nately. In this way the occurrence of clonus would seem to entail the action of opponent muscles, that is to say, the required tension of flexor muscles will be induced by the contraction of extensor muscles, and *vice versa*. But we have to remember that clonus may be induced in muscles without their opponents contracting. Probably the best example of this is seen in the induction of knee clonus by pushing down the patella, as can be done readily in cases of lateral sclerosis or descending degeneration. Here the desired tension is obtained by pushing down the patella. In both of our patients knee clonus or clonus of the quadriceps can also readily be induced in this way. Hence then in tremors or clonus we have to recognise in the functioning of the motor centres disturbed conditions as regards strength and co-ordination, whereby opponent muscles are caused to contract alternately, or single muscles or sets of muscles intermittently. Further, we believe that the maximum rate of these alternate or intermittent contractions varies in different muscles according to differences in their distance from their nerve centres.

So much for the symptoms in our patients. Let us now consider the progress and treatment. Here I have to confess to you that for some weeks I failed to recognise the malarial element in their causation. Our treatment, therefore, consisted in efforts to improve the general health and to counteract what we thought to be a rheumatic element. In addition to rest, good diet, and hot-air baths, the patients were therefore getting mainly iodide of potassium, salicin, and arsenic. Practically no improvement followed, and then we gave quinine (5 gr.) thrice daily. The amendment was as distinct as it was immediate: both patients felt stronger, physically and mentally; the tremors diminished and the bladder troubles improved.

Recognising then that we were dealing with a form of malarial neuritis, I, after two weeks, prescribed in addition strychnine hypodermically—I had withheld it previously owing to the evidence of nerve and muscle irritability—and this appeared also to act beneficially. Within the past few days I have substituted anarctoin for quinine in R.'s case, with good effect, as he had been complaining of deafness. Both patients are still somewhat feeble, but they are now looking forward with confidence to being able shortly to resume their occupations.

A PROBABLE DIAGNOSTIC SIGN OF TRICUSPID STENOSIS.

By JAMES MACKENZIE, M.D.,
Burnley.

In the study of the symptoms of heart failure far too little attention has been devoted to the consideration of the regurgitant waves of blood sent back from the heart and made manifest by movements in the veins and in the liver. If we consider for one moment the opportunity given for observing what is taking place inside the heart from a comparison of the venous pulse with the arterial pulse, we shall realise the value of more careful study of the venous pulse. When we feel the radial pulse, or when we take a tracing of the radial pulse, we obtain a glimpse of what is going on inside the heart only during a very short period of the cardiac revolution, namely, during the time of ventricular contraction, from the time of the opening to the time of the closure of the aortic valves. (Space *x* in the tracings 1, 7, 14). On the other hand, when we observe the venous pulse we get a knowledge of what is taking place inside the heart during the whole cardiac revolution, and not only of what is happening in one chamber, but partly directly and partly inferentially of what is happening in both auricle and ventricle. Thus in Fig. 1 we see the effects of the auricular systole producing the wave *b*, immediately followed by the auricular diastole, during which time the blood in the veins pours into the auricle and causes the great fall in the veins (*x*). At this time the right ventricle is pouring blood during its systole back through an incompetent tricuspid orifice into the auricle. Hence before the ventricular systole is at an end the auricle is filled, and the overflow rushes back into the veins producing the wave *a*. The time of closure of the pulmonary valves is shown by the notch on *a*. Then the ventricular

systole being at an end the diastole of the ventricle begins, and permits the blood to flow from the auricle and the veins, giving rise to the fall in the vein shown on the tracing at *y*, which fall is interrupted by the recurrence of the auricular systolic wave *b*, and the whole series of events begins over again.

The pulse, when it appears in the veins, is very susceptible to change occurring in the heart, and from its study there may be gathered evidence of cardiac changes when little or no difference can be observed either from exploration of the chest or study of the arterial pulse. In cases, too, of pulse irregularity the venous pulse reveals what is occurring in the various chambers of the heart during irregular action in a manner instructive as well as surprising. In this paper I propose to demonstrate how the modifying influence of tricuspid stenosis on the right auricle is shown by the venous and liver pulses.

In cases of slight cardiac debility, such as occurs in fat people, chlorosis, pregnancy, pernicious anæmia, and other forms of functional heart failure, this debility of the heart is often made manifest by a pulsation in the jugular veins. If a tracing of this pulsation be taken, there is found to be a series of waves similar to that in Fig. 1, but not so large. If these waves be carefully timed, as in Fig. 2, where the jugular pulse is taken simultaneously with the carotid, it will be found that the wave *c* is due to the carotid pulse; preceding this is the wave *b*, which like the same wave in Fig. 1 can only be due to the systole of the auricle. In such cases, if the venous pulse persists until the patient dies, its form does not alter, except that the waves may become more or less conspicuous; and when the patient is dying, and the radial pulse is no longer to be felt, we may rarely remark in the veins at the root of the neck the wave *b* due to the auricle continuing to act, thus demonstrating the auricle's right to the title of *ultimum moriens*. In such cases, however great the heart failure may be, there is never produced pulsation of the liver.

On account of the fact that the wave due to the auricle persists in such cases, I have called this type of the venous pulse the auricular form of the venous pulse. In cases where there is organic disease of the mitral valves the backward pressure of the blood becomes so great that usually the venous pulse becomes much altered. In the first instance the type of pulse may be auricular, but the powerful action of the hypertrophied right ventricle pumping the blood back through a large incompetent tricuspid orifice profoundly modifies the character of the venous pulse. I have already pointed out that the wave *a* in Fig. 1 is due to the ventricle. Usually it appears later than the arterial pulse, as in Figs. 2 and 14, because the auricle in diastole, interposed between the ventricle and the veins, absorbed the first portion of the ventricular backflow. When this regurgitation takes place the ventricular backflow becomes so large and forcible that it distends the auricle; the auricular wall becomes so thin that it gradually loses the power of producing any effect by its systole upon the regurgitant blood. Its systole being ineffective, its diastole is ineffective, with the result that the wave due to the ventricle gradually increases, while that due to the auricle decreases, until finally the ventricular wave occurs coincidentally with the arterial pulse, and no trace of the auricular wave remains. Thus in Fig. 3 there is but one large

Fig. 3.—Venous pulse of the ventricular type taken synchronously with the radial pulse. As the jugular pulse is nearer the heart than the radial, the difference may be found by calculating the time between the carotid and the radial. In this and some of the other tracings a few beats of the carotid are taken along with the radial for this purpose. (From a case of mitral stenosis and tricuspid incompetence.)

wave synchronous with and due to the ventricular diastole. Whenever there is a venous pulse of this ventricular type there is almost certain to be a pulse in the liver of the same

Fig. 1.—Tracings of the venous pulse, taken synchronously with the carotid pulse. *a*, Ventricular wave due to the systole of the right ventricle; *b*, auricular wave due to the systole of the right auricle; *y*, ventricular depression due to the diastole of the right ventricle; *x*, auricular depression due to the diastole of the right auricle. The notch on the ventricular wave, *a*, indicates the time of closure of the pulmonary valves; *E* and *E'* the period of ventricular outflow through the right and left arterial orifices. (From a case of tricuspid stenosis.) These letters have the same significance in the subsequent tracings. (The method by which the tracings in this paper were taken is fully described in the *Journal of Pathology and Bacteriology*, February, 1894.)

Fig. 2.—Tracings of the carotid and jugular pulses taken at the same time. The letters have the same significance as in Fig. 1. *c* in this and other tracings is a wave due to the carotid impulse. (From a case of pregnancy.)

Fig. 7.—Tracing of a venous pulse of the auricular type, taken at the same time as the radial pulse. (From the same patient as Fig. 6.)

Fig. 14.—Venous pulse of the auricular type from a case of chlorosis showing the same features as occur in the venous pulse of tricuspid stenosis. Compare with Fig. 7.

Fig. 11.—Tracing of an auricular liver pulse taken at the same time as the carotid pulse to show that the fall of the auricular wave, *b*, occurs earlier than the fall after the wave *p* in Fig. 10 (from the same patient as Fig. 6.)

Fig. 13.—Simultaneous tracing of the abdominal aortic pulse and the liver pulse, to show the independence of the liver pulse. (From the same patient as Fig. 5.)

Fig. 8.—Tracing of the apex beat and auricular liver pulse, taken simultaneously, to show the slight notch on the ventricular wave *a*, corresponding to the notch on the venous pulse in Figs. 1, 7, and 14. (From a case of tricuspid stenosis.)

Fig. 12.—To illustrate the same thing as Fig. 11 (from the same patient as Fig. 9.)

Fig. 10.—Simultaneous tracings of the carotid pulse and of the movement communicated to the liver by the filling and emptying of the ventricles (false liver pulse).

Fig. 4.—Liver pulse of the ventricular type. (From the same patient as Fig. 3.)

Fig. 6.—Simultaneous tracings of the liver and radial pulses. The liver pulse is of the auricular type. (From a case of tricuspid stenosis.)

type (Fig. 4). On account of the different manner in which the blood produces the wave in the vein and the liver, there is a slight difference in the character of the wave. In the vein the blood regurgitates into a thin-walled tube, collapsed and partly empty, while in the liver it distends a more or less sponge-like substance, and the limit of distension is not reached so quickly as in the vein.

The regurgitant waves of blood from the ventricle act directly upon the vein and the liver in the case from which Figs. 3 and 4 were taken, but if there be between the auricle and the ventricle a diaphragm-like interposition in the shape of stenosed tricuspid valves, there is obtained a pulse of a different character. The auricle does not become so enormously distended, and in consequence it never loses its power of independent contraction. Rather it would seem to gain greater force from compensatory hypertrophy, inasmuch as the wave due to it distends the vein enormously in some cases. Fig. 1 was taken from a case of tricuspid stenosis, where the bulbous enlargement formed by the junction of the subclavian and jugular veins had become of great size, and the wave due to the auricular systole could be detected at the distance of many yards. The valves at the root of the jugular vein immediately above the bulb were competent, so that no wave extended up into the neck. The closure of these valves produced a distinct sound, synchronous with the systole of the auricle, readily audible on auscultation, and of a loudness equalling the first sound of the heart. The liver in this case became at times enlarged, and could be felt pulsating, and, as will be seen from Fig. 5, the pulse in the liver was synchron-

Fig. 5.—Simultaneous tracings of the venous and liver pulses. Both are of the auricular type. (From the same patient as Fig. 1.)

nous with that in the jugular bulb. Here there was both presystolic tricuspid and mitral murmurs during life, and after death there was found stenosis of both tricuspid and mitral valves. The wave *a*, due to the ventricular systole, is not so evident in the liver pulse as it is in the other cases. In Fig. 6, for instance, it is a very marked feature. When a venous pulse exists it is of the same type as that of the liver pulse. Fig. 6 is from the same patient as Fig. 7, and the identity of the waves *a* and *b* due to the ventricle and auricle respectively can readily be made out in both tracings. There are certain features absent from the liver pulse that are evident in the venous pulse. Thus the notch at *a'*, due, as I have suggested, to the closure of the pulmonary valves, is absent. This I take to be due to the gradual distension of the liver blurring out the more delicate features. Occasionally, however, it can be demonstrated as in Fig. 8. The wave *c* of the venous pulse (Fig. 7) is absent from the liver pulse (Fig. 6). From a variety of reasons this wave, which can be recognised in tracings by many observers, I have been able to demonstrate as due to the carotid impact, and its absence from the liver pulse is one of those reasons. (Fig. 9.)

There are three difficulties which occur in differentiating the liver pulse. First, the movements of the liver produced by the action of the heart; secondly, the pulsation of the abdominal aorta; thirdly, the liver pulse of the ventricular type in its early stage. Concerning the first of these conditions, there are certain distinct movements of the liver caused by the filling and emptying of the ventricles. Such cases occur when there is enlargement of the heart, and it can be demonstrated even when there is not much cardiac increase, but when the lax abdominal walls permit the under surface of the liver to be explored. Thus Fig. 10 is taken

Fig. 9.—Simultaneous tracings of the venous and liver pulses showing the absence of the arterial wave *c*, from the liver pulse. From a case of tricuspid stenosis.)

from a case of aortic disease with no affection of the mitral or tricuspid valves. It will be seen that during the diastole of the ventricles, while the ventricle is filling and therefore enlarging, the liver is depressed, producing the wave in the tracing marked *p*. As soon as the ventricle begins to empty it shrinks and the liver recedes, producing a fall in the tracing exactly at the same time as the blood wave from the ventricle appears in the carotid. One distinguishing feature is that the fall occurs in this liver movement a little later than it occurs in the auricular liver pulse. That the time is very near will be evident when it is considered that the auricular contraction which produces the wave in the true liver pulse is immediately followed by the ventricular systole which causes the fall in the tracing of the false liver pulse. There is but a small interval of time between these events, and by an accurate comparison of the time in the false and true liver pulse one can make out the difference (compare Fig. 10 with Figs. 11 and 12). In addition, in this false liver pulse, caused by the emptying and filling of the ventricle, there is never the ventricular wave *a*, which is present in the tracings of the liver pulse.

That the auricular pulse is not due to the abdominal aorta is evident from this tracing (Fig. 13), which is a tracing of the pulse of the abdominal aorta taken simultaneously with the liver pulse. It will be at once seen that the wave *b* due to the auricle precedes the abdominal aortic pulse.

From what has already been said the time of the occurrence of the liver pulse of the ventricular type is different from that of the auricular type (see Fig. 4). My observations tend to confirm me in the opinion I expressed some years ago that the wave *a* in the tracings of the auricular venous pulse is really due to the ventricular systole, and, as the auricle in cases of tricuspid incompetence from mitral disease becomes more enfeebled, the wave due to it becomes smaller, while that due to the ventricle increases, till finally the auricular wave disappears and the ventricular is the only one left. It may be that the liver pulse in the early stages of tricuspid incompetence without stenosis will show this wave. On looking over my tracings I am not certain but what I have detected such a thing. But I have not been able to follow up such cases where the suspicion has arisen.

It is, then, the presence of the liver pulse of the auricular type that I suggest as being the diagnostic sign of tricuspid stenosis. The frequent absence of a presystolic murmur of tricuspid origin causes stenosis of the tricuspid valves only to be recognised on the *post-mortem* table in the majority of cases. That the stenosed tricuspid valves may, by protecting the auricle and preserving its functions, tend to modify the extreme backward pressure of blood upon the venous system, and thus prolong life, cannot be certainly averred; but in the study of these cases it has seemed as if life had been prolonged.

It will be noted that it is the liver pulse and not the venous pulse of the auricular type that I look upon as being the distinctive sign. The venous pulse is a frequent phenomenon in the mildest forms of functional dilatation of the heart (See Figs. 2 and 14) as in the most severe. By functional dilatation I mean that form when there is no organic disease of the valves. A liver pulse never occurs in such cases. The reason for this, I suspect, lies in the different causes producing enlargement of the heart. In cases of regurgitation from the heart due to valvular disease, there is a time when the

muscular substance is healthy, and with increased work it hypertrophies, and, acting with great vigour, sends the blood backwards as well as forwards with great force. While a feebly-contracting auricle may produce waves in a thin-walled vein, it requires a greater force to distend the sponge-like substance of the liver. In cases of functional dilatation the proximate cause of the dilatation is the weakness of the muscular walls of a heart, so that instead of a hypertrophic muscle producing an abnormally powerful contraction, there is a weakened muscle producing an abnormally feeble contraction. Whether the explanation be sufficient or not, I have sought carefully among a very large number of patients (amounting to many hundreds), with a venous pulse of the auricular type due to functional dilatation, but I have never found a true liver pulse.

The evidence for the statements here made is based on the examination of 7 cases of tricuspid stenosis; 5 of these cases have had the condition confirmed by *post-mortem* examination. While these cases are too few for one to be absolutely dogmatic, yet they are more numerous than usually falls to the experience of any single observer. A very large number of cases of liver pulsation of the ventricular type, due to great tricuspid incompetence and other cases of heart disease where there was no stenosis of the tricuspid orifice, have been carefully examined. In no instance, except those mentioned, has there been the slightest evidence of the liver pulse of the auricular type.

A CASE OF ANEURYSM OF THE HEART.

By T. SYDNEY SHORT, M.D.,

Physician to the Infirmary and Assistant Physician to the General Hospital, Birmingham.

W. H., aged 33 years, a small and somewhat delicate-looking man, first came under my notice in March, 1893. He was suffering from syphilitic enlargement of the testicles; the primary sore having occurred when he was 17 years of age. I had occasion to examine the heart carefully, and I know that nothing abnormal was then detected from physical examination. Nearly three years elapsed, and in December, 1895, he came to see me, complaining of dyspnoea on exertion, dry cough, general weakness, and loss of appetite, all of which had existed for six weeks. His heart was enlarged, the apex was just outside the vertical nipple line, and a loud systolic murmur could be heard almost all over the cardiac area, and was loudest at the apex. The pulse was about 100 a minute, of fair quality and regular. The lungs were quite free from adventitious sounds, but it was noted that with inspiration cogwheel interruptions existed all down the front of the right side. These were not heard at all on the left side. He said that soon after I first saw him in 1893 he gave up his work as a stamper, because he felt unable to bear the strain of so arduous an occupation, and took a small beerhouse. He was, and always had been, an abstemious man himself, a statement his wife corroborated, and as he was now doing no hard work he could not account for his heart trouble in any way. He had never had rheumatism or scarlet fever. For six months he was under my treatment, and improved very much on digitalis, strophanthus, and iron, so that in June of 1896 he was able to get up and downstairs without any dyspnoea, and felt well in himself. The heart's apex was now in the nipple line, the action was regular in rhythm but occasionally varied a little in force; the pulse was 96; the loud systolic murmur persisted. For four months he went on very well without any treatment, and then one day he carried several buckets of water up and downstairs, and washed out a cellar floor. He felt very fatigued after this; the dyspnoea returned, and some swelling of the feet ensued. This was in October of 1896, and he came once more to see me. He looked very ill. His face was swollen and of a peculiar leaden hue, suggesting argyria, but differing from that condition in that the shadows alone were blue, and the lighter parts of the face, namely, the cheeks and the forehead, were of a waxy and deathly pallor. The heart at this time was much enlarged; the apex beat was in the fifth space, half an inch external to the nipple line, and diffused. There was marked epigastric pulsation. A loud harsh systolic murmur was heard all over the cardiac area and, as before, was most distinct over the

apical region. The second sound at the base was reduplicated. The veins of the neck were distended but did not pulsate; the whole of the lower part of the neck looked bloated and swollen. The liver was enlarged and tender; the spleen was not enlarged; the urine contained no albumen. He said he was vomiting all his food, a result, I thought, of congestion of the stomach. He had dyspnoea unless at rest, and complained principally of a distressing bursting feeling over the heart, eased by lying flat on his back.

It was a most unusual sight to see a man livid, and apparently dyspnoeic, comfortable in the recumbent posture only, and unable to sit up without distress. From the symptoms and the complete absence of any signs of aortic aneurysm I thought there must be some communication between the right and left sides of the heart: and knowing that he was well until his 33rd year, and seeing that his nails were only just beginning to club, I thought it unlikely that he suffered from any congenital defect of his heart. It seemed more probable that he suffered from some syphilitic disease of the septum and this was the diagnosis made. He was so much worse altogether, that I advised him to come into the hospital under the care of Dr. Simon, who has most kindly allowed me to make use of the hospital notes of the case. Dr. Simon agreed with me in thinking that there was some defect in the cardiac septum causing admixture of the arterial and venous blood. Careful dieting and half-minim doses of creasote in capsules relieved his sickness, but otherwise he was not improved, and, becoming restless and fidgety, he decided to go home after a fortnight's stay in the wards.

After he left the hospital I saw him several times with Dr. Vincent Jones, who took charge of the case and visited him daily. The oedema increased with alarming rapidity, spreading from the legs to the penis and scrotum, then involving the lumbar region, and finally the skin of the chest and back. His colour varied from day to day in the depth of the cyanosis, but was always of the peculiar livid leaden colour already mentioned. The heart sounds varied also; some days distinct presystolic and systolic murmurs were heard over the apical region with marked triple rhythm of the beat, and within a few hours the rhythm would become double again, with only a loud blowing systolic *bruit* heard over the precordia and also over the scapular region at the back of the chest. It may be especially mentioned, in face of the occasional presystolic murmur, that the usual signs of mitral stenosis had been quite absent throughout. More than once a distinct to-and-fro friction was heard, very like a pericardial rub, but there was never any evidence of distension of the pericardial sac with fluid. He insisted upon getting up for some hours every day, and seemed no worse than when he was in bed. His great trouble lay in attacks of intense dyspnoea, which occurred once or twice in the first week, and nearly killed him. He declared that they began with wind in the stomach, and were eased when he could belch it up. Keeping the stomach as empty as possible, and especially forbidding large quantities of fluid food, eased him considerably. With the exception of these attacks he never suffered from shortness of breath, so long as he abstained from moving about, throughout the whole of his illness. No drug seemed now to have any beneficial effect; digitalis, I thought, made him worse. He wandered at night, and latterly in the day time as well. He died quite suddenly and quietly on the night of the fourteenth day after he left the hospital. His wife was with him at the time. He spoke to her and said that he felt very tired and wanted to go to sleep; then he turned over on his side, and in about ten minutes his jaw dropped and he changed colour. There was no dyspnoeic paroxysm at the end. Three days before his death his left axillary vein became plugged, causing oedema of the arm and hand and much pain.

NECROPSY.

Report by Dr. STANLEY. Permission was granted to examine the heart only. The heart weighed 1 lb. The sac was somewhat distended, and contained semi-opaque fluid. The surface of the epicardium was irregularly granular, and also showed some chronic thickening, as from previous pericarditis, this being most marked over the surface of the right auricle and upper portion of the right ventricle. At one place there was a fibrous adhesion connecting the two pericardial surfaces. On opening the left ventricle an