made and well-nourished girl. Thick layer of subcutaneous fat. The dura mater presented nothing abnormal. The convolutions appeared flattened, and the entire brain, especially the right half, felt abnormally soft. A firm contracted ante-mortem clot was found in the superior longitudinal sinus, which, at a point two inches and a half above the torcular was greyer, more flexible and adherent, and evidently of older date. The straight sinus contained a firm parti-coloured clot, dark for the most part, but lighter where it projected into the tributary vessels. Near the torcular it was much older and adherent, and extended through it for an inch and a quarter along the right lateral sinus. The remainder of this sinus was empty and of normal aspect. All the other sinuses were normal. All the arteries of the brain presented a healthy appearance. On raising the occipital lobes, the cut end of the thrombus in the straight sinus could be seen projecting from the great transverse fissure. The right centrum ovale majus was studded with minute multiple hæmorrhages. The right corpus striatum and optic thalamus were in a state of red softening. There had been capillary oozing from the choroid plexus of the right lateral ventricle, which had coagulated *in situ*. The ventricles were not dilated, and contained no fluid. The left centrum ovale contained a very few minute hæmorrhages. On raising the fornix, the veins of Galen were seen to be much distended and rod-like. They were both completely filled by firm half-discoloured ante-mortem clot. There was no marked injection of the meninges, nor was there any excess of subarachnoid fluid. No local cause for the thrombosis was discovered anywhere, although the most minute search was instituted. With the exception of a slight superficial erosion of the os uteri, all the other organs of the body presented normal naked-eye appearances.

Remarks by Dr. DOUGLAS POWELL.—The above case is perhaps best left without comment to accumulate with experience of other cases, since no satisfactory explanation of it can at present be offered. The patient was anæmic, but by no means extremely so. It is true that venous thrombosis is not uncommon in anæmia, and the naturally retarded circulation through the cerebral sinuses may be held to be favourable to the occurrence of thrombosis in them; but, practically, such thrombosis in simple anæmia is of most rare occurrence.

HIGH-TENSION PULSE WITH PERIPHERAL REFLUX IN ALBUMINURIA.

Dr. Douglas Powell, in the course of some clinical remarks upon a typical case of contracted kidney, with characteristic cardio-vascular changes, resulting in cerebral hæmorrhage, drew attention to a point with regard to the high-tension pulse of this form of albuminuria which the case before him illustrated very strikingly. On compressing the radial artery with one finger with sufficient force to close the vessel—and it required hard pressure to do this,—and applying a second finger to the vessel an inch further in its course, it was felt to pulsate with great distinctness and rhythmically with the central portion of the vessel. Now, keeping up the pressure with the first finger, and applying the second with just sufficient force to check the pulse wave under it, it could be distinctly felt that the blood wave impinged against the side of the finger directed towards the hand of the patient, whilst of course the pulse wave impinged upon the trunk side of the finger first applied. It was clear, therefore, that in this case the pulse was stopped by the first finger, and a peripheral reflux pulse was felt by the second finger of the observer. This phenomenon threw some light upon the "incompressible" high-tension pulse of albuminuria and in some other conditions, suggesting that the vessels were applied to the blood they contained with such a degree of tension that they became almost like rigid tubes; and when the overflow through an artery of a limb was stopped at a given point, the peripheral end of the vessel remained full and received a back impulse through the capillary system or anastomosis short of the capillaries with each systole of the heart.

DEWSBURY AND DISTRICT GENERAL INFIRMARY

SUBACUTE CATARRHAL NEPHRITIS.

THE following notes (for which we are indebted to Mr. Milne, house-surgeon) of a case of subacute catarrhal nephritis in a young man, followed by uræmic poisoning and convulsions, may be of interest as illustrating the good effects to be obtained by venesection in suitable cases.

J. B——, aged twenty-one, collier, presented himself at the out-patient department of the infirmary on July 5th, 1888. He had œdema of the face, legs, and feet, and on examination his urine was found to contain a large amount of albumen. He ascribed his illness to a chill caught through standing in water while at his work in the coal-pit some days before. Subsequently he suffered from pains in the loins, and noticed his urine to be dark and his face swollen. On being asked to become an in-patient, he refused, and nothing was seen of him till about noon on July 17th, when he was driven up in a cab for admission. The œdema had got very much worse, and he had had a severe attack of convulsions that morning.

On admission the man was in a semi-comatose condition, and scarcely recognisable from the enormous swelling of his face, body, and limbs. His breathing was stertorous, and he could not be roused to answer questions, and with difficulty was made to swallow. Calomel (twelve grains) and compound jalap powder (a drachm) were given, and a hot wet pack ordered. The latter made the skin act freely. Violent epileptiform attacks continued at intervals of about two hours; during them, notwithstanding the efforts of the nurse, his tongue got severely bitten. The seventh and last fit he had, and the first seen by Mr. Milne, occurred about 11.30 P.M., while he was standing at his bedside. It commenced with rotatory movements of the left arm, followed by clonic spasms over the whole body; his face became dark and congested, the muscles twitched violently, and the tongue was protruded. On the spasms ceasing he appeared to be dead; respiration had ceased, and only an occasional pulse beat was to be felt. A few slaps on the chest re-started respiration, and Mr. Milne injected twenty minims of ether. Thinking another fit might prove fatal, and as the pulse was now of fair strength and volume, Mr. Milne determined to try venesection. The œdema of the arms made opening a vein a little difficult, and failure was experienced in the left arm, but the median basilic of the right was opened. Twelve ounces and a half of blood were withdrawn, and immediate improvement followed. His breathing, which before had been loud and snoring, became much easier and quieter; in twenty minutes he was with some effort got to answer questions; in three hours he became completely conscious. The convulsions were not repeated. Finding the purgatives given had not acted, two minims of croton oil were given after the venesection. The bowels acted very copiously during the night. A hot vapour bath was given in the morning, and a diuretic mixture with five grains of citrate of caffeine ordered. On the 19th he passed 58 oz. of urine, and for subsequent days the quantities were 109 oz., 110 oz., 101 oz., 89 oz., and 64 oz. Afterwards about the normal amount was passed per diem. The hot vapour baths were continued for a few mornings, and the œdema rapidly disappeared. He left the hospital on Aug. 25th in good health, and with no albumen in his urine, although it was collected at various times (after being up for some time, and after meals), and tested repeatedly.

Remarks by Mr. MILNE.—I do not think there can be much error in this man's case in ascribing the immediate improvement after venesection to the good effect of that operation. To me at the time it seemed to be the means of saving the patient's life. In conclusion, I would point out that in this case the patient was a young man of good previous health, and his illness of short duration, so that vigorous measures in the way of bleeding, free purgation, and hot vapour baths could be adopted without danger.

ARARAT HOSPITAL, ARARAT, VICTORIA.

CASE OF SUPPURATING HYDATID OF THE LUNG, TREATED BY ASPIRATION AND CLEANSING WITH CARBOLIC LOTION.

(Under the care of Mr. GEO. PALMER.)

THIS case resembles in many points the one published by Mr. Joshua in our issue of Oct. 20th.

T. L——, aged twenty-five, was admitted on Oct. 26th, 1885, to this hospital, in a weak, hectic, and emaciated condition, suffering from hydatid of the right lung, which had suppurated and opened into the bronchi. Through the latter small cysts and fetid pus were being freely discharged.

As the natural efforts at expulsion of the contents of the

cyst were daily becoming weaker and less effectual, an attempt was made to supplement them by aspiration. Careful examination and exploration in various situations with the hypodermic needle showed that healthy compressed lung intervened between the chest wall and the suppurating cavity, which occupied the centre of the lung; aspiration was therefore resorted to below the inferior angle of the scapula. After the needle had penetrated to nearly its whole length, most offensive pus flowed freely into the syringe. More than half a pint of fluid was drawn off and replaced, without removing the needle, by injecting tepid carbolic lotion (1 in 80). This was withdrawn after a few minutes. The man felt faint but much relieved in his breathing after the operation. Hæmoptysis followed two nights afterwards, but did not recur. The aspiration and injections (in the same situation) were repeated twice at intervals of a week, the amount of pus withdrawn and lotion injected being less on each occasion. The patient gradually but markedly improved after each aspiration, and was discharged on Dec. 29th, 1885. The expectoration gradually lost its purulent character, and for the last two years he has been able to follow his occupation, that of a jockey, and has had good health.

Medical Societies.

PATHOLOGICAL SOCIETY OF LONDON.

Debate on the Morbid Anatomy and Pathology of Chronic Alcoholism.

AN ordinary meeting of this Society took place on Tuesday, December 4th, Sir James Paget, F.R.S., President, in the chair.

The debate was opened by an able address by Dr. J. F. PAYNE. He commenced his remarks by stating that he did not intend to discuss the action of drinks on the human body generally, nor all the diseases to which habits of excess could give rise, nor, still less, the moral or economical consequences of such habits, but solely the material changes which the use of alcohol in excess had been actually shown to produce in various tissues and parts of the body. He then proceeded to a historical study of the subject, taking in the first period the history of ancient medicine up to the sixteenth century. He could find here but scanty notices of the effects of inebriety, and no recognition at all of the anatomical changes due to it. The second period, including the sixteenth and seventeenth centuries, saw the rise of morbid anatomy, which now began a separate existence as a science, at first under the protection of her elder sister—normal anatomy—and gradually afterwards assuming a more independent position. Anatomical changes were now for the first time referred to inebriety. In Harvey's lectures, though nothing could be found referring directly to alcoholism, diseased livers evidently produced by this cause were described. One case he saw was "russet, hard, contracted, absque, sanguine"; apparently it was a small cirrhotic liver. Another was "russetish, ingentum et durum, plane scirrus tumour, absque fere sanguine, aspera superficie": a large hard liver, evidently like a scirrhous tumour, almost bloodless, and with a rough surface, which could hardly have been anything else than cirrhosis. Harvey wrote in 1616, and not long after this time some notices of alcoholic diseases began to appear, but the only lesion referred to this cause by writers of the seventeenth century was cirrhosis of the liver and its consequent ascites. The earliest case of this kind which he could find was of date 1626, though published many years later, in the great storehouse of such observations, Bonet's "Sepulcretum") (Geneva, 1679), where it was quoted from Gregorius Horstius. Dr. Payne quoted another case from the same work, and in both these and many others there related strong wine was clearly recognised as the source of evil. English medical literature of this period yields few valuable observations. There was one by Walter Harris, the correspondent and friend of Sydenham, the author of a book on the diseases of children and of "Pharmacologia Anti-empirica" (London, 1683); the case was related in the latter work. John Browne, surgeon to St. Thomas's Hospital, published an account of a case in the "Philosophical Transactions," vol. xv., 1685, entitled "A Remarkable Account of a Liver, appearing Glandulous to the Eye." It was accompanied by a figure, "accurately taken down by Mr. Faithorn," an eminent artist and engraver of the day. Dr. Payne had copied this picture and exhibited it to the Society. It appeared from the account that Mr. Browne did not recognise the dependence of this lesion upon alcoholic drinks. The drawing was an excellent one of atrophic cirrhosis, and appeared to be the first published illustration of this lesion. The third period dealt with by Dr. Payne was from 1700-1850, the earlier part of this period being marked by the introduction of distilled spirits as a beverage, and then followed a rapid increase of alcoholic diseases. In 1724 the College of Physicians made a public representation as to the evils of spirit drinking, and the Rev. Stephen Hale, the physiologist, exerted himself to check the practice. J. C. Lettsom was the first to notice some of the symptoms of alcoholic paralysis, and James Jackson of Boston, U.S.A., in 1828, gave a good account of the same affection under the name of arthrodynia. The classical work of Magnus Huss on "Alcoholismus Chronicus," contained careful descriptions of the morbid changes met with in all parts of the body in drunkards, and he regarded the nervous disturbances, being unaccompanied by any change in structure, as symptoms of a certain kind of poisoning. The fourth period, from 1850 to the present time, embraced the era of the rise of pathological histology, the most conspicuous advance being the demonstration of minute changes in various parts of the nervous system, and the uniformity of the action of alcohol throughout the whole body had become clearly manifest. We were now able to clearly recognise the toxic action of alcohol, and to compare it with the action of other poisons. The third section of the subject was discussed by Dr. Payne under the head of the "general pathology of alcoholism." He considered at some length the question whether alcohol was a poison, taking as the pathological definition of a poison "a substance capable of injuring the body, either by causing damage to the tissues or by producing functional disturbance." He used the word "poison" not as a term of unqualified condemnation, but as meaning something capable of producing injury, though not necessarily doing so; it would be as absurd to condemn alcohol as to condemn common table salt, because a large dose of either of them might be fatal. He defined a functional poison as one disturbing the mode of action of the tissue elements without permanently altering their composition; a tissue poison as one damaging the structure of the tissue elements themselves. Tissue poisons acted on all or most tissues of the body, which they reached in proportion to the degree of concentration in which they might be present and to the susceptibility of the different parts; and they all had, within certain limits, the same action, or at least there were certain modes of action common to all. Studying alcohol in this light, he found that its action on the human body was threefold:—1. It checked oxidation, and thus favoured the accumulation of fat, producing fatty infiltration or steatosis in parts naturally disposed to it. This mode of action resembled that of phosphorus. 2. It acted as a functional stimulus, or, in a larger dose, as a functional poison on the nervous system, especially on the brain. This action was not here regarded except in so far that prolonged functional derangement might give rise to structural change. 3. It acted as a tissue poison, destroying the vitality of some tissue elements, and setting up inflammation in others. Generally speaking, the parenchymatous elements, nervous or epithelial, suffered degenerative or necrotic changes, while the connective-tissue elements or stroma proliferated and underwent chronic inflammation. These two classes of changes were concurrent effects of the same poison, not one dependent on the other. This action of alcohol was comparable to that of mineral poisons. In the fourth and last section Dr. Payne dealt with the morbid changes produced by alcohol in various organs. He confined his remarks especially to the liver and nervous system, taking the morbid changes in these parts as types of the effects produced in the organs generally. He referred, in passing, to the stomach, the mucous membrane of which showed degenerative and necrotic changes, whilst the walls sometimes became sclerosed. With regard to the liver, he discussed first fatty infiltration, in which the essential structure of the organ was preserved. This change was an example of steatosis, and was produced especially by the dilute forms of alcohol and in those who were well fed. He questioned if it ever passed into cirrhosis, and thought the accumulation of fat was, so far as it went, evidence of the destruction of some alcohol. In reference to the pathology of cirrhosis,