on his return home to the Isle of Man. On admission into the Liverpool Infirmary I found him suffering from calculus in the bladder. Upon examining the calculus with the lithotrite it proved to be large, soft, and peculiarly shaped. The last-mentioned circumstance made me resolve to cut instead of to crush.

On June 27th I performed lateral lithotomy, and removed a large phosphatic calculus, which broke under the grasp of the forceps, and disclosed the whalebone mouthpiece of a tobacco pipe, to which a small piece of rotten string was attached. It was noticed that the smell of tobacco in the mouthpiece was quite distinct. As the phosphatic envelope crumbled under the pressure of extraction, it was impossible to estimate correctly its weight. It must have been very large. About an ounce of these fragments was collected. The exact size of the mouthpiece is shown in the drawing. The patient made a good recovery, and left the infirmary on July 28th.

On the fourth day after the operation I showed him the foreign body, and asked whether he could offer any explanation as to how it got into his bladder. He remarked that "he was glad to see it again. He had swallowed it whilst playing with some companions on board ship three years ago. He felt no pain at the time, nor until last December, when the symptoms of stone became manifest." He was frequently spoken to in reference to the improbability of such an explanation, but he would never admit of any other construction being placed upon the word "swallowed" than that usually adopted. In this statement of the patient will be found the first point of interest. Though so highly improbable, was it possible? The only case I know of which would at all substantiate the idea that a foreign body of this nature could find its way into the bladder from the intestines is one recorded by Sir Alfred Roberts, of the Sydney Hospital, who removed by

lithotomy a piece of slate pencil two and a quarter inches long, which it was alleged had been swallowed. Commenting on this case, the author says:—"I have left no stone unturned to elucidate the truth in this very interesting case, and can only state that after much hesitation I have arrived at the conclusion that the pencil was swallowed by mouth and made its way by inflammation and ulceration into the bladder."¹

The second point of interest is in reference to the circumstances generally which determine us in the selection of lithotomy or lithotripsy. These can never be regarded in the light of hard-and-fast rules. In this instance, in the absence of all history, I had every reason to believe that I had to deal with a calculus which, though large, was still, by reason of its softness, quite within the limits of crushing. Had such an operation been attempted, the splitting of the nucleus into fragments would almost certainly have been attended with a fatal result. In this instance the peculiar shape of the calculus rendered me suspicious that I had something unusual to deal with; further, there was a consciousness that there was not that easy access, with the open jaws of the lithotrite, to all parts of the calculus, which is an important feature when crushing has to be undertaken. On these points I mainly relied in deciding in favour of lithotomy, a choice which was more than vindicated by the very unexpected discovery that was subsequently made. In a case previously reported,² where I extracted a pencil-case three and a half inches long from the male bladder with the lithotrite, the patient came under my notice within a few hours of the alleged introduction, by other persons, of this article up his urethra, and before sufficient time had elapsed to permit of the deposition of phosphates upon it.

Liverpool.

¹ Medical Times and Gazette, July 30th, 1859.

² THE LANCET, Aug. 11th, 1877.

CASE OF REMARKABLY SLOW PULSE WITH EPILEPTIFORM SEIZURES; AUTOPSY.

By A. T. GIBBINGS, M.D. LOND.

THE following case, read in connexion with one reported by Mr. St. George Mivart, in THE LANCET of Jan. 3rd, seems to me of sufficient interest to be put on record.

J. B—, aged sixty-six, a tall spare man, with well-marked arcus senilis, had pleurisy as a young man, and presents the marks of cupping for the attack; he says that his pulse has been very frequently intermittent ever since he can remember; has worked rather hard at his office, and has had a good deal of anxiety and worry in domestic matters. There is no family history of any neurosis. He has never had syphilis, gout, or rheumatism, and has been strictly temperate. He consulted me in May, 1882, for weakness and difficulty of breathing, especially on exertion. He complained also of attacks of dyspnoea at night, lasting sometimes for a few minutes, sometimes for an hour or so; also of a troublesome noisy cough. Urine normal; heart and lung sounds normal; pulse intermittent, about 60. He attended more or less regularly for some two months, improving considerably under treatment, though not altogether losing his dyspnoea. From Oct. 21st to Nov. 21st he was confined to the house with an attack of bronchitis, attacks of dyspnoea occurring irregularly, chiefly at night. He returned to business on Nov. 21st, and attendance was discontinued until Dec. 2nd, when I was again sent for. I was struck by the slowness of his pulse, which was 44, and quite regular. He was ordered a mixture of arsenic and strychnia. He went in a cab to business, but had frequent feelings of faintness, for which he was obliged to resort to alcohol or ammonia. On January 3rd, 1883, he was seen in consultation with Sir Andrew Clark, having become steadily worse. He now complained of dyspnoea on the least exertion, and of frequent feelings of faintness. His pulse was now 34, regular and synchronous with the heart beats. Walking up and down the room did not increase the frequency of the heart beats. Sir Andrew Clark recommended that he should be kept in the recumbent position, be fed every three hours, and have six ounces of port or burgundy in the twenty-four hours. He continued to take considerable quantities of nourishing food, which he digested well. Notwithstanding this, the sensations of faintness became very frequent, the pulse steadily declined in frequency, and now a new symptom showed itself. This consisted in frequent epileptiform attacks. The face became deadly pale, the pupils dilated, and the eyes fixed; the pulse ceased to be felt at the wrist. After a few seconds it returned feebly; the face then flushed all over, and clonic convulsive tremors lasted for five or six seconds. The first indication of an attack was the non-recurrence of the usual pulse beat. Almost directly there was a moaning sound, and the face became pale and fixed. This sequence was observed repeatedly, so that with the finger on the pulse I knew that an attack was beginning a second or two before the face changed. He had generally an aura, consisting of a feeling of hot fluid at the back of the neck. On Jan. 20th he was seen in consultation with Dr. Moxon, when his pulse was 22, full and regular, his heart beats corresponding in time. Dr. Moxon could find no evidence of disease of the heart or lungs. The brain was active and clear, and there were no nervous symptoms. Dr. Moxon recommended a trial of nitro-glycerine and atropia. Matters did not mend at all. The epileptiform attacks became very frequent and distressing; rising in bed would generally bring them on, but they also occurred when at absolute rest in the recumbent posture. On the 26th Dr. Moxon again saw the patient, and it was agreed to try bromide of potassium in thirty grain doses, with the hope of checking the fits, which were now most distressing. This he took for two days, but became decidedly worse. The pulse fell to 13, then to 12 on January 28th. The nurse's journal says on the morning of the 28th, "Free from attacks for one hour during the night, all the rest of the night he passed from one fit into another." A considerable amount of albumen was found in the urine. I now decided to discontinue all medicine. He began again slowly to improve, and gradually by February 26th the pulse had reached 31; the albumen had soon cleared away. After this he got up every day and worked two or three hours at

ROYAL COLLEGE OF SURGEONS OF ENGLAND.—The Library of the College will be closed on Friday (this day) till Monday, the 16th inst., for the purposes of the Hunterian Oration and Festival. The Oration will be delivered in the theatre by Prof. John Marshall, F.R.S., LL.D., on Saturday, the 14th inst., at three o'clock precisely.

his account books. He felt altogether better, but there was dyspnoea on exertion. He continued to take nourishment well, the pulse keeping over 30, and varying from that to 33 or 34. On March 6th, while at stool, he fainted, and died in two or three minutes. The temperature, taken occasionally, the last time on February 21st, varied between 96.6° and 97.4°. It never reached 98°. The respirations varied from 18 to 24. It should have been stated that the heart's apex was in the normal position, the impulse was normal, and the heart sounds distinct and without murmur. The infrequency of the beats was owing to prolongation of the diastole.

Autopsy.—Dr. Moxon, Dr. J. W. Hunt, and myself were present. The right pleura contained about one pint of serous fluid. Beyond that the appearances were almost entirely negative. The valves and muscular walls of the heart were healthy, the coronary arteries being pervious. The arteries generally were remarkably free from atheroma. The liver, kidneys, brain, and medulla oblongata were to all appearance normal.

Remarks.—Two questions suggest themselves in connexion with this case—1st, what was the cause of the gradual slowing of the pulse? 2ndly, what was the cause of the epileptiform seizures? The latter were evidently caused by sudden anæmia of the brain. The absence of the recurring pulse beat was always the first in order of the phenomena of the seizures. An intermission of the heart-beat when the pulse is only sixteen or twenty must cause a temporary deficiency of blood in the cerebral vessels. The patient, even in health, had very frequently suffered from intermittent pulse. The cause of the former is more obscure. Post-mortem examination gave nothing but negative indications, while Dr. Moxon's well known skill and experience in this department is a guarantee that no gross change was overlooked. I think there can be little doubt that we must look to the nervous mechanism regulating the heart's action as the factor in its causation. Physiology teaches that stimulation of the vagus will produce this inhibitory or slowing action. Czermak, as is well known, was able at will to stop the beating of his heart, by pressing his vagus against a small osseous tumour in his neck, and thus mechanically stimulating the nerve. In Professor Foster's "Physiology" it is stated that "stimulation of the vagus produces in the ventricle simple lengthening of the diastole without change in the force of the systole." But there is another way in which this retardation may be brought about, and that is by the bringing into abeyance the action of the intrinsic cardiac ganglia, the normal inhibitory action of the vagus being then unopposed. To quote Professor Foster again:—"If a ligature be drawn tightly round the junction of the sinus venosus with the auricles, or if the auricles be separated by an incision carried along the boundary line between the two, a standstill is produced closely resembling a very prolonged vagus inhibition." Probably, then, the cause of the gradual slowing of the pulse must be sought either in an irritative lesion of the vagus or in a degenerative lesion of the cardiac ganglia. Nothing at present is known of changes in these ganglia. The pleuritic effusion no doubt occurred during the last two or three weeks of life, when, finding the patient dressed at the time of my visits, I did not continue to make careful examinations of his chest, as I had done previously at frequent intervals.

Jan. 22nd, 5.20 P.M.: Pulse 22; temperature 97°. 23rd, 1.30 A.M.: Pulse 21. 7 A.M.: Pulse 20. 11.20 A.M.: Pulse 22. 8.30 P.M.: Pulse 20. 24th, 3.50 A.M.: Pulse 19. 7.30 A.M.: Pulse 20. 10.40 P.M.: Pulse 19. 12 P.M.: Pulse 18. 25th, 3 A.M.: Pulse 19. 26th, 9 A.M.: Pulse 23. 5.40 P.M.: Pulse 25. 27th, 3 A.M.: Pulse 22. 10.30 P.M.: Pulse 24. 12.45 P.M.: Pulse 20. 2.15 P.M.: Pulse 19. 5 P.M.: Pulse 17. 28th, 3.15 A.M.: Pulse 17. 10 A.M.: Pulse 13. 11 A.M.: Pulse 12. 12.20 P.M.: Pulse 15. 2.15 P.M.: Pulse 17. 29th, 12.30 A.M.: Pulse 15. 2.15 A.M.: Pulse 17. 7.20 A.M.: Pulse 21. 3 P.M.: Pulse 22. 30th, 12.30 A.M.: Pulse 20. 9.40 A.M.: Pulse 24. 2.20 P.M.: Pulse 24. 9.20 P.M.: Pulse 21. Quite free from fits all day. 31st, 8.50 A.M.: Pulse 20. 2.15 P.M.: Pulse 20. 6.30 P.M.: Pulse 22. Quite free from fits all day.—Feb. 1st, 11 A.M.: Pulse 22. 6.30 P.M.: Pulse 22. One slight fit. 2nd, 9.30 A.M.: Pulse 22. 2 P.M.: Pulse 25. 8.30 P.M.: Pulse 21. Had several slight fits during the evening. 3rd, 9.15 A.M.: Pulse 24. 10.30 P.M.: Pulse 20. Had severe fits during the afternoon; ordered a mixture of solution of arsenic and solution of strychnia three times a day. 4th, 6 A.M.: Pulse 17. 8 A.M.: Pulse 24. 11.30 A.M.:

Pulse 21. 11.30 P.M.: Pulse 18. Had several severe fits until 2.30 A.M., then slept an hour. 5th, 8 A.M.: Pulse 20. 3.30 P.M.: Pulse 22. 8.40 P.M.: Pulse 23. 6th, 9.30 A.M.: Pulse 25. 1.45 P.M.: Pulse 20. 6 P.M.: Pulse 18. Fits continued. 7th, 1.30 A.M.: Pulse 20. 9 A.M.: Pulse 25. 1.40 P.M.: Pulse 20. 8.30 P.M.: Pulse 25. 8th, 3.30 A.M.: Pulse 21. 9 A.M.: Pulse 24. 11 A.M.: Pulse 26. 5.30 P.M.: Pulse 24. 9th, 11 A.M., 3.15 P.M. and 6 P.M.: Pulse 25. 10th, 9.15 A.M.: Pulse 27. 11.15 A.M.: Pulse 24. 1.30 P.M.: Pulse 26. 12 P.M.: Pulse 24. 11th, 9 A.M.: Pulse 27. 5.30 P.M.: Pulse 26. 10.30 P.M.: Pulse 28. 12th, 9 A.M.: Pulse 24. 13th, 1.30 P.M.: Pulse 26. 14th, 8.30 A.M.: Pulse 28. 8.30 P.M.: Pulse 27. 15th, 9.40 A.M.: Pulse 28. 4.40 P.M.: Pulse 28. 16th, 8.40 A.M.: Pulse 26. 17th, 8.30 A.M.: Pulse 29. 18th, 9 A.M.: Pulse 29. 5.30 P.M.: Pulse 29. 19th, 8.30 A.M.: Pulse 27. 3.30 P.M.: Pulse 27; temperature 97.4°. 20th, 5.30 P.M.: Pulse 30. 21st, 8.30 A.M.: Pulse 30. 1.30 P.M.: Pulse 30; temperature 97.4°. 22nd, 9.30 A.M.: Pulse 30. 9 P.M.: Pulse 27. 23rd, 11 A.M.: Pulse 30. 3 P.M.: Pulse 30. 24th, 8.30 A.M.: Pulse 30. 7 P.M.: Pulse 31. 25th, 10 A.M.: Pulse 30. 7 P.M.: Pulse 31. 26th, 8.30 A.M.: Pulse 31. 12 A.M.: Pulse 31.

Richmond-road, Dalston, E.

ARTIFICIAL CIRCULATION AS A MEANS OF RESUSCITATION AFTER APPARENT DEATH.

BY CHAS. EGERTON JENNINGS, F.R.C.S. ENG.,

ASSISTANT-SURGEON TO THE CANCER HOSPITAL, ASSISTANT-SURGEON TO THE NORTH-WEST LONDON HOSPITAL.

CHLOROFORM POISONING.

(Concluded from p. 247.)

Experiment 8.—July 29th, 2 P.M.: One grain of morphia was injected into a terrier weighing 14.38 lb. The administration of chloroform was commenced at 3.34 P.M. When the animal was anæsthetised, a ligature was passed through the tongue. It was noticed that firm traction on this organ did not satisfactorily withdraw the epiglottis, which can be readily seen in the dog. A ligature of fine silk was passed through the epiglottis by means of a small curved needle, so that traction could be made on the epiglottis directly. Respiration stopped at 3.46 P.M. The pulse stopped at 3.51 P.M. The right external jugular vein was now exposed, and the cannula inserted. Depletion showed that the blood was not nearly of so dark a colour as that drawn in the previous experiments; doubtless owing to the access of air to the lungs afforded by traction on the epiglottis. Altogether 20 oz. of saline fluid (temp. 90° F.) with ten minims of liquor ammoniæ were injected. Artificial respiration was continued, and normal respiration returned at 4.2 P.M., the circulation being re-established first; in fact, very shortly after the saline injection was commenced. The blood lost by depletion was partly caught in a porcelain dish, but a considerable quantity escaped beneath the dog, and some also ran through perforated holes on the table to a shelf below. The experiment was therefore carried further than was necessary to restore animation, and afterwards, when the quantity of blood lost, which was in excess of the saline liquid injected, was examined, it was clear that sufficient had not been left within the vessels to maintain life for more than a short period. This experiment was performed by Dr. Boddaert and myself. At 6 P.M. it was reported by one of the laboratory porters that the dog had been "dead" for some time, probably for about fifteen minutes. The body was quite warm, but respiration and circulation had certainly ceased, and the forelegs were stiff. Half a minim of liquor ammoniæ with twenty minims of water were injected directly into the left ventricle through the chest-wall, and the injection was immediately followed by a return of pulsation. The tongue was drawn out of the mouth, and the epiglottis grasped with artery forceps. The laboratory porters were directed to perform artificial respiration whilst I prepared a saline solution. On exposing the left external jugular vein, that vessel was found much collapsed; twelve ounces and three drachms of saline fluid were injected. (Transfusion of blood was strongly indicated here, for the animal was dying from hæmorrhage, and the blood in the body was already very much diluted. Nevertheless it seemed better to inject saline fluid instead, and to watch