lower limbs are particularly involved, and the condition is slowly progressive. Bilateral nerve deafness often preceded by tinnitus, and bilateral visual deterioration leading to optic atrophy, are both common in this condition. It has been reported that the peripheral nerves show segmental demyelination,[3] though some further evidence for that may be desirable.

With the interest in cyanide metabolism arising from studies of tobacco amblyopia and Leber's optic atrophy[4-6] attention turned to the possibility of chronic cyanide intoxication as a possible cause of this condition. It was immediately recognized that the staple diet in many endemic tropical areas was a root or other vegetable rich in cyanogenic glycosides. Thiocyanate is found in high concentration in the blood of patients with this disease, and it has been suggested that this arises as a result of detoxification of cyanide.[1 7] A recent study of a similar condition in East Africa has confirmed the findings in Nigeria.[8] In the latter country it has been shown that patients with the neuropathy have a higher intake of cassava than people who did not develop the neuropathy.[1 7] The clinical improvement resulting from a reduction of the amount of cassava in the diet and the substitution of non-cyanogenic foods confirms this association.

The exact role played by vitamin B_{12} in the neuropathy is not clear, despite considerable discussion. The total serum vitamin B_{12} level is high in patients with a toxic tropical neuropathy.[1 8 9] The proportion of vitamin B_{12} present as free hydroxycobalamin and as cyanocobalamin still requires to be defined. Though it has been suggested that vitamin B_{12} has a primary role in the detoxification of cyanide, this is not finally proved. Much remains to be elucidated about the effect of cyanide on the nervous system. For instance, an entirely different symptom complex of amyotrophic lateral sclerosis, Parkinson's disease, and dementia is seen in Guam.[10] It has been suggested that this results from intoxication with cycasin, a cyanogenic glycoside present in food prepared from the cycad nut. Experimentally this glycoside causes a cerebellar degeneration in experimental animals.[11]

[1] Osuntokun, B. O., *Brain*, 1968, **91**, 215.
[2] Osuntokun, B. O., Durowoiu, J. E., McFarlane, H., and Wilson, J., *British Medical Journal*, 1968, **3**, 647.
[3] Williams, A. O., and Osuntokun, B. O., *Archives of Neurology*, 1969, **21**, 475.
[4] Smith, A. D. M., *Lancet*, 1961, **1**, 1001.
[5] Wilson, J., *Brain*, 1963, **86**, 347.
[6] Wilson, J., Linnell, J. C., and Matthews, D. M., *Lancet*, 1971, **1**, 259.
[7] Osuntokun, B. O., Monekosso, G. L., and Wilson, J., *British Medical Journal*, 1969, **1**, 547.
[8] Makene, W. J., and Wilson, J., *Journal of Neurology, Neurosurgery and Psychiatry*, 1972, **35**, 31.
[9] Monekosso, G. L., and Wilson, J., *Lancet*, 1966, **1**, 1062.
[10] Hirano, A., Malamud, N., Elizan, T. S., and Kurland, L. T., *Archives of Neurology*, 1966, **15**, 35.
[11] Hirono, I., Shibuya, C., and Hayashi, K., *Proceedings of the Society for Experimental Biology and Medicine*, 1969, **131**, 593.

Duodenal Diaphragm

The commonest cause of obstruction to the gastric outlet in the adult patient is peptic ulceration, usually duodenal, but occasionally in the pyloric or prepyloric region of the stomach. Second to this comes carcinoma of the gastric antral region. Other obstructive lesions are numerous but rare. They include adult pyloric hypertrophy, benign tumours, infiltration by one of the reticuloses or an adjacent carcinoma of the pancreas, heterotopic pancreatic tissue, and prepyloric mucosal diaphragm.[1] One of the least common but nevertheless interesting causes is a persistent congenital diaphragm in the second part of the duodenum.

Congenital duodenal occlusion presenting as a neonatal emergency may be due either to a diaphragm or to a complete obliteration of the duodenal lumen, which ends as a cul-de-sac. Though the latter may result from vascular occlusion in utero, it is probable that the diaphragm represents incomplete vacuolation of the proliferating epithelial lining of the duodenum, which leads to its temporary obliteration in the sixth and seventh weeks of fetal life.[2] About 60% of cases have some other congenital anomaly, and about one-third of the patients have Down's syndrome (mongolism).

If the occlusion is complete, symptoms appear within hours of birth. But in some cases a lumen may be present in the diaphragm, so that life is sustained for months or even years. Rarely the lumen is wide enough to allow survival into adult life. Long-standing incomplete obstruction may result in dilatation of the proximal duodenum,[3] and in some cases the mechanical action of the food may balloon the septum into the distal duodenum like a windsock to produce an intraluminal duodenal diverticulum. Zatzkin and his colleagues[4] have given a clear description of the radiological appearances of such a case (the third to be reported), and a remarkable example of such a diverticulum was reported by A. J. Levi and L. Kreel[5] in a young man of 24 who had undergone gastrojejunostomy at the age of 6 days for what was thought to be a duodenal atresia but which, in adult life, proved to be a duodenal diaphragm.

J. L. Bilton and S. Yap[6] have recently summarized the features of duodenal diaphragm as well as reporting two more examples of this condition. The history, in adults, is usually one of persistent vomiting. The vomitus may or may not contain bile, this depending on whether the diaphragm is below or above the ampulla of Vater. There may be epigastric distention and loss of weight, and there may be melaena or haematemesis. Though the diagnosis is not often made preoperatively, careful *x*-ray studies, with hypotonic duodenography, may show the obstruction of the second portion of the duodenum with a small trickle of contrast material passing through the orifice in the diaphragm.

In a condition as rare as this it is not possible to be dogmatic about surgical treatment. Though duodenojejunostomy is regarded as the treatment of choice in infants, F. D. Threadgill and A. Hagelstein[3] report two cases in which this procedure was done and met with failure. One patient was left without relief, the other failed to respond to subsequent gastrojejunostomy and was eventually cured by partial gastrectomy. C. N. Hudson[7] reported a case in which excision of the diaphragm and transverse closure of the duodenum was followed by excellent results, and others have had similar experiences.[4 5] Of the two patients reported by Bilton and Yap the first responded well to this simple excision with duodenoplasty, but the second, suffering from severe gastrointestinal haemorrhage, was treated by vagotomy and gastrojejunostomy, with equally good results.

[1] Ellis, H., in H. N. Harkins, and L. M. Nyhus, *Surgery of the Stomach and Duodenum*, 2nd ed. Boston, Little, Brown, 1969.
[2] Krieg, E. G., *Annals of Surgery*, 1937, **106**, 33.
[3] Threadgill, F. D., and Hagelstein, A., *Archives of Surgery*, 1961, **83**, 878.
[4] Zatzkin, H. R., Macy, J. J., and Kveton, F. W., *American Journal of Roentgenology*, 1959, **82**, 1036.
[5] Levi, A. J., and Kreel, L., *Proceedings of the Royal Society of Medicine*, 1963, **56**, 168.
[6] Bilton, J. L., and Yap, S., *American Journal of Gastroenterology*, 1971, **56**, 457.
[7] Hudson, C. N., *British Journal of Surgery*, 1961, **49**, 234.