to Ferrier's centre for sight. Now had I not operated on it I should have regarded it as a case in which the visual centre had been destroyed, but the complete return of that sense after operation indicated that it was really only in abeyance. In other words, the organ of sight was temporarily involved, owing to the disturbance set up in the mechanism of the brain by the presence of such an irritant as we have seen that this hydatid is. This return of a function that seemed destroyed as soon as the source of irritation was removed, is only another instance of how careful we should be in drawing conclusions from experimental inquiries, a caution fully borne out by Goltz's numerous physiological experiments.

GEORGE T. BRATSON, B.A (Cantab.), M.D. (Edin.).

Tripier on Anæsthesia from Cortical Lesions. (*Revue mensuelle*, January and February 1880.)—In this memoir M. Tripier reviews the various explanations which have been given of the paretic or paralytic symptoms resulting from cortical lesions, and gives the details of some recent experiments he has made (in four dogs and one monkey), as well as several clinical cases in support of the thesis, that lesions strictly cortical cause not only motor symptoms but also defect of sensibility, principally, but not exclusively, tactile.

In his previous experiments, in which he had shown that dogs never really recover entirely from cortical lesions, and that the defect in mobility could always be reproduced by morphia, he had failed to detect any deficiency in sensation; but in the experiments here recorded, made with the special object of testing the sensibility, he finds that after lesion of the sigmoid gyrus, there is not merely paresis of the limbs, but also deficient reaction to cutaneous stimuli—pricking with a pin—on the affected side. This is most marked in the face and paw, but is more or less general, and may be observed immediately after the lesion, or even up to nine months afterwards.

He concludes from this that the sensibility is blunted or deficient.

He, however, opposes the views of Schiff, &c., that the defect in mobility is dependent on the deficient sensibility, and gives experimental evidence of the fact that division of the sensory nerves of the limb does not cause such motor disorders as result from cortical lesions. The motor symptoms are therefore the direct effect of the cortical lesions, but these are also combined with defect in sensibility, though the two are independent of each other.

The clinical cases adduced are cases of hemiplegia combined with hemianæsthesia, of greater or less degree, in which no other lesion was discernible post-mortem than cortical lesion, the ganglia and internal capsule being to all appearance intact. He is of opinion that there is no special sensory region in the cortex, but that the lesions which mainly cause hemiplegia (fronto-parietal), also cause hemianæsthesia. The latter, however, is more transient.

[M. Tripier's argumentation is not very conclusive. His clinical facts prove nothing. He might have cited cases with similar symptoms without discernible lesion in the cortex or elsewhere. It has been indisputably proved, in opposition to M. Tripier's views, that we may have most complete motor paralysis from cortical lesions, with full retention of sensibility both to tactile and painful stimuli.

Assuming the complete accuracy of M. Tripier's experimental data, they prove nothing beyond deficient reaction to stimuli. That this deficiency is on the sensory side is an inference which is not admissible until a clear discrimination can be made between psycho-motor and mere reflex reaction. Such a standard M. Tripier has not supplied, and to do so is no easy matter. An animal may not react to a cutaneous stimulus, not because it does not feel it, but because its volitional power is impaired. Even as M. Tripier's own data show, the reaction to a painful stimulus, such as would normally excite reflex movements, is as distinct on the one side as the other.]

Sommer on Diseases of the Cornu Ammonis (*Archiv für Psychiatrie*. Bd. x. Heft 3).—Sommer tabulates 90 cases, collected from various sources, in which the hippocampus was affected along with other cerebral regions, from which it is clear that epilepsy is frequently associated with degeneration in this region and its neighbourhood. He estimates the proportion at 30 per cent.

The lesions are divided into two principal forms: 1, the classic form, described first by Meynert, in which there is atrophy and sclerosis in one or both cornua; and 2, lesions of various kinds, such as softening, softening with redness, redness and punctiform hæmorrhages, pressure, &c. The proportions in which the various affections occur are stated in a tabular form. A detailed description is given of the microscopical characters of the degeneration which occurs.

Affections of the hippocampus in epileptics are associated in a

large number of cases with excentric sensory disturbances, indicating that the sensory centre is here, or in close relation with it.

Of the 90 cases analysed, 38 had sensory disturbances in the form of hyperæsthesia, paræsthesia, and anæsthesia.

Of the 52 negative cases, the records are either incomplete, or the conditions were such as to preclude any observations on this point.

Of the 38 positive cases, 27 relate to affections of common sensation; anæsthesia 7 times, hyperæsthesia 3 times, paræsthesia (formication, &c.), and actual hallucinations 15 times, and lastly 2 in which, owing to the mental condition, it could only be inferred. Frequently the common sensory disturbances were associated with illusions of hearing and sight, less frequently of smell and taste. Occasionally, however, these latter were alone affected without implication of common sensation.

Our author regards it as beyond doubt that there is a direct relation between degeneration of the hippocampus and the disturbance of common sensibility.

He next proceeds to the consideration of the experimental, anatomical, and other clinical evidence in favour of the view that the hippocampus is the centre of common sensation.

The first is furnished by Ferrier's experiments, which are briefly analysed.

The anatomical evidence is as yet incomplete. He refers to the researches of Meynert and others, in which it appears that there is a direct relation between this region and the sensory tracts of the spinal cord.

The clinical evidence is also as yet very deficient. There are only two cases on record, one of these even doubtful, which indicate the sensory function of the hippocampus.

The first of these, related by Chvostek (*Wien. med. Wochensch.* 1871, No. 37-39), was a case of typhus with delirium, headache, and other cerebral symptoms. This was in 1853. Diminution of the sensibility of the left arm and leg, with formication and slight paresis remained, and at a later period paralysis agitans showed itself on the left side. Death occurred from phthisis in 1871. Section showed atrophy and sclerosis of the right hippocampus and portion of the neighbouring cortex, without other cerebral lesion.

The second case related by Claus (*All. Zeitsch. für Psychiat.* 1878, p. 335), was a case of cerebro-spinal sclerosis, in which there were paræsthesia and impaired sensibility in the upper and lower extremity and trunk. On section, along with other scleroses, there was marked sclerosis in both hippocampi.

D. FERRIER.