The falx cerebri attaches anteriorly at the crista galli in proximity to the cribiform plate and to the frontal and ethmoid sinuses. Two layers of dura mater form the falx during its embryological development, later becoming coherent. In view of the close proximity of the anterior attachment of that structure to potential sources of bacterial infection, it seems fortunate that experience is limited with infection that enters the falx and dissects apart its two layers to form an abscess. No previous case with this complication has been found. It is possible that such a case may have been previously reported as a complication of brain abscess. Reported in that manner, it may not be easily revealed through the usual methods of literary research. The case reported here is the only one in the author's experience. Inquiry of associates indicated that they too had not heard of this entity. Consequently, it is believed worth while to bring it to attention.

It may later become evident that the use of antibiotics is the reason that such an abscess can develop. Previously the patient would have expired from one or more brain abscesses before sufficient time had elapsed for the development of a dissecting abscess of the falx cerebri. No doubt a considerable period of time must elapse for the separation of these two layers of dura mater. It also seems probable that antibiotics do not reach this region as thoroughly as they do the brain in view of the relative avascularity of the region as compared to the brain.

**CASE REPORT**

The patient was a white male college student, 30 years of age, who was admitted to the Hedgecroft Hospital, Houston, Texas, on Aug. 34, 1953. A history was obtained of headache of 3 weeks' duration. On the day prior to admission he had noted weakness of his right leg. It was thought by the examining physician that his speech was slow.

A spinal puncture was done on Aug. 34, 1953. The pressure was not measured. The cell count was 85 WBC, of which 70 per cent were polymorphonuclear and 30 per cent mononuclear cells. The protein was 35 mg. per cent. A provisional diagnosis of polioencephalitis was made by the admitting neurologist in view of the presence of a local epidemic of poliomyelitis in which cerebral manifestations were not uncommon.

_Examination_. When he was first seen by the author on Aug. 38, 1953, the findings were as follows: Temperature 101.4°, pulse rate 72, and blood pressure 130/63. The appearance was that of an acutely ill young white male. His neck was quite stiff. Kernig's sign was positive. There was complete motor aphasia. He was apparently able to understand, and he obeyed simple requests well. He would look toward a noise and toward a spoken voice. There was a
mild divergent squint but no paralysis of extraocular movements. There was no papilledema. He would not attempt to swallow, although it was stated that he did swallow on the day prior to this examination. He was able to squeeze both hands quite well on request. There was no inequality of the strength of the hand grip, although there was generalized weakness accompanying his acute illness. All extremities were appreciably spastic. He was unable to move the right leg at all. There was good strength in the left leg. Tendon reflexes were approximately symmetrical. Babinski response was positive on the right and normal on the left. Abdominal and cremaster responses were absent.

It was believed probable that the patient had a cerebritis on the basis of a bacterial infection. It did not appear likely that he had a localized abscess at that time.

Subsequently a second spinal puncture was done on Aug. 29, 1952, which showed an initial pressure of 520 mm. of water and a cell count of 400 WBC, of which 94 per cent were polymorphonuclear cells.

Course. Beginning on Aug. 25, 1952, and for 3 weeks he received antibiotics (500,000 units of penicillin IM, q. 2 hr., and 200 mg. of Terramycin IV, q.i.d.). During this period his clinical findings gradually improved. Spinal punctures on Sept. 4, 8, and 22, 1952 showed progressive drop in pressure, the pressures being 450 mm., 380 mm., and 280 mm., respectively. The spinal fluid protein also dropped from 84 mg. per cent to 23 mg. per cent. Pleocytosis diminished to 10 cells, 2 of which were polymorphonuclear. The aphasis cleared satisfactorily. He was free of focal symptoms except for mild weakness in his right leg. However, manifestations of increased intracranial pressure occurred in spite of the improvement in aphasis and paralysis. Bilateral papilledema was noted first on Sept. 1, 1952. The papilledema progressed in spite of his other improvement. On Sept. 27, 1952, after 3 days off of antibiotics, his headaches recurred with vomiting. On Sept. 29, 1952, for the first time since early