notes of 15 cases, has been given by Dudgeon.¹ In the great majority of cases (13 out of the 15 in the series quoted by Dudgeon) the affection comes on after a fracture of the arm or forearm has been treated by splints and bandages. But in a case of Mr. Wallis it arose after the use of an Esmarch's bandage applied for one hour to the arm; and in a case of Professor Schloffer it resulted from thrombosis of the axillary artery. This contracture is caused by pressure on the muscles of the forearm, depriving them of blood. Some of the fibres of the delicate and highly organized striated muscle then die or undergo rigor mortis, and are replaced by fibrous tissue or, in rare instances, by bone (myositis ossificans). This causes contracture, wasting, and pseudo-paralysis. The feature in which both my cases differ from most of the recorded cases of ischaemic paralysis is that pronation is much more marked than flexion of the fingers and wrist. But this is accounted for by the fact that there has been a constant effort to move the fingers, which has probably overcome the flexor contraction, of which the first case gives a distinct history. The pronation, on the other hand, is the position of rest assumed by the hand when held in a sling, and also is that used in all ordinary acts, such as writing or using a knife or fork. So that there was probably no voluntary or systematic effort made to stretch the contracted pronators, and now you can feel in both these boys a tense, fibrous cord in the position of the pronator radii teres when supination is attempted.

In 6 out of Dudgeon's series of 15 partial or complete pronation of the arm was left as a permanent condition in Volkmann's contracture.

The actual shortening of the bones is difficult to account for. It is present in almost all cases of Volkmann's contracture occurring in children, and therefore its presence in these two cases confirms the idea of the condition being one of this contracture. The most likely explanation seems to be that when the muscles become atrophied and partially functionless, their blood supply is much lessened; and as all the blood vessels which supply the growing ends of the bones are branches of muscular vessels, it is easy to understand how a lessened blood supply will lead to diminished growth.

It seems at first sight a contradiction in terms to speak of ischaemic paralysis as occurring in haemophilia. But this contradiction is only one of words and not of ideas. In both the cases I have described there was a great haemorrhage into the soft tissues of the forearm, and this, confined under the tense skin or fascia, compressed the muscles and brought their circulation to a standstill. There was too much blood outside the muscles and too little in them.

In the case of the younger boy no splints were used at all, and in that of the elder his description leaves no doubt that the typical contracture had developed before the splints were applied. As far as I am aware, there is no other instance recorded of the occurrence of this peculiar contracture in haemophilia, and it is very remarkable that I should have come across these two cases at the same time who happen to live in neighbouring streets.

In conclusion, I must say a few words about the treatment of haemophilia. From a surgical point of view it may be summarized as—rest and pressure. When the joints become swollen they should be firmly bandaged, and kept at rest until the effusion subsides. It is by neglect of this treatment that the ligaments become stretched and weakened, and the changes of osteo-arthritis hastened. For the external bleeding do nothing but keep the patient in bed. One of the fatal cases I have referred to was bleeding from a scratched gum for fourteen days, during which he walked about from doctor to doctor, trying each new remedy, and bleeding steadily in spite of all, until at last he found refuge in the infirmary, only to die from the resulting anaemia. No remedies, local or general, have any effect on the haemorrhage, and therefore leave that alone and turn all your attention to treating the patient's general condition. If this rule was always followed from the first onset of the bleeding probably many lives would be saved.

REFERENCE.

¹ *Lancet*, January 11th, 1902, p. 78.

COR TRILOCULARE BIATRIATUM.

By J. DIXON MANN, M.D., F.R.C.P.,
PHYSICIAN TO THE SALFORD ROYAL HOSPITAL.

THE following case borders on the unique:

History.

On December 22nd, 1905, I saw a man, aged 35, in consultation with Dr. Webb of Gorton. The patient was propped up in bed, and presented the usual appearance of one suffering from chronic heart disease. His breathing was difficult, but he was not cyanosed nor were the fingers clubbed; he had a cough with bronchial expectoration. There was some oedema about the ankles. The area of cardiac dullness was considerably increased; the cardiac impulse was more extensive than usual, the apex beat being difficult to locate. The centre of the impulse was about the fifth interspace, a little within the nipple line. There was no thrill. On auscultation, the first sound was found to be replaced by a loud, rushing, systolic murmur, which extended over the whole cardiac area. No other murmur could be distinguished. The second sound was muffled. Some amount of oedema was present at the base of the lungs. The liver dullness was slightly increased. There was no ascites.

Subsequently I learned that the patient improved for a time; he then relapsed, and expressed a strong wish to go into hospital. He was so determined that (whilst recognizing his critical condition) Dr. Webb reluctantly consented, and he was admitted under my care on May 28th, 1906, into the Salford Royal Hospital. His condition was most distressing. He was deeply and universally cyanosed and was gasping for breath. The heart beats were tumultuous and irregular; the veins were overfilled, and those in the neck pulsated. There was general oedema, with effusion into the pleural and peritoneal cavities; the lungs were oedematous. The urine was almost suppressed. The surface of the body was cold and clammy. The patient was fully conscious, and earnestly pleaded for something to relieve the breathing; but in spite of all treatment it became worse, and he died on the second day after admission.

Necropsy.

An examination of the body was made by Dr. Mair, pathologist to the hospital, on May 31st, who reported as follows:

General oedema of the body, especially marked in the lower extremities, penis, and scrotum. Cyanosis of face, neck, and arms. There is considerable serous exudate in the pleurae on both sides. The lungs show passive congestion and oedema.

The Heart: The ventricular portion is much enlarged, and there is no difference in thickness of wall indicative of right and left ventricle. On incising the ventricular wall, it is found that there is complete absence of the septum between the two ventricles. Into the common ventricle open the mitral and tricuspid orifices in their normal positions. Two arterial trunks emerge from the ventricle in the relative positions of aorta and pulmonary artery. The anterior of these gives off the two coronary arteries; its wall is thicker, while its lumen is smaller, than that of the posterior trunk. The latter is thin-walled, and the valves at its commencement are extensively sclerosed and calcified. Unfortunately, these trunks were not followed to their distribution; but it seems likely that the anterior is the aorta and the posterior the pulmonary artery, there being thus transposition of these vessels. The auricular portion of the heart presents nothing abnormal, except that the foramen ovale is patent. The heart weighs 1½ lb.

There is a large amount of serous exudate in the peritoneal cavity. The liver shows advanced "nutmeg condition." The spleen is enlarged and firm. The kidneys show passive congestion.

The illustration is reproduced from a photograph, taken after Professor Lorrain Smith had arranged the incised wall of the ventricle, so that the four openings of the arteries and auricles may be seen communicating with the one cavity. The mouth of the aorta, slit open, is seen immediately above a glass rod, there placed to hold the ventricular wall apart. Behind the left end of the rod a cavity is seen, an inch and a quarter in depth and five-eighths of an inch in diameter; this appears to represent a rudimentary right ventricle.

Professor A. H. Young, to whom I am indebted for the photograph from which the illustration is taken, exhibited the heart and described the developmental anomalies causative of the malformation at a recent meeting of the Anatomical Society of Great Britain and Ireland. A description will appear in the *Journal of Anatomy*.

Malformation of the heart as above described is exceedingly rare, and when encountered in adult life it is phenomenally so. W. T. not only lived to the age of 35 years, but, with the exception of the last six months, he enjoyed average good health throughout the whole of his life. I am indebted to Dr. Webb for the following

THE Hungarian Chamber of Deputies has passed a Bill providing that insurance against sickness and accidents shall be compulsory.

short history furnished by letter after the result of the autopsy had been communicated to him:

"I have known W. T. for six or seven years. As a boy he was not really weakly, but he was unable to join much in boys' sports. On one or two occasions I have attended him for a day or two on account of breathlessness, cough, or headache, with gastric trouble; on each occasion a little medicine soon put him right again. Indeed, till the last long illness of several months, I cannot recollect that I ever saw him in bed. Two or three years ago he took a trip to America, which did him good. From my own personal knowledge, and from what I elicited from his family, he was *never cyanosed* till the last three weeks of his life."

The literature of cardiac malformation reveals only a few instances in which the possessor of a three-chambered heart lived to adult age. Kreyzig¹ records a case seen by Wolf of a man who suffered from cyanosis and died at the age of 23 years, whose heart was found to contain two well-formed auricles, with distinct auriculo-ventricular apertures, and only one ventricle, from which two arteries took their origin. The pulmonary valves were imperfect, and an opening was present in the foramen ovale. Wittke² found, in the body of a man aged 24, that the heart had only one ventricle, with walls three times the usual thickness; and without the least trace of inter-ventricular septum. The vessels of the heart are stated to have been in their normal position, their orifices being dilated. The foramen ovale was closed. This man suffered from dyspnoea and was cyanotic. Max Mann³ found complete ab-

sence of the ventricular septum in the body of a woman aged 22 years. The aorta and the pulmonary artery were transposed; the latter was stenosed at its mouth owing to adhesions of the valves; beyond the valves the artery was very much dilated. Throughout life the face, hands, and feet were cyanosed. Durozier⁴ records a remarkable case of a man, who lived to the age of 39 years, whose heart showed absence of the ventricular septum, with transposition of the aorta and the pulmonary artery. The pulmonary artery was contracted, and yet there was no cyanosis. The last two cases bear a close resemblance to that of W. T. In all three there was transposition of the pulmonary artery and aorta, with stenosis of the former. In one of the three there was pronounced cyanosis, whilst in the other two there was no cyanosis. An analysis of cases of congenital malformations of the heart points to a correlation between absence or defect of the inter-ventricular septum and stenosis of the pulmonary artery, which appears to be the commonest of associated cardiac abnormalities. The pulmonary valves in these cases are frequently described as sclerosed or adherent; in W. T. they were also calcified. It is a matter of conjecture whether the defective condition of the pulmonary valves may not be due to the excessive strain to which they are

subjected by the forcible propulsion through them of the blood, either entirely or partially, from a ventricle many times more powerful than the normal right ventricle.

The developmental aspect of the abnormality having been discussed elsewhere, there only remains something to be said from the physiological standpoint. First, as regards the mode in which the circulation was effected. So far as can be seen there was absolutely nothing to direct the blood, preferentially, either to the aortic or the pulmonary orifices. At each systole the powerful ventricle drove the mass of blood which it contained through both channels, with an efficiency that closely rivalled the function performed under normal conditions by two distinct cavities. Second, as regards the absence of cyanosis. The cause of congenital cyanosis is far from clear. Among the assumed causes are: admixture of venous with arterial blood, deficient aëration of the blood, back pressure affecting the veins, general vasomotor paralysis, with imperfect oxygenation of the red corpuscles. The cyanosis due to developmental defect of the heart is usually much more pronounced than the cyanosis of disease; a man with congenital cyanosis is a veritable *homo coerulesus*. As previously stated, the pulmonary valves have frequently been found to be diseased when the inter-ventricular septum is absent or is greatly defective; constriction

of the pulmonary artery has also been observed. Stenosis of the pulmonary artery has been reported in other cardiac malformations which were accompanied by cyanosis; this gave rise to the view that the cyanosis was due to impeded access of the blood to the lungs. A considerable number of abnormally-developed hearts have been recorded,

A. Aorta. P.A. Pulmonary artery. L.A. Left auriculo-ventricular opening. R.V. (?) Rudimentary right ventricle. R.A. Right auriculo-ventricular opening.

however, including Durozier's and my own, in which there was no cyanosis, although the pulmonary artery was stenosed, and others in which cyanosis occurred although the pulmonary artery was of normal calibre throughout. Mere admixture of venous with arterial blood is not sufficient to account for cyanosis, and to many of the cases which demonstrate this that of W. T. is to be added. It is obvious that throughout the patient's life his venous and arterial blood were mingled, yet it was not until his heart began to fail that cyanosis occurred. The cyanosis which then appeared was of the nature of that which occurs in disease apart from cardiac malformation—in capillary bronchitis, for example, when the heart is failing. Under such conditions, imperfect aëration of the blood and back pressure are probably the chief causal factors, which, however, do not come into play in cases of congenital malformation of many years' survival until disease or heart failure is superadded. Recently Variot⁵—who distinguishes congenital from asphyxial cyanosis by the fact that the former cannot be modified by the inhalation of oxygen—attributes congenital cyanosis to general vaso-motor paralysis with numerical increase of the red corpuscles which he assumes have only a feeble affinity for oxygen, and thus cause the blood to be dark in colour.

There is no doubt about the numerical increase of the red corpuscles in congenital cyanosis; this, together with a high haemoglobin percentage, has been ascertained by many observers. It seems probable that the increase is the direct result of the cyanosis or, more exactly, of the condition which gives rise to the cyanosis. If, owing to the admixture of venous with arterial blood, the percentage of oxygen conveyed by the haemoglobin is much below the normal, the internal respiration will be unequal to the demands of the tissues, which will proportionately suffer. This continuing, calls forth the resources of the organism to provide compensation. The only way open is to increase the oxygen capacity of the blood, which is accomplished by multiplying the red corpuscles and raising the percentage of haemoglobin; so that, although the individual corpuscles are still only partially oxygenated, and consequently continue to yield a deficient amount of oxygen to the tissues, their numerical excess counterbalances the deficiency. Apart from congenital malformations of the heart in which venous and arterial blood are mingled, crippling of the heart's movements appears to be capable of causing polycythaemia. Some observations of Lorrain Smith⁶ bear on this question. In a case of excessive thickening of the pericardium by which the heart was literally hidebound, and was unable to keep the blood circulating with sufficient rapidity, cyanosis occurred. Repeated examination of the blood showed increase in red corpuscles (nearly 7,000,000), with increased percentage of haemoglobin (103 to 114); the amount of haemoglobin was estimated by Haldane and Smith's⁷ CO method. Here there was no question of admixture of venous with arterial blood; but apparently as the internal respiration was insufficient to meet the requirements, the oxygen-carrying power of the blood was increased. Yet, again, chronic cyanosis with polycythaemia occurs independently of heart trouble. Several cases have been investigated by Osler⁸ and others, in which enlargement of the spleen was accompanied by chronic cyanosis and by great increase in red corpuscles—from 8 to 12 millions; the haemoglobin was also increased to 120 to 150, and even in one case to 165, per cent. A like condition of the blood, with splenic enlargement, may occur without cyanosis. Weber⁹ records such a case, in which the red corpuscles reached 10 millions, and the haemoglobin 135 per cent. In a recently published paper, Senator¹⁰ describes two cases of splenomegaly with dark red coloration of face and mucous membranes, and with the usual increase in erythrocytes and percentage of haemoglobin. The colour index was normal, so that the increase of haemoglobin was due to the numerical increase of the red corpuscles. The O tension of the blood was normal, as was also its molecular concentration. The intake of O₂ and the output of CO₂ were both above the highest normal, and closely corresponded to the increase in haemoglobin. To account for the increased internal respiration, Senator suggests either that the augmented supply of blood and oxygen stimulates the tissues to greater activity, or that a common abnormal stimulus is the cause of the increase in blood cells and of their augmented reception of oxygen, with an equivalent delivery of it to the tissues. In the same class of cases Bence¹¹ found that splenic cyanosis is not due to increase of CO₂ in the blood, as is the case in the cyanosis of disease; in other words, that the splenic cyanosis is not due to colour change in the haemoglobin. He is disposed to accept the view that dilatation of the superficial vessels is concerned in the production of the cyanosis. Both he and Senator attribute the polycythaemia to increased production, and not to delayed destruction. Bence and also Senator found the viscosity of the blood to be three or four times above the normal, and to this Bence attributes the general condition. He regards the diminished O₂ tension as only relative; it is set aside by the absolute increase of erythrocytes, so that the combination of a normal O₂ quantity with an abnormally low O₂ tension is rendered possible. Bence, Koranyi, and others have assumed, in these cases, that the haemoglobin is deficient in O₂ combining power; this view is not corroborated by Senator's case. In Weber's case this point has not yet been investigated; but he quotes observations by Boycott on two other cases of megalosplenic polycythaemia in one of which the O₂ capacity was practically normal; and in the other, although the O₂ capacity of the haemoglobin was possibly slightly deficient, the blood as a whole had an O₂ capacity

of at least 10 per cent. more than that of a normal man. Senator, Bence, and Koranyi found that the inhalation of oxygen tended to diminish the polycythaemia; in one case in which improvement was in progress, Koranyi considers that the cure was furthered by the inhalation.

It appears that any condition which gives rise to cyanosis, not necessarily of long standing, tends to produce polycythaemia; the blood from a patient suffering from severe bronchitis with dilated heart and a moderate degree of cyanosis, yielded 6,500,000 red corpuscles and 106 per cent. of haemoglobin.

The conditions which give rise to the cyanosis of cardiac malformation remain obscure. In addition, however, to the results which are directly due to the action of the malformed heart on the blood current, it appears probable that a permanently dilated vascular system shares in the production of the cyanosis.

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ON THE USE OF CALCIUM SALTS AS CARDIAC TONICS IN PNEUMONIA AND HEART DISEASE.

BY SIR LAUDER BRUNTON, M.D., F.R.C.P., F.R.S.,
CONSULTING PHYSICIAN TO ST. BARTHOLOMEW'S HOSPITAL.

FIFTEEN years ago, in the BRITISH MEDICAL JOURNAL of January 23rd, 1892, Dr. Prickett and I published a paper on the use of oxygen and strychnine in pneumonia. This paper was hurriedly put together, half of it having been written in a country house while watching a case of pneumonia and the other half scribbled in a railway train on my way back. The reason for our hurried publication was the prevalence of pneumonia at the time, and the hope that the treatment which we had found so promising might prove useful in the hands of others. The event has justified our expectation, and the treatment of pneumonia by oxygen and strychnine is now widely and successfully employed. A similar reason induces me to write this short paper on the use of calcium salts, because in the present epidemic of post-influenzal pneumonia there seems to be a great tendency to cardiac failure, and this, I think, may to some extent be averted by the free use of calcium salts which have a tonic action upon the heart.

Nearly twenty-five years ago Dr. Sidney Ringer discovered that when the frog's heart is perfused with a solution of sodium chloride in distilled water the heart soon loses its power of contraction. This result does not occur if the solution is made with tap water. Professor Ringer's scientific acumen soon led him to the important discovery that the maintenance of the contractility of the heart when tap water is used is due to the calcium salts contained in the water. This discovery is constantly utilized in physiological laboratories, but, so far as I know, has not had any practical application in medical treatment. A good many months ago I was called to see a case of pneumonia, and, fearing that the heart would fail, I thought over all the remedies which were likely to avert such an unfortunate result. It then occurred to me that calcium chloride ought to be useful. It then occurred to me it was likely to do was to increase the coagulability of the blood, but any risk of this sort was small in comparison to that from cardiac failure, so I prescribed it. It is not so easy to judge of the usefulness of calcium salts in threatened cardiac failure as it is of the action of oxygen and strychnine, because the results are not so immediately apparent. As the patient for whom I prescribed the calcium made a good recovery I was encouraged to give it again, and have done so in a considerable number of cases. In some of them a fatal result has ensued in spite of everything that could be done, but in a number of them the effects appear to me so encouraging as to deserve a wider trial for the medicine. I usually give it in 5 to 10 gr. doses every four hours, simply dissolved in water. As it is very deliquescent it can only be kept in solution. It has a very disagreeable saline taste, but this is well covered by saccharine, one minim of the elixir of saccharine containing one-twentieth of a grain of