anxiety, varying in nature with the individual case. The author adds that he believes such anxiety symptoms, though of psychogenic origin, arise on a foundation of metabolic and endocrine dysfunction, which needs further study, and that the emotional disturbance in its turn adds to such dysfunction; hence treatment must include such drugs, diet or physico-therapy as may be appropriate. By such methods it may be possible to alter the morbid reflex and remove the underlying disposition to the formation of others of a similar kind.

In discussing the investigation of the first stages of the habit-formation, he suggests that psycho-analysis is unnecessary and useless if these are intellectually known to consciousness. (This is, of course, incorrect, since mere intellectual knowledge does not remove the compulsion. His methods may often be useful, but cases which do not yield to them may yet resort hopefully to analytic treatment). M. R. BARKAS.

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7. Pathology.

Blood in Personality Disorders. (*Arch. of Neur. and Psychiat., June,* 1925.) *Henry, G. W., and Mangam, E.*

These authors examined the blood of 200 consecutive admissions for carbon dioxide combining power, and found it unaffected unless there is some underlying physical disease. Determinations of the urea nitrogen of 143 cases gave negative results. Further studies of the non-protein nitrogen, uric acid, dextrose and chloride content of the blood gave negative results. Glucose tolerance tests showed more or less characteristic changes in the glucose content of the blood of patients in either phase of affective psychoses and in the acute stages of dementia præcox. These changes indicate a definite retardation of functions of the vegetative nervous system in manic-depressive depression and in the acute stages of dementia præcox, and an acceleration of these same functions in manic-depressive excitement. G. W. T. H. FLEMING.

On Dysoxidative Carbonuria [*Über dysoxydative Karbonurie*]. (*Zeits. für Arzt. Fortb., August,* 1925.) *Bickel, A.*

Attention is drawn here to a series of conditions in which the proportion of carbon in the urine is increased as a result of defective bodily oxidation processes. The carbon in the urine appears in nitrogen-containing substances, such as urea, amino-acids, and creatinin, and in nitrogen-free bodies, such as carbonates, oxalates, and a dextrin-like carbohydrate; in diabetes also as dextrose.

In normal individuals on an ordinary mixed diet and with moderate exercise the 24-hour excretion is roughly in the following proportions:

	Through kidneys.	Through lungs.	Through skin.	Through intestine.
C	10 grm.	270 grm.	2·3 grm.	3·0 grm.
N	15·6 ,,	0 ,,	traces	0·9 ,,

Thus the ratio of C/N in the urine is approximately 0·6–0·7. An increase of fat in the diet does not alter this much, but a diet consisting chiefly of carbohydrate raises the quotient to 0·9, since an overloading with carbohydrate leads to incomplete oxidation to CO_2.

Under pathological conditions there may be an increase of the carbon-containing nitrogen bodies or other carbon compounds. Disorders of the carbohydrate metabolism are important here. In diabetic urine, even after the removal of glucose and acetone bodies, the C/N quotient is found as high as 2·0, probably from the presence of glycuronic and oxalic acid and their salts. The author has also found a raised C/N quotient in conditions of avitaminosis, which he regards as pre-diabetic, and where there is also a lowered utilization of oxygen in the lungs, though reducing carbon derivatives do not appear in the urine. He suggests that these conditions of *non-glycosuric dysoxydative carbonuria* deserve further clinical and pathological investigation. Vitamin deficiency is only one of them; various endocrine and metabolic disorders, wasting and obesity, polyneuritis, tendency to boils, etc., may come under this head. He adds that the restoration of a normal C/N ratio by the injection of insulin has been found an aid to diagnosis of such abnormalities.

Details of his method of determining the ratio are not given, nor are there any references to published work. M. R. BARKAS.

The Carbophosphides and their Reaction on Blood-serum [*Della Carbofosfide e della sua azione sul siero di sangue*]. (*Rendic. della R. Accad. Naz. dei Lincei, Roma*, 1923.) *Three notes by Cuneo, Gerolamo.*

(1) The first paper describes the method worked out by the author for preparing the substance carbophosphide, in which the N of urea is replaced by P. He passes gaseous hydrogen phosphide through a toluene solution of carbon oxychloride, when a yellow insoluble substance forms (CO_2PH_4). It cannot be completely purified, being wholly insoluble, and is probably a polymeric form of the carbophosphide, though the molecular weight could not be determined. Its analysis corresponds well, allowing for some impurity, with the above formula. Its action on blood-serum is of interest; a gradual reaction occurs when about 0·4 *per cent.* is added to centrifuged and filtered serum, and kept at a constant temperature of between 38–40° C. The serum does not decompose even after a month, but within two days there begins to form an opaque flocculent white precipitate, and the alkaline reaction of the serum becomes faintly acid. At the end of 12 days all the albuminous substances are precipitated and the precipitate of carbophosphide and phosphoalbumen is treated with dilute potash, which dissolves the latter, so that it can be re-precipitated by acetic acid and thus purified. This albuminoid contains phosphorus in organic combination and has a smell like ozone. A solution in sodium carbonate injected subcutaneously or intraperitoneally in dogs produced a rise of