upstroke) and partly from the right ventricle (the fall). This tracing shows that both ventricles shared in the slow action.

In these four tracings (Figs. 1, 2, 3, and 4) there seems to be graphic evidence of the contractions of all four chambers of the heart. The small waves (a) in Fig. 1 are jugular waves, due to contraction of the right auricle; the small waves in Fig. 3 appear to be due to contraction of the left auricle; the contractions of the left ventricle are shown in Fig. 3, taken from the (left) apex beat; and the contractions of the right ventricle are shown in Fig. 4.

Fig. 3.—Tracing from the apex, showing the infrequent contractions of the left ventricle. The small waves between the large beats are due to the systole of the left auricle.

Fig. 5 appears to be wholly right ventricle and shows the inverted cardiogram in contrast with that represented in Fig. 3 from the left ventricle. Fig. 6 is, no doubt, left ventricle and shows the long pauses with auricular waves very distinct. Fig. 7 is a simultaneous tracing of the apex beat and of the pulsation in the neck; it was made on April 11th.

Fig. 4.—Taken over the right ventricle, showing a great fall during systole (the inverted cardiogram characteristic of the systole of the right ventricle); it also shows that the right ventricle participated in the infrequent action with the left ventricle.

April 11th. The patient appears to be slightly better this morning. The ventricular contractions and the radial pulse number 30 per minute. No change in the strength and character of the heart beats; a long, loud systolic and a short diastolic murmur still present; the radial pulse is rather weaker. During this day the patient had several attacks of very severe angina

Fig. 5.—Tracing from the fifth left interspace, made on April 11th, 1907. This tracing appears to be wholly due to the right ventricle, and shows the inverted cardiogram in contrast to Fig. 3.

pain; he stated that he felt as if he were being crushed between two buffers. The radial pulse during the attacks of pain was almost imperceptible and somewhat irregular. The jerking movements of the head with the apex beats of the heart still continue. There has been a great deal of vomiting. Amyl nitrite inhalations, strychnine subcutaneously, and a menthol mixture were prescribed.

Fig. 6.—Tracing from the apex beat of the heart made on April 10th, 1907. This tracing appears to be due to contraction of the left ventricle, and shows the long pauses between the ventricular contractions with small auricular waves interposed.

April 12th. Much worse; very restless all day. Pulse down to 24 per minute. The pain in the heart has been very severe and distressing. Morphine has been given at frequent intervals. Towards midnight the pulse went down to 22. The vomiting has not been so bad to-day. The pupils now measure from 5 to 6 mm. in diameter. The patient is perfectly sensible.

Fig. 7.—Combined tracing from the apex beat and neck made on April 11th, 1907.

April 13th, 2 a.m. The cardiac pain was so severe that morphine had to be given on several occasions. Pulse 19; respirations 17. An interval of 8 seconds was counted between two of the cardiac beats on one occasion. The radial pulse is very irregular. The patient died at 4.30 a.m.

Post-mortem Examination.

This was made by Dr. Shennan on April 13th, 1907. Both lungs were in places attached to the chest walls by old adhesions. The pericardial sac was obliterated as the result of old

pericarditis. The exterior of the pericardium was adherent to the thoracic wall opposite the apex of the heart.

The heart was injected with formalin in order that the auriculo-ventricular bundle might be carefully dissected out.

On passing the fingers through the auricles a large calcareous mass could be felt, on the upper border of the inter-ventricular septum posteriorly, apparently measuring about 1½ in. antero-posteriorly by about ½ in. vertically and about 1½ in. transversely. On further examination, after fixation, this corresponded to the anterior cusp of the mitral valve, which was extremely calcareous.

There was some bronchitis and congestion of the lungs, but no consolidation. The liver was enlarged, congested, and fatty. The kidneys were large and congested, with some swelling and fatty change. The spleen was firm, large, and congested.

Subsequent Examination of the Heart.

General adherent pericarditis; dilatation of all the cardiac cavities; hypertrophy of the ventricles; old endocarditis with calcareous deposits in the aortic and mitral valves; the anterior flap of the mitral valve markedly calcareous; some narrowing of the mitral orifice; thickening of the tricuspid with some dilatation of the orifice; pulmonary cusps slightly but uniformly thickened.

Dr. E. B. Jamieson of the Anatomical Department kindly dissected out the auriculo-ventricular bundle. His report on its condition is as follows:

The auriculo-ventricular bundle of His has apparently undergone fibrous degeneration, and the few muscular fibres remaining on the surface are very pale. A calcareous nodule situated at the right end of the attachment of the anterior mitral flap extends into the septum, invades and well nigh obliterates the bundle towards its auricular extremity.

NOTE ON THE PULSUS BIGEMINUS.

By E. E. LASLETT, M.D., B.Sc.,

HULL.

My object in publishing this note is to draw attention to a curious, and I believe hitherto undescribed, form of jugular pulse associated with the pulsus bigeminus, which in itself is not a very uncommon phenomenon.

The patient, a man aged 40, was first seen on June 27th, 1906. He had been working at his trade as a painter up to a week before. He had had heart disease to his knowledge for seven years but had never been laid up on this account. At one time he weighed 16 st., but for some months he had been losing flesh. He had been temperate as regards both alcohol and tobacco.

He suffered greatly from dyspnoea, which necessitated his being propped up in bed. He was pale and flabby, and there was considerable oedema of the legs and trunk but no ascites. The pulse was 100, regular, with a large, markedly collapsing beat, and the marked throbbing of the arteries, characteristic of aortic disease, was very noticeable, and perceived by the patient himself. The heart was enlarged to the right, and to the left to 1 in. outside the nipple line. There were double murmurs at all areas of the heart but no thrill could be felt. It was obviously a case of failure of compensation in aortic disease. Tracings taken at this time showed that the pulse was quite regular, while there was a very large auricular wave in the jugular pulse, which was otherwise normal. He was much troubled at night with cardiac asthma, and the usual drugs were tried, without any but temporary relief to this symptom. In spite of treatment by rest in bed and cardiac drugs the oedema and other symptoms of distress persisted, and he died on July 31st. For the last fortnight he was taking 5-minim doses of tincture of strophanthus with citrate of caffeine.

A week before his death his pulse took on a bigeminal character, which was continuous, and persisted until the day before he died, when it became too feeble to detect its true rhythm. The tracing of the jugular and radial pulse when this condition was present is shown in the accompanying figure, from which it will be seen that at the extra beats which are the cause of the bigeminal rhythm, the auricles contracted during the ventricular systole, and there is one large wave synchronous with the extra beat in the radial artery. The increased size of this wave is due, as Mackenzie has pointed out, to the fact that the right auricle is unable to empty itself into the contracted ventricle, and therefore a larger quantity of blood than usual is sent into the jugular veins. The unusual feature in this tracing lies in the fact that the combined wave commences distinctly beyond the middle line—that is, nearer to the post-extra-systolic auricular wave than to the pre-extra-systolic. This puzzled me so much that I sent this and another tracing of two days' earlier date to Dr. James Mackenzie, who most kindly examined them

The figure shows the character of the jugular pulse associated with the bigeminal rhythm in the radial pulse. The appended diagram explains the nature of the events. *As* signifies auricular systoles; *Vs*, ventricular systoles; and *A-V*, the conduction between auricle and ventricle.

for me. He confirmed the existence of this peculiarity, and kindly indicated the diagram appended to the tracing, which shows very clearly the nature of the events. Mackenzie divides extra-systoles into three types—auricular, ventricular, and nodal—the ventricular including the rare form of interpolated extra-systole. He has suggested that the probable origin of the extra-stimulus lies somewhere in the remains of the primitive cardiac tube, in auricle, ventricular bundle, or auriculo-ventricular node. In the nodal extra-systole the auricles and ventricles contract together, or nearly so, and usually prematurely. It may, however, happen that the combined wave appears at the normal period of auricular contraction, and it may then be difficult to determine whether the extra-systole is of nodal or ventricular origin. In the present instance, from the regularity and persistence of the bigeminal character, and the fact that the auricular wave is always synchronous with the ventricular, it is probable that the extra-systoles are of nodal origin. Although there is no time record in the tracing, it may be taken as certain that the *a-c* interval is increased. In an earlier tracing, in which the combined wave was nearer the middle point, the *a-c* interval was less. The increase of the *a-c* interval is no doubt due to the incomplete restoration of conductivity as a result of the short period between the extra-systole and the following normal auricular beat.

I am unable to explain this peculiar rhythm. It is evident that alternate auricular contractions failed to appear at the normal period, but whether the sinus rhythm remained regular or not it is impossible to say. On the day of death the pulse became very feeble and quick, and the patient was too ill to allow of a tracing being taken. It is consequently uncertain whether the heart in the final stage became continuously irregular (nodal rhythm).

THE INEFFECTIVENESS OF CALCIUM SALTS AND OF CITRIC ACID AS USED TO MODIFY THE COAGULATION TIME OF THE BLOOD FOR THERAPEUTIC PURPOSES.

WITH A DESCRIPTION OF A MODIFICATION OF MCGOWAN'S METHOD OF ESTIMATING THE COAGULATION TIME OF THE BLOOD.

By T. ADDIS, M.D., M.R.C.P. EDIN.,
CARNEGIE SCHOLAR.

By means of a more accurate method than Sir A. E. Wright's, I have shown that he was mistaken in his statement that soluble calcium salts and citric acid are capable of altering the coagulation time of the blood of people in health. The fact that this initial experimental result of his cannot be confirmed is sufficient in itself to throw great doubt on his later work in this connexion. Since 1893 he has published a number of papers on the use of calcium in the treatment of haemophilia, and of haemorrhage in many other conditions, and also in what he terms "serous haemorrhages"—a group of conditions said to be characterized by an abnormally long coagulation time, which could be cured when the coagulation time was reduced by the administration of soluble calcium salts. Further, citric acid was recommended as a prophylactic against thrombosis in typhoid fever. Although clinical

reports as to the value of this method of treatment have been conflicting, the general idea that the coagulation time in disease can be shortened or lengthened at will, by giving the proper dose of calcium lactate or citric acid, has never been effectively disputed, and has been very widely accepted and acted upon.

It was not possible to test the accuracy of this belief by the method of estimating the coagulation time of the blood which I have described, since it is not applicable clinically because of the presence of a greater or less degree of auto-agglutination of the red blood corpuscles in very many pathological conditions. Another method was therefore necessary, and the modification of McGowan's method, which is described below, is one which is applicable clinically, and which yields results which can be relied upon within certain limits.

In reviewing the different methods I found that the means adopted by Sabrazès and McGowan of determining the presence of a certain definite amount of coagulation is more accurate than those employed in the other methods, although it is not possible to obtain constant or comparable results with either of their methods, because in the case of the former the means of maintaining a constant temperature is not sufficient, and in the latter is wanting altogether.

McGowan believed that for clinical purposes it was superfluous to have an apparatus to maintain his glass tubes at a constant temperature, since variations of temperature between 15° C. and 20° C. did not appreciably affect the coagulation time.

This, however, as I have already shown, is very far from being the case. With my method the following coagulation times were found to correspond with the temperatures between 15.5° C. and 20.5° C.:

15.5° C., 11 min. 46 sec.;	18.5° C., 7 min. 34 sec.;
16.5° C., 10 min. 10 sec.;	19.5° C., 6 min. 2 sec.;
17.5° C., 8 min. 7 sec.;	20.5° C., 5 min. 22 sec.

The necessity of the maintenance of a constant temperature is, therefore, fully apparent. The variable results which are obtained by these and other coagulometers are said by those who use them to indicate true variations in the coagulation time of the blood, but the coagulation time is found to be constant when a more accurate method is used, and those apparent variations are really due to experimental errors, of which the most important is a deficiency in, or a total neglect of, the maintenance of a constant temperature for all observations.

Description of the Method.

McGowan's method consists in partially filling a capillary glass tube, 6 in. long and 1.5 mm. in diameter, with blood under certain precautions, sealing up one end, and at intervals of a minute breaking off a small part until a fine fibrin thread is seen on slowly drawing the fractured ends apart. The coagulation time is the time elapsing between the filling of the tube and the first appearance of fibrin.

Undoubtedly the simplest and most accurate way to keep the tubes always at the same temperature would be to use a gas regulator. But in the wards of most hospitals there is no gas, and it is absolutely necessary that the whole procedure should be carried out by the bedside of the patient. An electrical thermostat would be sufficient, but is expensive, so it was better to dispense with any mechanical regulator, and to make use of the following device.*

When a closed cylindrical vessel is placed in the centre of a similar but considerably larger vessel, and both are filled with

* The apparatus can be obtained from Paul Altman, scientific instrument maker, 56, Louisen Strasse, Berlin, for 36 marks.