Seizures occurring as a complication of chronic renal failure represent part of the generalized encephalopathy which occurs in about 15% of children with prolonged renal dysfunction (1,2). Clinical epileptiform activity often follows myoclonus and is almost always generalized in nature. Focal seizures suggest structural brain disease such as that produced by a major intracerebral hemorrhage. In 1977 Foley described a progressive encephalopathy in 5 of 35 infants and children with chronic renal failure; their age range was 9 months to 5 years. The symptoms occurred from 4 to 5 years following the initial diagnosis of renal disease. The onset of symptoms could not be related to dialysis, hypertension, or the duration of renal failure. All of the patients had evidence of slowing on EEG in addition to polyspike activity which became worse on repeated records. Only 2 of the 5 children responded well to anticonvulsants. Finally, the 2 patients in Foley's series which were transplanted did not demonstrate improvement in terms of the neurological signs and symptoms of encephalopathy.

Encephalopathy in chronic renal failure may be related to a number of features including 1) uremia, 2) calcium dysmetabolism, 3) hypertension, 4) dialysis, or 5) heavy metal intoxication, in particular aluminum. The question of whether or not uremia is important in the development of cerebral symptomatology remains uncertain since the BUN may be as low as 25 or as high as 400 at the time of neurological symptomatology. Calcium metabolism problems may occasionally contribute in a dramatic fashion. While an elevated parathyroid hormone in conjunction with a normal or low calcium is common in chronic renal failure, an occasional patient has high serum calcium along with a high PTH at the time of encephalopathic symptomatology. In these unusual cases a parathyroidectomy may produce a significant benefit in cerebral function (2,3).

Hypertensive encephalopathy may be manifested by 1) headache and altered consciousness, 2) nausea and vomiting with associated visual
disturbances, 3) generalized seizures or 4) focal neurological signs and symptoms including focal seizures. In a recent review emphasizing clinical pathological correlation, Chester et al. identified 20 cases with post mortem results (4). In addition to the symptoms and signs just mentioned, they pointed out that all of these adults had ophthalmological changes generally associated with hypertension and all had significant uremia. Symptoms 1 and 2 were the most common and the visual disturbances were described as principally blurred vision. Seizures and vocal findings were considerably less common than categories 1 or 2. The neuropathological findings in all cases included fibrinoid necrosis of the arterioles, thrombosis of arterioles and capillaries, micro-infarcts, and petechial hemorrhages. No significant edema was noted.

Another author reported on 9 children with significant hypertension secondary to renal disease associated with neurological signs or symptoms. The most pronounced features in these children were altered mental status and bilateral upper motor neuron signs. The optic fundi were normal in 6, with papilledema present in 2 and flame hemorrhages present in 1. Two of these 9 children had abnormal CT scans with a hemorrhage in 1 and a large area of decreased attenuation in another. Of significance is the fact that all 5 children recovered without neurological residual (5).

The disequilibrium syndrome or dialysis encephalopathy has no single proven etiology. The areas receiving consideration over the past several years include dopamine depletion, slow virus infection, asparagine deficiency, potassium dilution or depletion, rapid change in acid base balance, urea lag, or heavy metal accumulation. It is of interest that in one study serial EEGs appeared to predict the development of encephalopathy in several patients by the appearance of diffuse slowing with spike and wave abnormalities several days prior to neurological symptomatology (6).

There are a number of reports which weave together a body of evidence in support of aluminum as being an important consideration. The features which make aluminum toxicity an important cause include: the syndrome is most common in areas with a high aluminum in the city water, there are several reported cases which improved after decreasing the aluminum intake, in more than one case a blood aluminum level of well over the normal range of 40 was discovered during the encephalopathic symptomatology, and the neuropathological picture fails to demonstrate any structural cause suggesting a metabolic or toxic etiology (6). It should also be pointed