days after injury is reported. The reason for the absence of neurological signs in the presence of a large extradural lesion may be explained by the location and source of bleeding. Investigation for this lesion should be made in post-traumatic cases with symptoms suggesting increased intracranial pressure, even in the absence of focal neurological signs. Early surgical intervention is indicated to prevent pathological changes in the subjacent brain.

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OSTEITIS FIBROSA CYSTICA LOCALISATA OF THE SKULL

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The localized form of osteitis fibrosa cystica in the skull is a rare disease. It is with this entity that the present case report will be concerned. In the localized form, it must be borne in mind that there is no pathological change in the parathyroid glands nor are there associated changes in the blood calcium, phosphorus or phosphatase. The condition is mentioned only briefly in most treatises, the most complete being that of Chorobski and Davis. In a rather general discussion of bone cysts in the skull, they describe one case of the localized form of osteitis fibrosa cystica. Geschickter and Copeland consider some solitary bone cysts a form of osteitis

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fibrosa cystica. These are found almost without exception in the long bones rather than in the skull, and the age incidence is 6 to 42 years. Compere\(^3\) writes in some detail of osteitis fibrosa cystica localisata and stresses the fact that the body chemistry remains unchanged. Albright\(^1\) in his recent monograph describes the localized form of the disease but makes no mention of its occurrence in the skull. The following case report concerns a 13-year-old girl who had been seen in the Clinic some years before and whose plain skull x-rays were normal at that time.

**REPORT OF CASE**

A 13-year-old colored female was first admitted to the Presbyterian Hospital in August, 1944. At that time, she had epileptic seizures which began in 1940. These were mild in nature and of the petit mal variety. The attack lasted 30 seconds. There was no aura nor loss of consciousness and the convulsive movements, clonic in type, involved all extremities. The birth and developmental histories were normal.

**Examination.** Temperature 99.2, pulse 85, respirations 20, B.P. 110/70. She was a normally developed 8-year-old colored female. The throat was injected. There was no evidence of abnormality of the skull. She had mild bilateral nystagmus and her gait was ataxic. The remainder of the neurologic findings were within normal limits. The ataxia and nystagmus had cleared by the time the patient was ready to be discharged from the hospital. Roentgenograms of the skull at this time were normal. The lateral film is illustrated in Fig. 1. A pneumoencephalogram showed normal ventricles and normal cortical markings. No EEG was done during this admission. She was placed on phenobarbital gr. ss b.i.d. and discharged.

**Interval History.** During the next 5 years, the child got along fairly well. She had occasional seizures similar to those described at the time of her first admission. On May 25, 1949, gastroenteritis developed, presumably due to food poisoning, and during this illness her seizures became more frequent. Once again too, she became somewhat stuporous, nystagmus developed and her gait was ataxic. All of these cleared after several days. During this time she was seen in the Clinic and new x-rays of the skull were taken. These showed a single, rounded area of bone destruction with a sclerotic margin in the left frontoparietal area (Fig. 2). Because of this finding, it was felt that the patient should be readmitted to the hospital for further evaluation.

![Fig. 1. This shows film of skull made prior to the development of the cystic lesion.](image1)

![Fig. 2. Film taken 5 years later, showing the cystic area of bone destruction in the frontoparietal skull area.](image2)