possible cases; 8 controls suffering from random psychoses were all negative; this suggests the predominance of the role of tuberculosis in dementia præcox, 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 præcox 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 mgrm. per kgrm. 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 præcox and experimental catatonia in man. He still adheres to his view that the catatonic symptoms of dementia præcox 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 hæmorrhagic. 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.