Clinical Section

President T C Hunt DM

Meeting November 11 1960

Cases

Carotid Body Tumour Associated with Diarrhœa and Abdominal Pain

Zoë D Chamberlain MB (for T C Hunt DM)

Mr F C, aged 54

History: 1944: A small mass was noted in the right side of the neck. It was excised and, as it was thought to be a secondary deposit of carcinoma, the area of excision was irradiated.

1951: The mass recurred and gradually increased in size. Biopsy showed the appearances of a carotid body tumour.

1956: The patient began to experience severe cramping abdominal pain and bouts of diarrhœa (7–8 loose motions daily, without blood or mucus). Investigation elsewhere suggested steatorrhœa and he was put on a gluten-free diet without effect.

1958: Reinvestigation suggested intestinal hurry rather than steatorrhœa.

1960: Admitted to St Mary's Hospital (Dr T C Hunt). Symptoms unchanged. During the previous four years had received opiates, chlorpromazine and ganglion-blocking drugs without effect.

On examination: Florid complexion. Firm swelling on the right side of the neck, irregular, tender, approximately 5 × 4 in. (Fig 1). BP 110/70. Pulse 70 regular. Heart normal. Chest clear. Tendon reflexes absent, C.N.S. otherwise normal. Slight tenderness in the epigastrium and left hypochondrium. Liver palpable 1½ in. below right costal margin. Sigmoidoscopy normal on several occasions.

Investigations: X-rays: Barium meals: January 1958, normal. November 1958, barium reached the colon in 20 mins and the rectum in one and a half hours; normal colonic mucosa. August 1960, barium reached the rectum in one and a half hours; otherwise normal.

Chest: normal. Cervical spine: narrowing of C.V. 6/7 disc space.

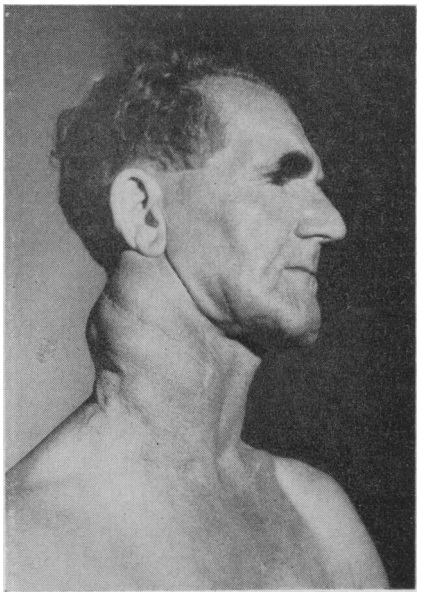

Fig 1 *Photograph showing tumour before excision*

Skull: no evidence of bone erosion.

Urine: 5 hydroxy indole acetic acid output normal. No catechol amines.

Stools: No intestinal pathogens. Fæcal fats 2·4 g/three days. (80 g fat/day diet.)

Liver function tests: S.G.O.T. 35, S.G.P.T. 50 units/ml. Total bilirubin 0·7 mg/100 ml. Alkaline phosphatase 4·1 K.A. units/100 ml. Thymol flocculation negative.

Serum protein: Total 6·9, albumin 4·2, globulin 2·7 g/100 ml.

Glucose tolerance test normal.

Serum calcium 9·3 mg/100 ml. Plasma electrolytes and blood urea normal.

Hb 13·6 g/100 ml. W.B.C. 7,000 (normal differential count). E.S.R. 5 mm in one hour (Wintrobe).

ECG normal.

Operation: The mass was removed in two stages – the first was performed by Professor C G Rob (under hypothermia) on 29.8.60 and the second by Mr J Fairgrieve on 13.10.60. The locally extensive tumour was excised without ligation or grafting of the carotid vessels and without the patient developing a hemiplegia as has so often followed operations of this type.

Histology: On microscopy the mass was shown to be a carotid body tumour of the type resembling the normal carotid body. The chief cells were polygonal (with large volumes of pale staining, finely granular cytoplasm and hyperchromatic nuclei), divided into clusters by fibrous vascular stroma. It is unusual for carotid body tumours to metastasize to lymph nodes but one section of the mass showed such a node with lymphatic and tumour tissue within the capsule.

Comment: The mass caused minimal local disturbance to the patient despite its size and slight tenderness, but the diarrhœa and abdominal pain have caused him great distress. His symptoms of diarrhœa and pain have improved very considerably post-operatively; he now has an average of 2 motions a day instead of the previous 7 or 8. This case was shown as we suggest that there may be a relationship between the tumour (first noted in 1944) and the severe diarrhœa with intestinal hurry (first occurring in 1956). It seems possible that the secretion of some substance by the carotid body tumour affected the gut and caused the symptoms although, as yet, this substance has not been identified.

Lead Encephalopathy

A K Thould MD MRCP (for M G Ashby MRCP)

Lead encephalopathy is now very rare but occasional examples are still found in emergency admissions to general medical wards.

History: Mrs W, aged 46, had been employed in a lead accumulator factory for the five years before this admission in April 1960, during most of which time she had supervised the spreading by machine of a moist mixture of litharge and red lead on to lead accumulator plates. She was inadequately protected from fumes and her environment was very dusty. She had been in good health previously, and had had one live child now aged 14, and one miscarriage at twelve weeks four years before becoming exposed to lead.

In September 1959, glycosuria was found and a glucose tolerance test gave a mildly diabetic type of curve. She was treated by weight reduction and was in good health until January 1960 when she developed frontal headaches when tired. In March 1960 two generalized epileptiform convulsions occurred followed by transient diplopia and forgetfulness. She had one further epileptiform convulsion and, two weeks before admission on 29.4.60, she became delirious. After treatment she remembered that she had had some colicky abdominal pain for two or three months.

She was admitted to the Whittington Hospital with a provisional diagnosis of an intra-cranial space-occupying lesion.

On examination: moderately obese, slow in speech and thought, lethargic. Marked bilateral papillœdema and three-flame-shaped hæmorrhages visible in the left fundus. She had an obvious lead line round the base of the teeth, but not at the anus. Heart clinically normal, BP 105/65. (The blood pressure was normal throughout), pulse 110, afebrile. No evidence of muscular weakness; all reflexes present, though sluggish. Conjunctivæ pale.

Investigations: Hb 75% Haldane, R.B.C. 4,165,000. P.C.V. 35%, M.C.V. 84 cu. μ M.C.H.C. 30%. Basophilic stippling was present in approximately 3,700 red cells per million counted. E.S.R. 32 mm in the first hour (Westergren), W.B.C. 10,700 (polys. 68%, lymphos. 30%). The red blood cells appeared hypochromic. C.S.F. clear, protein 45 mg%, white cell count 10/c.mm, sugar 75 mg%, this specimen being obtained from the ventricles. Coproporphyrin III was present in excess in the urine, and a twenty-four-hour specimen of urine contained 400 μg of lead (normal for this laboratory 30–80 μg). Blood urea 18 mg/100 ml, a glucose tolerance test showed a normal curve, no glycosuria. Total urinary creatinine 960 mg/24 hours, creatine nil. S.G.O.T. 26 units ml.

A ventriculogram was normal (Mr I R McCaul) and a diagnosis of lead poisoning with lead encephalopathy was made.

Treatment: Calcium di-sodium versenate, 3g in 50 ml of normal saline, was given by intravenous injection, over 5–10 minutes, once daily for five days, as recommended by Sidbury (1955). Urine was collected for the first and last twenty-four hours of this course and the lead content estimated. Only a small quantity of lead was found in