stain a dusky slate blue, the parathyroid adenomata all stain dark blue to purple. In most patients the thyroid gland and the strap muscles are tinged a light blue. Thyroid cysts when present seem to take up the dye and stain dark blue, but this does not prove confusing. No troublesome side effects were encountered and, in particular, no evidence of myocardial toxicity as reported occasionally with toluidine blue (Yeager and Krementz, 1969). Undoubtedly the pseudocyanotic pallor imparted to the patient during surgery and for a few hours after deprives the anaesthetist of one of his vital signs and calls for extra caution in assessing oxygen requirements. We found it wise to warn the ward staff that a "blue" patient was being returned from the theatre and to tell patients that their urine would be blue for a week or so.

Identification of Glands

Case No.	Preoperative Infusion Time	Parathyroids			
		R. IV	R. III	L. IV	L. III
1 ..	2½ hours	0	—	—	—
2 ..	2 „	—	—	—	0
3 ..	1½ „	—	—	—	0
4 ..	1 hour +	0	—	0	—
5 ..	1 hour	0	0	0	000
6 ..	1 „	—	0	0	000
7 ..	1 „	0	—	0	—
8 ..	1 „	—	0	—	0
9 ..	1 „	0	0	—	0
10 ..	1 „	—	000	—	—
11 ..	1 „	0	—	—	000
12 ..	1 „	0	000	—	0
13 ..	1 „	—	—	—	000
14 ..	1 „	00	00	00	00
15 ..	1 „	0	—	0	000
16 ..	1 „	0	000	0	0
17 ..	1 „	0	000	0	0

0 = Normal parathyroid. 00 = Parathyroid hyperplasia. 000 = Parathyroid adenoma.

Discussion

On the basis of these observations this technique is recommended when faced with the problem of identifying parathyroid tumours. I suggest that it is likely to be useful also where preservation of normal parathyroids is difficult as in total thyroidectomy for carcinoma. So far it has not been possible to determine by histological techniques where methylene blue is taken up in the parathyroids, for the routine methods of fixation decolorize the dye and unstained sections are so pale at the required thickness that no detail can be made out. Precipitation of the dye is being attempted to elucidate this point, while tagging methylene blue with a radioactive isotope marker is an exciting prospect and could if practicable enable preoperative scanning of the neck and mediastinum to locate an adenoma. This possibility is being explored.

I am grateful to Mr. A. S. Till, Mr. M. H. Gough, and Mr. G. E. Moloney for their encouragement and permission to initiate this technique on cases under their care and to the patients themslves for allowing me to stain them in this way.

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MEDICAL MEMORANDA

Renal Artery Dysplasia with Hypertension in Neurofibromatosis

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Fifteen cases of renal artery stenosis associated with neurofibromatosis have been reported, mostly in the European literature (Allan and Davies, 1970; Smith *et al.*, 1970). We report a further case. In addition to stenosis of the main renal artery an unusual endothelial cell proliferation of the intima with atrophy of the media and elastic layers of the arterioles was shown.

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Case Report

An 11-year-old boy (weight 22 kg) was admitted to hospital with headaches and vomiting. Apart from one sibling who had nerve deafness the family history was negative. He had a pronounced kyphoscoliosis, multiple café au lait spots, a plexiform neuroma in his thenar eminence, and two pea-size nodules in the intercostal spaces. His blood pressure was 220/150-270/220, he had papilloedema and retinal haemorrhages, but there were no signs of congestive failure. E.C.G. showed severe left ventricular strain. Proteinuria was not found. Blood urea was 24-34 mg/100 ml. Urinary catecholamines were repeatedly normal. Aortography showed a left renal artery stenosis (Fig. 1).

On the daily regimen of methyldopa 3 g, bethanidine 80 mg, chlorothiazide 500 mg, with potassium supplements and propranolol 120 mg the blood pressure remained in the same range. Prompt control was achieved with the addition of intravenous diazoxide (300 mg). This drug had to be repeated initially after six days and subsequently at intervals of two to four days. The requirements increased over succeeding weeks until after one month he needed diazoxide 300 mg eight-hourly. It was decided to attempt a surgical repair. At operation, because of difficulties imposed by the skeletal abnormalities a nephrectomy was carried out. The stenosed segment of the main renal artery was not obtained. An intercostal nodule was excised.

Histologically the nodule was a neurofibroma. The kidney (75 g) was macroscopically normal. Microscopically the cortex showed occasional hyalinized glomeruli. Most of the glomeruli were normal. The most striking features were randomly distributed lesions in the arterioles; many looked relatively normal. In others (Figs. 2 and 3) a pronounced intimal proliferation of endothelial cells was seen, at times almost obliterating the lumen. In places there was

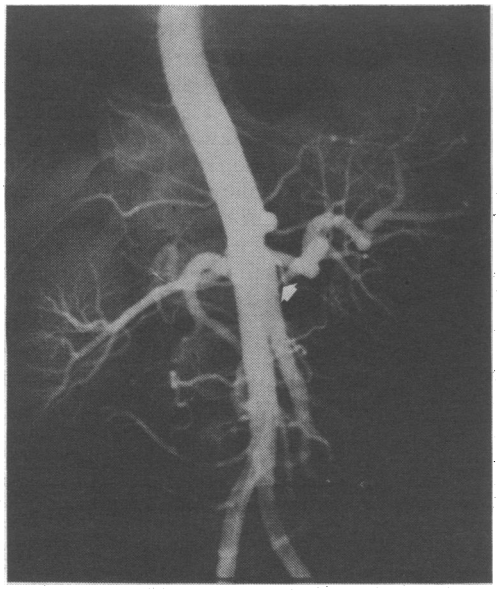

FIG. 1—Renal arteriogram showing stenosis of the left main renal artery (arrowed) close to its origin with post-stenotic dilatation.

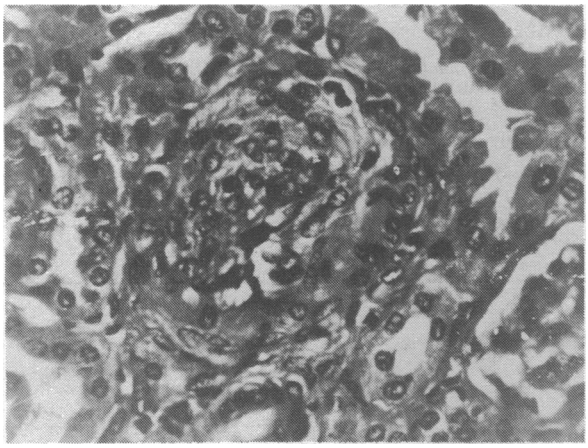

FIG. 2—Renal arteriole showing pronounced endothelial proliferation encroaching on the lumen. The media is thinned. (H. and E. × 137.)

a profuse fibrous transformation of the intima, with thinning and fragmentation of the elastic layer and atrophy of the media.

One year postoperatively the blood pressure was maintained within the range 120/75-130/90 on a daily dosage of methyldopa 1 g, bethanidine 90 mg, frusemide 20 mg, and spironolactone 75 mg. His blood urea was 42 mg and E.C.G. was normal.

Comment

It would appear that the association between neurofibromatosis and renovascular hypotension is more than coincidental. In addition to stenosis of a major vessel involvement of small arteries and arterioles has been reported (Khalatbary, 1959). Reubi (1945) described three histological variants. (1) A pure intimal form where vessels 50 to 400 μm in diameter show

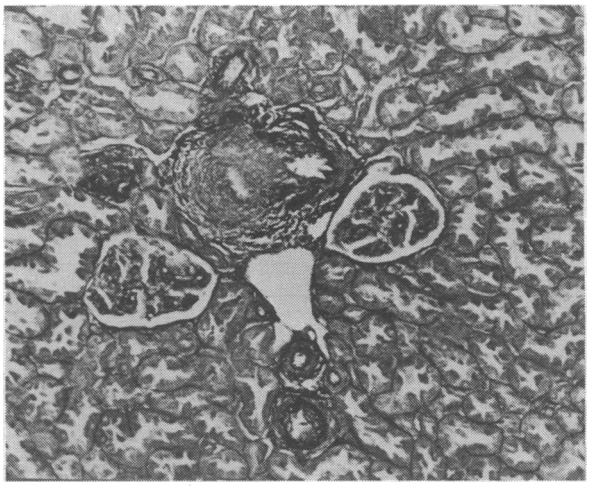

FIG. 3—Branching renal arteriole showing pronounced fibrous intimal thickening. There is virtually complete loss of the elastica in its left-hand branch. There is advanced atrophy of the media. (Elastic and Van Gieson stain. × 40.)

layers of endothelial cell proliferation. The media and elastic layers are thinned and atrophied. The adventitia is normal. In more advanced forms it is characterized by fibrous transformation of the intima. These lesions were shown in the present case (Figs. 2 and 3). (2) An intimal and aneurysmal form where larger vessels show fibrohyaline thickening of the intima and fragmentation of the elastic lamina, leading to aneurysm formation. (3) Fusocellular nodules between the media and adventitia in vessels 200-700 μm. Feyrter (1959) described a further variant, an epithelioid form, where a proliferation of fusiform cells involves the entire vessel wall.

In 15 out of 16 cases of renovascular hypertension associated with neurofibromatosis the diagnosis was made before the age of 20. One-third of the cases had an associated coarctation. In four cases the renal artery stenosis was bilateral.

The somewhat disappointing response to surgery in this and other cases reported suggests the possibility of the microscopic forms of vascular lesions in the contralateral kidney. This possibility was suggested from the review of Khalatbary (1959); in this review of cases where histological vascular lesions were found, out of 12 patients whose blood pressure measurements were available 6 had hypertension. Thus a cautious approach to surgery is advised. This is emphasized by the fact that in most reports attempts at reconstructive surgery were unsuccessful and nephrectomy was required.

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