lower than the mitral stenosis lines in Diagram II. In Diagram IV, which is II and III combined, the high points are the same when the tolerance is good; when it is poor they are the same, and at a higher level than in those with good exercise tolerance.

From these figures it can be stated—and it is the only point which I desire to make—that when there is an equal degree of distress after effort, the pulse rates rise to the same level in soldiers with early mitral stenosis and D.A.H.

The study of the symptoms in patients with mitral stenosis leads one to consider the signs of mitral stenosis and the methods employed in recognizing this valvular defect. I have described the symptoms occurring in patients with early mitral stenosis in whom there were no signs of cardiac failure. It is not sufficient to say that these men had a presystolic murmur at the apex and no venous engorgement. These observations will be incomplete if I do not define what I mean by early mitral stenosis and what I consider signs of heart failure.

I regard early mitral stenosis as including patients possessing the earliest signs by which the disease may be recognized. In those with unmistakable signs but no increase in the size of the heart, or only a moderate degree of enlargement, the valve defect may be of long standing, but the condition is not one of advanced heart disease; the former I refer to as patients with early signs, the latter as having developed signs of the disease. Many of these patients were very early cases of mitral stenosis; in others there were unmistakable signs of the disease. In all but a few there was little or no enlargement. The signs of mitral stenosis may be taken as an indication of myocardial disease. It is rare to have valvular disease of this type without pathological changes in the heart muscle. The agent that has damaged the valve has at the same time injured the myocardium. Disease of the myocardium means myocardial weakness, and leads to enlargement of the heart.

If there is in mitral stenosis much enlargement of the heart—that is, two fingerbreadths or more beyond the

nipple line—the disease may be said to have reached an advanced stage of development. There may be no signs of cardiac failure, but the breakdown is likely to occur sooner than if the heart were not enlarged, or only slightly increased in size. Symptoms are of little help in fixing the onset of the disease. A history of a recent attack of rheumatic fever may be a guide in arriving at the date of origin of the disease. Rheumatic fever in childhood may indicate that the disease is one of long standing. In patients who give no history of rheumatic fever—and these form 60 per cent. of my series—or in those in whom there is no history of any infection we must rely upon the signs alone in estimating the number of years during which the disease has been present. Patients with mitral stenosis and no enlargement are not cases of advanced heart disease. In those with unmistakable signs of mitral stenosis and little or no enlargement of the heart the disease may have been of long standing, and yet the pathological changes have occurred so slowly that the disease has not reached an advanced stage in its development. These patients I refer to as early cases with developed signs of mitral stenosis. Patients with signs that are recognized only by special methods of examination I consider early cases, with early signs of mitral stenosis. It is important to classify early mitral stenosis in this way, for we gain information concerning the course of the disease in the past, and we are better able to forecast the number of years remaining before heart failure sets in.

The diagnosis of early mitral stenosis can only be made in the presence of a diastolic rumble. In its absence the diagnosis is not sound. There are some who hold that an impure first sound at the apex, or perhaps a reduplication or an accentuation of the first sound, with a loud pulmonary second sound, are early signs of mitral stenosis. They may be an indication for a more careful examination, but they are untrustworthy signs. Such heart sounds are frequently heard in normal over-acting hearts, but as signs of valvular disease they are not to be relied upon.

Listen at the apex for a low-pitched murmur or a rumble in presystole, leading up to a sharp accentuated first sound. If uncertain of the presence of this murmur, and the examination is being made in the upright position, examine the patient lying down, or reclining on the left side. If still hesitating, give the patient an exercise test, sufficient to raise the heart rate to 140 or more. If there is no murmur to be heard in diastole it is highly probable that there is no narrowing of the mitral orifice. Adopt the same procedure in examining for a thrill. Do not be misguided by the pseudo-thrill of an over-acting heart, which is systolic in time, and lacks the purring quality of the presystolic thrill of mitral stenosis. A murmur and thrill that are brought about by change of posture or effort are early signs of mitral stenosis. There may or may not be enlargement of the heart. When the murmur is present at all times, standing or lying, or the thrill is felt in one or more positions, it is sound to diagnose the condition as one of developed mitral stenosis. The murmur may be heard in presystole as a rumble, or throughout the whole of diastole.

As the disease progresses the full pulsating veins of the neck stand out conspicuously with the patient lying flat, and they do not collapse when the head is raised above the level of the front wall of the chest. The ears and lips are cyanosed, the liver is palpable, and there is tenderness over it. With the increase of venous engorgement pulmonary congestion and other signs of cardiac failure are added.

The heart may or may not be enlarged in mitral stenosis. Cardiac enlargement is one of the most important signs which we possess of myocardial mischief. The ordinary methods of estimating the degree of enlargement are inexact, but for clinical purposes they are sufficiently reliable as an aid in diagnosing structural disease of the heart. It is best to take the left border of the heart, as determined by palpation and percussion, as an index of cardiac enlargement. When there is a defined impulse, unmistakably outside the nipple line, and on palpation it is felt as a thrust, it is sound to consider the heart enlarged. It is wrong always to regard such enlargement as due to hypertrophy or dilatation of the left ventricle, for a large right ventricle may give an impulse to the left of the nipple line. It is not safe to draw conclusions from percussion to the right of the sternum in gauging the size of the right heart. Increase in the area of cardiac dullness to the right, in hearts that are not displaced, and in the absence of pericardial effusion, is a sign of dilatation of the right auricle. The right ventricle increases in size in a

downward direction, and more to the left than to the right. When the right auricle is dilated there is usually engorgement of the veins of the neck and other signs of cardiac failure.

To map out accurately on the chest wall the right border of the heart is difficult, if not impossible, even in the hands of experts. It is better to rely on signs of venous engorgement, which all can recognize as indicating dilatation of the right auricle. It is certainly wrong to consider increase in the size of the heart to the right as a sign of enlargement of the right ventricle. Epigastric pulsation is too inconstant in mitral stenosis, and too frequently observed in patients with normal hearts to be of service as a sign of right ventricular hypertrophy. It is generally safe when there is a sign of enlargement of the heart, to the left to consider the whole heart as increased in size. It is true that in mitral stenosis the hypertrophy which predominates is usually a right ventricular one, but it is equally true that both ventricles are usually hypertrophied. Observations which Lewis and I made have shown this.¹ We weighed separately the ventricles of a large number of hearts with mitral stenosis, and found the hypertrophy to be general, but with a relative increase in the weight of the right ventricle. Such pathological findings, together with electro-cardiographic records, do not support the hypothesis that the valvular defect is the only factor in the production of hypertrophy in mitral stenosis. Some other cause must be found to explain the increase in weight of the muscle in both ventricles.

Prognosis is difficult in early mitral stenosis. By the symptoms alone as I have described them one may gauge the man's present capacity for work. The exercise tolerance, as determined by a simple exercise test, gives useful information concerning his present disability. As a measure of physical endurance the reaction to exercise is of prognostic value. As a test to be employed in predicting the duration of life it is not to be relied upon. Good exercise tolerance is never observed when there are signs of heart failure. Poor exercise tolerance is not necessarily associated with a cardiac breakdown. It is true that there are symptoms which so incapacitate that total disability is at once recognized, and the duration of life may be estimated with a fair degree of accuracy. I refer to such symptoms as breathlessness, associated with cyanosis and pulmonary congestion, and the pain of angina pectoris; such symptoms are associated with definite signs of heart failure. The disease is no longer in its early stages; our problem is an easy one, total disability is obvious, and prognosis is of less consequence. The prognostic task becomes a more difficult one when we are required to foretell, in the patient with early heart disease, the number of years remaining before the final stage of total disability has been reached. When will the patient with mitral stenosis and no symptoms have symptoms due to cardiac failure? With symptoms after effort when may we expect signs of venous engorgement? These questions cannot be answered unless we understand the symptoms and the relation they bear to the signs. Until we know what symptoms arise from loss of cardiac reserve through myocardial disease, and can recognize the early symptoms of heart failure, and identify symptoms due to other causes, it will not be possible to prognose with any degree of certainty in early mitral stenosis.

We know from experience that the course of the disease is a progressive one, the disability an increasing one, and heart failure the end of most patients with mitral stenosis. The duration of life may be shortened by repeated attacks of rheumatic fever, or other infections, or prolonged by the mode of living and proper care. A single attack of rheumatic fever many years back, with good health since, provided the heart is not enlarged, or only slightly increased in size, may be an indication that the disease is stationary. With the heart much enlarged the onset of heart failure in most cases is not far off.

REFERENCE.

¹ Cotton: "Observations on hypertrophy," *Heart*, vi, 217.

THE Board of Trade has received through the Norwegian Consul-General two silver cups awarded by the King of Norway to the master and the surgeon, Dr. A. Humphreys, of the steamship *Haverford*, of Liverpool, in recognition of their services to rescued members of the crew of the Norwegian steamship *Augsald* in the North Atlantic Ocean on July 4th, 1918.

APPARENT SPONTANEOUS RUPTURE OF A
NORMAL SPLEEN.

BY

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AFTER considering possible alternatives I have come to the conclusion that the following must be counted a case of spontaneous rupture of the normal spleen.

A private aged 43 years, whilst walking from his officer's billet to his own at 11.30 a.m. on December 20th, 1917, was seized with pain in the region of the umbilicus, so severe that it caused him to fall to the ground. He vomited, and the pain subsided sufficiently to allow him to walk to his billet. He endeavoured to carry on his duties, which were light, but, the pain getting worse, at 3.30 p.m. he reported sick, and was seen by his medical officer, who came to the conclusion he had colic and gave him a dose of castor oil, which he immediately vomited. As the pain was getting steadily worse he was sent to hospital.

When he arrived, at 6.30 p.m., I found him very collapsed; the temperature was 96.8° F., the pulse 130 and very feeble; the face was ashy grey. There were no external signs of injury. The abdomen was flat and breathing entirely thoracic. He lay with his legs drawn up. The abdomen was tender and there was board-like rigidity of the abdominal muscles. The liver dullness was normal. Both flanks were dull whilst lying on his back, shifting on turning over. Physical signs of bronchitis were present; the heart was normal. He vomited bile-stained fluid once after admission. The patient was so ill that he could not give a clear history of his previous life, but it was subsequently elicited that he had always been healthy except for chronic bronchitis during the winter. Eighteen months previously he had been buried in the trenches and was admitted to hospital suffering from severe abdominal pain, for which he was kept in bed for three weeks. Since his discharge from hospital he had had no recurrence of that pain and had been well in every way except for the bronchitis. He had never been in a malarial country.

Diagnosis.

The diagnosis of rupture of an abdominal viscus was based on the marked abdominal tenderness and board-like rigidity of abdominal muscles and free fluid in abdominal cavity, but there were no localizing symptoms. The patient refused immediate laparotomy, but three hours later he consented. Morphine gr. $\frac{1}{2}$, atropine gr. $\frac{1}{100}$ given.

Operation.

At 10 p.m. the abdomen was opened in the middle line above the umbilicus; a large quantity of fluid and clotted blood escaped on opening the peritoneum. The abdomen was hurriedly mopped out; the liver was found normal, but on passing the hand into the splenic region a large quantity of clot was displaced and free bleeding was found to be going on. A large rupture in the spleen was felt. When the splenic vessels were grasped the bleeding stopped. The wound was then extended transversely towards the costal margin, the left rectus being divided. The condition of the patient was too serious to allow an attempt at suture. The splenic vessels were isolated and ligatured with linen thread and the spleen removed. The wound was sutured with linen thread. The gall bladder was packed with stones, but was not touched. Subcutaneous saline was given continuously during the operation; warm ether was the anaesthetic administered. The pulse improved at the end of the operation, and he continued to make satisfactory progress during the night. In the morning the temperature was 98° and the pulse 108.

Description of Spleen.

The spleen was normal in size and weight; the tear, which extended from the convex surface to the hilum, divided the organ into two equal portions. There was no stripping of the capsule to suggest that at first there had been subcapsular haemorrhage later tearing through the capsule. It was clear that the tear in the spleen and capsule were present from the first, allowing of free intra-abdominal haemorrhage. Microscopical examination showed the spleen to be normal.

Progress.

The temperature ranged from about 100° to 102° F., but this was accounted for by the state of the chest. The pulse rate gradually dropped to 80. The stitches were taken out on the tenth day. The wound healed by primary union. No glandular enlargements were observed; he made a rapid recovery from anaemia, and when he was evacuated to England four weeks after the operation he was apparently in the best of health.

The question arises whether the injury he received eighteen months previously tore the spleen. If it did, there should have been some adhesions around the spleen or some sign of the old tear on the organ at the time of operation. I found no adhesions of any kind, and on examination of the spleen after the operation I found no evidence of an old tear; this was borne out later by pathological examination.