The question of the diagnosis is obviously difficult.

(1) Superficially one would have little hesitation in classing the patient under the heading of katatonia. But there are many points in the history that seem fairly definitely to contra-indicate dementia præcox.

(2) Is the patient living in a prolonged state of epileptic automatism? The absolute amnesia of the waking periods prompts the thought, but there is little else in the evidence to justify the assumption of epilepsy.

(3) From a comprehensive survey of the history it appears that long ago, in March, 1917, the patient encountered some disturbing, explosive force that induced a disintegration of the personality, followed by a dissociation of psychic activity—a dissociation in series rather than in parallel.

Whatever may be the true diagnosis, the case appears to be of sufficient interest to warrant setting forth in some detail, and I wish to express my indebtedness to Dr. Turnbull for permission to quote his article and to publish the further notes of this case.

(1) Turnbull, R. C., "A Case of Katatonia," *Journal of Neurology and Psychopathology.*

A Case of Cerebral Hemiatrophy (?) or Unilateral Hydrocephalus (?).

By Fred Wilson, M.B., Ch.B.Aberd., Assistant Medical Superintendent, Central Mental Hospital, Tanjong Rambutan, Federated Malay States.

A. M—, a Chinese girl, æt. 22, was admitted to the Central Mental Hospital on September 14, 1923. She was transferred from another hospital, in which she had been for one year. She was there diagnosed as an imbecile, and was reported as having frequent epileptic seizures and impulsive outbursts.

On admission she was a slightly-built girl, who looked about 14 years old. She was not fully developed sexually, and never menstruated while in hospital. She had spastic paralysis of the whole of the right side of the body. Right facial paralysis and right homonymous hemianopia were noted. The right upper extremity showed advanced wasting and contractures, and the right lower extremity showed the same to a less degree. She was able to walk with difficulty, dragging the right foot along the ground. She was unable to speak, but could make a few inarticulate sounds. She understood simple questions and commands, could feed herself and was clean in habits.

Soon after admission she had an attack of dysentery, and later she developed pulmonary tuberculosis. She became progressively weaker; the contractures and wasting of the lower extremity became more marked; she was wet and dirty in habits, and mental deterioration became more profound. She had no seizures, nor was she ever impulsive or troublesome while in hospital.

She died on November 13, 1924.

Post-mortem examination.—The skull was symmetrical and showed no abnormality. Dura was normal and non-adherent. There was some slight excess of fluid beneath the dura. Cerebellum, pons and medulla showed no gross lesion, nor did the right cerebral hemisphere, of which the membranes were natural; the right lateral ventricle was of normal size, and the brain substance healthy.

The left hemisphere, however, presented a very different picture. The membranes were thickened and opaque, and were almost devoid of blood-vessels. The lateral ventricle was enormously distended with fluid. The ependyma was quite smooth. The brain substance was reduced to ¼ in. in thickness, and it was impossible to make out any distinction between grey and white matter. The substance presented a white, jelly-like appearance, and to the naked eye appeared completely atrophied. The membranes could be stripped with difficulty, and it was just possible to see the remains of the convolutions as little dimples on the surface. The weight of the left hemisphere (with membranes) was 3¾ oz., while the right weighed 17 oz.

The cause of death was pulmonary tuberculosis, both lungs being full of cavities.

Unfortunately no previous history of this case could be obtained, and the mental condition of the patient precluded the possibility of a satisfactory examination of the nervous system.

The age on admission to an institution (21 years), the absence of any malformation of the skull, and the complete cessation of what were evidently very severe epileptiform seizures, all suggested the possibility that this was a progressive condition, and that the case was more one of dementia than of amentia.

In any case, it must be rare to find such a hydrocephalic condition confined to one lateral ventricle.

I am indebted to the Medical Superintendent, Dr. W. F. Samuels, for permission to publish this case.

The Neutral Sulphur Excretion in Dementia Præcox following Sodium Thiosulphate Ingestion.

By S. A. MANN, B.Sc.Lond., F.I.C. (From the Pathological Laboratory of the London County Mental Hospitals, Maudsley Hospital.)

SOME years ago in conjunction with the late Waldemar Koch the author made chemical examinations of the brain in a series of cases of mental disorder (1); in dementia præcox changes in the sulphur partition were found which seemed to indicate that in this disease there was a deficiency for oxidation processes. The following note represents an attempt that has been made to investigate further this point by the administration of sodium thiosulphate to a series of dementia præcox cases and subsequent examination of the urinary sulphur partition. Sodium thiosulphate, $Na_2S_2O_3$, taken by mouth in the main is excreted in the urine as fully oxidized sulphate (2), unless the dosage is excessive, when a diarrhœa is induced. Trachtenberg (2) states that upwards of 18 grm. will cause diarrhœa, but the effect of dosage may be variable. Nyiri (3, 4, 5 and 6), investigating the fate of sodium thiosulphate in the organism, gives the following results. For 1 grm. sodium thiosulphate introduced intravenously from 30–40 *per cent.* reaches the urine