

*The Shellac Reaction on the Cerebro-spinal Fluid* [*La reazione della gomma lacca nel liquido cefalo-rachidiano*]. (*Il Cervello*, vol. xl, May, 1932.) de Marco, A.

The author used the shellac reaction as first described by Urechia and Danetz in 1923. He found the curve in general paralysis to be 321,000,000. The results were similar to those obtained by the gum-mastic reaction. A concentration of 8% of salt solution was used.

G. W. T. H. FLEMING.

*A Contribution to the Citochol Reaction of Sachs-Witebsky* [*Contributo alla reazione (Citochol) di Sachs-Witebsky*]. (*Il Cervello*, vol. xl, July, 1932.) Zara, E.

The author carried out the citochol reaction of Sachs-Witebsky on 250 sera and 64 spinal fluids, and controlled his results with the Wassermann in the sera, and the Wassermann together with other reactions in the spinal fluid.

He concludes that this reaction may well be used as a complement to the Wassermann reaction in the sero-diagnosis of syphilis.

G. W. T. H. FLEMING.

*The Cerebro-spinal Fluid in 230 Cases of General Paralysis after Malarial Treatment.* (*Journ. of Neur. and Psychopath.*, vol. xiii, Jan., 1933.) Reid, B.

The author finds that the spinal fluid improves after treatment with malaria, and may become normal. This usually takes several years to occur. The improvement in the fluid occurs in patients who have improved mentally and also in those who do not improve.

G. W. T. H. FLEMING.

*A Study of the Cerebro-spinal Fluid in General Paralytics Treated with Malaria* [*Estudio del Liquido Cefalo-Raquideo en los Paraliticos Generales Malarizados*]. (*University of Buenos Aires*, 1932.) Kafer, J. P.

This study is based on the investigation of 17 cases. The author distinguishes four periods, as a purely schematic arrangement: (1) A malarial period characterized by attenuation of the fluid pressure; (2) a primary post-malarial period, extending over the year after the cessation of fever, and characterized by attenuation of the pleocytosis; (3) a secondary post-malarial period, extending over the second year from the cessation of the fever, during which the attenuation of the pleocytosis is continued; (4) a later period, during which, in some cases, the reactions become normal.

M. HAMBLIN SMITH.

*Oxygen Consumption ("Basal Metabolic Rate") in Schizophrenia.* (*Arch. of Neur. and Psychiat.*, vol. xxviii, Dec., 1932.) Hoskins, R. G.

The author found the rate of oxygen consumption in 214 male schizophrenics of average age 30.2 years to be 88.3% of the standard normal. The rates for each sub-group were: catatonic 87.9%, hebephrenic 89.4%, paranoid 87.9%, simple 95.0% and indeterminate cases 88.8%.

The average lowest reading was 81.1%. The author considers that this more nearly represents the true basal rate than does the average mean rate (88.3).

The data reported offer no evidence as to whether the downward displacement of the rate is causative of, consequential to, concomitant with or integral in the psychosis proper.

The author found a polymodal distribution in all but the paranoid group, and thinks that this casts some doubt on the nosological homogeneity of these other groups.

G. W. T. H. FLEMING.

*pH of Blood of Psychotics Measured by the Glass Electrode.* (*Biochem. Journ.*, vol. xxvi, No. 5, 1932.) Hurst, R. H.

The author, using the glass electrode outfit described by Kerridge, found no significant difference of schizophrenics from the normal, either in the resting condition or in the early stages after alkali ingestion. As the result of exercise the

schizophrenic has a blood-pH lower than that of the normal for a given lactic acid content. The author thinks that this is due to diminished excitability of the respiratory centre.

G. W. T. H. FLEMING.

*Blood-urea Changes in Abnormal Mental States after the Administration of Amino-acid.* (*Biochem. Journ.*, vol. xxvi, No. 3, 1932.) Lockwood, M. R., and Davies, D. R.

The blood-amino-acid changes after the administration of gelatin or glycine to 17 psychotics of varying types showed no material difference from the normal. The characteristic blood-urea curve of agitated and simple retarded melancholia is typified by rapid and uniform changes, the total extent of the changes showing a marked increase over the normal. There is a rapid rise to a high level, followed by a rapid fall within the 5-hour period to the fasting level.

The authors describe a second curve in which, while the early changes are fluctuating, the maximum reading is obtained later than in the normal, the actual changes again being larger than the normal. This is found to occur during recovery from melancholia and in apathetic states generally. The rise in urea level is slow, reaching its peak at the fifth hour of the test period. The authors found marked variations between individuals in the dementia præcox group as a whole, and so were unable to draw any conclusions from this group. They think that the changes noted may be due to some disturbance of factors which control the rate of entry into, and disappearance from, the blood of urea, since Addis, Barnett and Shevkey have shown that adrenaline and pituitrin regulate urea excretion.

G. W. T. H. FLEMING.

*Oxidations by the Brain.* (*Biochem. Journ.*, vol. xxvi, No. 3, 1932.) Quastel, J. H., and Wheatley, A. H. M.

Human grey matter shows least activity towards the oxidation of succinate. The rate of oxidation of added substrates to the brain varies inversely as the size of the animal. Glucose, sodium lactate and sodium pyruvate at equivalent concentrations are oxidized at approximately the same rate by brain-tissue. Glucose is not appreciably oxidized by brain in Ringer's solution. The addition of phosphate buffer to Ringer's solution restores the rate of oxidation due to glucose. Lactate is probably completely oxidized by brain. The addition of serum increases the oxygen uptake by the brain. Iodo-acetic acid (M/4,000) inhibits the oxidation by brain of glucose, fructose and mannose, but only partially inhibits the oxidation of lactate, pyruvate and glutamate, and has no effect on succinate oxidation. In a mixture of lactate and succinate, the oxidation of the latter is inhibited by an amount exactly equivalent to the oxidation for which the lactate alone is responsible. This action is probably due to the competition of lactate with succinate for the oxidized form of a carrier, possibly cytochrome, which Holmes has shown to be abundant in the grey matter.

G. W. T. H. FLEMING.

*Dehydrogenations by Brain Tissue; The Effects of Narcotics.* (*Biochem. Journ.*, vol. xxvi, No. 5, 1932.) Davies, D. R., and Quastel, J. H.

Quastel and Wheatley had shown that the exposure of fresh brain-tissue to narcotics at low concentrations results in specific inhibitory effects on the oxidations of the brain. The narcotics exert a profound inhibitory action on the oxidation of substances important in carbohydrate metabolism, *i.e.*, glucose, lactic and pyruvic acids. The narcotics at the same concentrations do not interfere with the oxidation by the brain of sodium succinate or p-phenylenediamine. Narcotics of the same chemical type with the greater hypnotic activity have the greater inhibitory action on the oxidation by the brain of glucose or lactic acid. In the present research the authors found that the dehydrogenating power of grey matter is three to four times as great as that of white matter. Glucose, fructose, galactose and mannitol are activated as hydrogen donors by fresh brain-tissue. The activities of glucose and fructose are less than those of sodium succinate, sodium lactate and sodium