neutropenia. He takes, however, as his normal limits for lymphocytes 18–22%—a very low figure. The eosinophils were increased in about 25% of cases, and decreased in about 20%. The basophils were normal in more than half the cases, decreased in about 36%. Myelocytes were scarce. The Arneth count was normal in only 30 cases out of the 171, was deviated to the right in 20, and to the left in 71%. The Schilling index behaved in a similar manner. The nuclear inversion of Velez (the ratio of nuclei with 3 lobes to those with 2, which is normally greater than unity) was present in 63% of cases. The blood changes were more marked in catatonia than in either hebephrenia or paranoid dementia praecox. In the latter disorder the blood changes become less marked as the disorder becomes more chronic.

G. W. T. H. FLEMING.


The author applied the Congo red method to 100 cases of schizophrenia. The normal index of absorption varies between 50–70. In severe toxic and infective conditions from 80–100% is recovered from the blood. The author found a normal index in 34% of cases, and an increased index in 66%. He compares his results with those of Meyer and Levi, both of whom found a much greater percentage between 70–100. He concludes that there is in schizophrenia a severe impairment of the reticulo-endothelial system.

G. W. T. H. FLEMING.

Intestinal Factors in Dementia Praecox [Componenti enterogene della demenza precoce]. (Riv. di Pat. Nerv. e Ment., vol. xli, p. 483, May June, 1933.) Buscaino, V. M.

From a summary of 54 cases in the literature the author finds that enteritis was present as a factor in 53% of cases, gastritis in 50%, and colitis in 42%. In some cases there were zones of intense sclerosis of the mucosa, submucosa or sub-peritoneal coats.

G. W. T. H. FLEMING.

The Virus of Tuberculosis in Dementia Praecox. (L'Encephale, vol. xxviii, p. 561, Sept.—Oct., 1933.) Claude, H., Coste, F., Valtis, J., and van Deinse, F.

Having mentioned the considerable work done previously on the connection between tuberculosis and schizophrenia, the theory is put forward that the "intoxication" with the virus of tubercle may be responsible for psychic and psychomotor disorders along the lines of catatonia produced by bulbocapnine and other substances.

The lack of uniformity in the results of other workers in this field is regarded as being due to a failure to perceive that the schizophrenic reactions have no uniform etiology, and that the hebephrenocatatonic type, more or less toxic in origin, differs in this respect from the schizoid states which are due in the main to psychic trauma.

Some space is given to the discussion of the findings of other workers, and to the discovery, or otherwise, of tuberculous lesions at autopsy.

It is stated that using suitable cases of the hebephrenocatatonic type, and investigating the serological status with the technique of Besredka, it has been established that tuberculosis and dementia precox form a pair, but not that there is a bond of causality between them.

About 30 cases were examined. Serum and cerebro-spinal fluid were injected into guinea-pigs. Three pigs were used for each case; one was used as a control, and two were treated with an acetone extract of the bacillus.

Twelve cases yielded positive results. These are formulated as follows: The Bacillus tuberculosis was present in blood or cerebro-spinal fluid of 12 out of 19 cases.
possible cases; 8 controls suffering from random psychoses were all negative; this suggests the predominance of the role of tuberculosis in dementia praecox, and the possibility of a specific low resistance to invasion.

It is pointed out that although the cerebro-spinal fluid contains the virus of tuberculosis, yet there is no meningitis. This suggests variations in the resistance of the virus; the possibility of ultra-virus infection is considered as a special type of infection, and an analogy is drawn from syphilitic infections of the nervous system, which is striking in its closeness. Remove the hemispheric cloak of the nervous system, say the authors, and general paralysis will become tertiary syphilis and dementia praecox ordinary tuberculosis. The tentative nature of the paper is stressed, and it is stated that further research is being done.

W. McC. Harrowes.

Experimental Catalepsy. (The Action of Bulbocapnine in Cats and Monkeys, with Various Experimental Lesions of the Nervous System) (State Hospitals Press, 1932.) Ferraro, A., and Barrera, S. E.

The authors found that bulbocapnine intoxication with 25 mgm. per kgm. gave rise in cats and macacus monkeys to cataleptic symptoms and to salivary, respiratory and gastro-intestinal vegetative manifestations. In catalepsy there are always present, but in varying degree, loss of motor initiative, maintenance of passively impressed postures, maintenance of posture against gravity, resistance to passive movements, and variation in muscle tonus.

Bulbocapnine catalepsy can be reproduced in cats and monkeys in which part or the whole of the cortex has been removed. The same manifestations occur when the striatum is removed, when the mesencephalon is split through the mid-line, when the cord is hemi-sectioned, when the anterior or posterior root is sectioned, and when sympathectomy is performed. These results show that the presence of the cortex is not necessary for cataleptic symptoms after bulbocapnine. Bulbocapnine intoxication only reproduces the motor component of catatonia; catalepsy itself is not catatonia.

G. W. T. H. Fleming.

Experimental Catatonia in Man (Catatonia sperimentale nell' uomo). (Riv. Sper. di Freniat., vol. lvii, p. 374. June, 1933.) Buscaino, V. M.

The writer, after reviewing the results from bulbocapnine injections, decides that there is a very close clinical resemblance between catatonic dementia praecox and experimental catatonia in man. He still adheres to his view that the catatonic symptoms of dementia praecox are due to some toxic mechanism, the toxin concerned being of an amine type. In both bulbocapnine catatonia and clinical catatonia there are increased postural reflexes, decreased calcium and potassium in the serum, decreased chlorides in the urine, decreased depth of respiration, and diminished basal metabolism.

G. W. T. H. Fleming.

A Contribution to the Histopathological Study and Localization of So-called Experimental Catatonia (Contribución al estudio histopatológico y de localización de la llamada catatonia experimental). (Arch. de Neurobiol., vol. xiii, p. 451, May-June, 1933.) Rojas, L.

The experiments were conducted upon cats, by means of injections of bulbocapnine. The results indicated that catatonia is of toxic origin; that the anatomical substratum is essentially vascular; that the vascular condition produces results of a destructive character in the nervous parenchyma, these lesions being embolismal and hemorrhagic. The localization is in the fronto-rubral region of the brain, but the possibility of a diffuse toxæmia of the nervous system must be admitted.

M. Hamblin Smith.