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.

REFERENCES.

- ¹ *Krankheiten des Herzens*, vol. iii, 1817. ² *Hufeland's Journal*, vol. lxvi, 1828. ³ *Ziegler's Beiträge*, vol. vi, 1889. ⁴ *Journ. de Conn. méd. prat.*, 1885. ⁵ *Journ. de Clin. et de Therap. Inf.*, 1897, and *Ann. de Méd. et Chir. Inf.*, 1904. ⁶ *Trans. Path. Soc.*, 1902. ⁷ *Journ. of Physiol.*, 1900. ⁸ *Amer. Journ. Med. Sci.*, 1903. ⁹ *Lancet*, 1905-6. ¹⁰ *Zeitschr. f. klin. Med.*, 1906. ¹¹ *Deutsch. med. Wochenschr.*, 1906.

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

saccharine is sufficient to cover the taste of 10 gr. of calcium chloride. This mixture may be given either in water or in milk, and it does not interfere with the use of any other remedies.

I have also used it in cardiac disease, where the ventricular wall appeared to be losing power, and here also the results have been encouraging. In cases of pneumonia, where one wishes to get rapid action, I think the chloride is the best salt of calcium, but in cardiac disease other salts may be employed, such as the lactophosphate or the glycerophosphate. It is quite possible that the great benefit one frequently observes from a milk diet in cases of heart disease, may be due, in part at least, to the large quantity of calcium salts which the milk contains.

At the present moment, when there is a good deal of antivivisection agitation, it may be interesting to note that the plan of treatment by oxygen and strychnine recommended by Dr. Prickett and myself fifteen years ago, and the one which I now recommend, both owe their origin to laboratory experiments, and any benefits which may result to patients from either plan must be regarded as the fruits of experiments upon animals.

DELAYED CHLOROFORM POISONING.

By J. CRAWFORD RENTON, M.D.,

SURGEON AND LECTURER ON CLINICAL SURGERY IN THE WESTERN INFIRMARY, GLASGOW.

THE following are notes of 2 cases in which symptoms of delayed chloroform poisoning were observed in adults.

CASE I.

A. B., aged 26, was sent to the Western Infirmary by Dr. Thomson on March 2nd, 1906, suffering from acute appendicitis. She was operated on an hour and a half after her arrival and a large diseased appendix removed. The temperature fell to normal the next day, but persistent vomiting continued for two days in spite of washing out the stomach and rectal feeding. On the fourth day after operation she complained of pain in the cardiac region, which was not relieved by strychnine or digitalin, and she died on the fifth day after operation.

I am indebted to Professor Muir for notes of the *post-mortem* examinations in this and the following case.

Post-mortem Examination.—There was no trace of peritonitis, and the site of operation showed normal healing over the point where the appendix had been removed. The stomach contained a considerable quantity of altered blood, but there was no ulceration, and no source of the haemorrhage could be detected. The liver was in a condition of extreme fatty degeneration, the colour being almost a uniform yellow. The organ was not enlarged. The kidneys showed a diffuse fatty degeneration, though rather less marked than was in the liver. The cardiac muscle also showed considerable fatty change, this being pretty generally distributed. The coronary arteries were healthy. The lungs had a distinct acetone-like odour. The spleen was healthy. Microscopic examination gave confirmative results. In the kidneys the convoluted tubules, Henle's tubules, and also many of the straight tubules showed profound fatty degeneration, the cells being practically filled with small fatty globules. The fatty change in the liver was also very advanced and almost uniform in its distribution. In the heart the fatty degeneration was very diffuse, practically all the fibres containing fatty globules, but was not of extreme degree. The changes noted correspond with those in late chloroform poisoning, and point to the action of some toxic substance on the organs mentioned. There was no evidence of septicaemia or of any other condition which might explain the fatal result.

CASE II.

B. C., aged 28, was sent to the Western Infirmary by Dr. Ingram of Helensburgh. She was suffering from pyloric stenosis, and for this a gastro-enterostomy was performed on November 11th. After the operation she was much troubled with sickness and faintness, and died on November 15th. She had as anaesthetic 5 c.cm. ethyl chloride and 3½ oz. of A.C.E. mixture. The anaesthesia lasted for fifty minutes.

Post-mortem Examination.—A posterior gastro-enterostomy had been performed, and the peritoneum was in a healthy condition. The stomach showed signs of chronic catarrh, and its mucous membrane was somewhat rugose. There was no haemorrhage. The liver showed extreme fatty degeneration as in the previous case, and microscopic examination showed the condition to be of similar nature and degree. In the kidneys fatty change was present, but it was much less marked than in the previous case; and it was found on microscopic examination that the fatty globules were only present in the basal portions of the cells. The spleen was normal, and cultures made from it gave only a few colonies of the *Bacillus coli*. The heart muscle was somewhat atrophied, but was not fatty. In the right lung there was an area of chronic grey

tubules with evidence of caseation at places, but this was of small extent.

In the second case the changes in the liver and kidneys were somewhat similar to those in the first, but were of less degree. The case is possibly of the same nature, but considerable emaciation was present, and death may have resulted simply from inanition.

I record these cases owing to the importance of cases of delayed chloroform poisoning and the interest that has recently been taken, more especially regarding deaths among children. In the first case the patient had chloroform alone, in the second case ethyl chloride and A.C.E. mixture was given. This latter combination may have accounted for the symptoms in the second case not being quite so pronounced. So many feeble patients have operations done of even a more serious nature than the above and get perfectly well, that it is to a certain extent satisfactory to be able to give a definite cause as to the fatal termination in two such comparatively young patients as the above. The practical question naturally arises, What is to be done in order to prevent the possibility of acetone poisoning taking place?

CHRONIC PERITONITIS CAUSING ELEPHANTIASIS.

BY

JULIUS BERNSTEIN, and FREDERICK W. PRICE

M.B.,

M.B., M.R.C.P.,

ASSISTANT PATHOLOGIST AND

MEDICAL REGISTRAR,

CURATOR OF THE MUSEUM,

WESTMINSTER HOSPITAL.

A WOMAN of 43 years of age was admitted to Westminster Hospital under the care of Dr. de Havilland Hall, to whom we are much indebted for permission to publish the case, on September 15th, 1905. She complained of shortness of breath and swelling of the legs and abdomen.

She had been an in-patient at several other London hospitals, and we are much indebted to Dr. Jex-Blake for kindly allowing us to make abstracts of his notes taken during the patient's stay in St. George's Hospital.

HISTORY.

Patient has had five miscarriages and ten children; the menses ceased fifteen months ago. She denies alcoholic excess. In March, 1904, she began to suffer from dyspnoea on exertion, loss of strength and flesh, and languor; in April from vomiting; in June and July from protrusion of the eyeballs; in July, swelling of the thyroid gland; about the same time she complained of nervousness, and the colour of the skin became darker; in August the abdomen became enlarged; in October she suffered from bronchitis and palpitation. A diagnosis of Graves's disease was made. In January, 1905, the following was noted: Abdomen enlarged, walls thick and brawny; no evidences of ascites; lower extremities large, brown stained, and in a condition of hard oedema; no albumen in urine. In February patient suffered from diarrhoea, which continued for two months. On April 9th condition was as follows: Orthopnoea; pulse irregular, small, of low tension; apex beat fourth and fifth spaces, 4 in. to 4½ in. from middle line; mitral systolic bruit conducted toward axilla; jugular veins distended and fill from below; signs of fluid at both bases, and scattered rhonchi; tongue small, red, and raw; abdominal wall hard and irregularly pitted; legs much swollen, very tense; skin of body regularly pigmented; moderate exophthalmos; moderate, non-pulsating, bilateral goitre. In June there was oedema up to the level of axilla, evidences of ascites, the legs were of brick-red colour, brawny, and barely pitted.

On admission to Westminster Hospital the patient presented the following condition: Orthopnoea; moderate exophthalmos; movements of eyeballs very quick; thyroid gland moderately enlarged, soft, and elastic, with slight pulsation and thrill; severe palpitation on excitement; pulse 100 per minute, irregular, rather full, low tension; apex beat in fifth interspace, diffuse and feeble; loud, blowing, mitral systolic bruit propagated to axilla; fainter bruit in pulmonary area; carotids pulsating vigorously; jugular veins distended and refill from below; much thirst, tongue dry; abdomen extremely distended, walls in lower part hard and brawny; signs of free fluid in cavity; liver not palpable, upper border at level of fourth rib; external haemorrhoids; lower limbs enormously enlarged and condition well represented by photograph; calf measures 20 in.; skin obviously much thickened, does not pit, hard and brawny, sulci deep, general surface shows closely aggregated minute prominences, colour bronzed, with reddish tint; skin of body bronzed; no pigmentation of mucous membranes; scattered rhonchi over lungs; patient highly nervous and flushes; fine tremors of upper limbs; urine normal.