purpuric eruptions on the trunk and limbs. On the following day he developed bluish discoloration of several toes of both feet suggesting early gangrene. The skin overlying the area was warm and the peripheral pulses were palpable. The purpuric eruptions became more extensive and he had several bouts of haematemesis. At this stage, the possibility of disseminated intravascular coagulation was considered. No specific diagnostic laboratory test was then available, however, his platelet count was only 25,000 cu.mm. and thrombocytopenia is probably a constant feature in intravascular coagulation.

It was decided to treat the child with continuous intravenous heparin. The decision to anticoagulate the child was not an easy one as we were then faced with the paradoxical situation of gangrene on one hand and spontaneous bleeding on the other. After 48 hours of heparin therapy the child's condition improved remarkably, with rise in platelet count and cessation of spontaneous bleeding. The progress of gangrene was halted. During the course of several days the discoloration of the toes gradually disappeared, apart from small black necrotic areas at the tip of two toes. Heparin therapy was discontinued after seven days. Apart from loss of nail beds of his toes his feet are now perfectly normal.

The diagnosis of intravascular coagulation in this case was made mainly on clinical grounds and it does not fulfil the criteria of this syndrome as suggested by Corrigan and others;—namely, the combination of the thrombocytopenia, reduction of coagulation factors X, V, VII, and fibrinogen, and presence of fibrinolitic split products in the serum, but the dramatic response to heparin therapy was gratifying and possibly diagnostic. Disseminated intravascular coagulation or consumption coagulopathy, a relatively new terminology, is well documented in relation to virus diseases.2—We are, etc.,

A. K. R. CHAUDHURI
PETER MCKENZIE
Belvidere Hospital,
Glasgow E.I.

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2 McCready, D. G., and Margarrett, W., Archives of Internal Medicine, 1967, 120, 129.

Temporary Cardiac Pacing Using the Subclavian Vein

STIR.—We were delighted to see the paper by Drs. M. B. Macaulay and J. S. Wright (24 October, p. 207). We hope that these authors' experience with supraclavicular percutaneous introduction of the pacing electrode into the subclavian vein will popularize this little-known technique. Wider appreciation of its enormous advantages over arm-vein routes in terms of electrode stability and comfort for the patient should do much to counter the nihilistic attitude towards the treatment of postinfarction heart block which now prevails in too many centres.

We adopted the subclavian technique some three years ago and can confirm its simplicity, safety, and patient-acceptability. Thanks to clear instructions from Dr. B. W. Lassers and the enthusiasm of Dr. J. H. N. Bett (now of Melbourne, Australia) the method proved easy to learn. The left subclavian vein is preferred here where temporary pacing is required in patients with chronic heart block so that later use of the right internal jugular vein (for insertion of a pacemaker) is not precluded. Experience of the left subclavian may also be more convenient for the right-handed operator and the natural curve of the temporary electrode favours correct placement of the tip in the right ventricular apex when introduced from the left side. There are unimportant differences in the equipment used and one potentially important difference—that, we use only No. 5 F.G. bipolar electrodes, having known the stiffer No. 6 F.G. electrodes to perforate the heart in times gone by.

The Table shows the few complications we have encountered in 60 attempts to establish pacing with the subclavian technique and compares them with the

<table>
<thead>
<tr>
<th>Subclavian Vein</th>
<th>Antecubital Vein</th>
</tr>
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<tbody>
<tr>
<td>Technique Attempted</td>
<td>60</td>
</tr>
<tr>
<td>Pacing Established</td>
<td>60</td>
</tr>
<tr>
<td>Failed Procedure*</td>
<td>6</td>
</tr>
<tr>
<td>Subsequent Electrode Displacement</td>
<td>2</td>
</tr>
<tr>
<td>Phlebitis</td>
<td>0</td>
</tr>
<tr>
<td>Wound Infection</td>
<td>0</td>
</tr>
</tbody>
</table>
| Patient Discomfort | ≤ | +1 + + +

(*includes 2 subclavian artery punctures due to inexperienced complications of 19 temporary pacing procedures attempted during the same period using antecubital veins. No instances of pneumothorax, brachial plexus injury, or air embolism were encountered. The latter complication might be anticipated in patients with low central venous pressure1 unless tilted head down, and in those with obstructive airways disease and uncontrollable coughing; the subclavian technique is unsuitable for use in such cases.

Failure to introduce the electrode into the subclavian vein is now rare (a foam plastic pad, 8 cm. thick, beneath the occiput appears important to success). In a recent case where difficulty was encountered the patient suffering recent asymptotic arrest throughout, satisfactory pacing was speedily established via the right internal jugular vein as described by Hoffman and Sokol. We are, etc.,

DAVID W. EVANS.
M. CLARKE.

REFERENCES

Skin Disease and the Gut

STIR.—I have read with interest your leading article (1 August, p. 240) and the subsequent correspondence (29 August p. 521, and 5 September, p. 586) on skin disease and the gut and the latest contribution of Dr. Janet Marks and Professor S. Shuster on dermatoglyphic enteropathy (12 September, p. 618). I am writing to draw attention to the similarities between dermatoglyphic enteropathy and the enteropathy associated with extraintestinal tumours—which can be considered as “neoplastic enteropathy”—and to suggest that folate acid deficiency is mostly responsible for the small bowel dysfunction in both conditions.

Although jejunal mucosal abnormalities have been described in two patients with extraintestinal neoplasia1 two recent investigations have shown that such changes are indeed very rare. However, as in dermatoglyphic enteropathy, malabsorption does exist in the absence of morphological mucosal abnormalities. While the incidence and severity of malabsorption is proportional to the extent of rash in patients with skin disease, they appear to be related to the presence and extent of metastases in patients with carcinoma.

Folic acid deficiency is quite common in patients with extensive skin disease, as well as in patients with malignant neoplasia, and it is generally believed to be due to an impaired synthesis of D.N.A. A defect in folate deficiency. As the cell turnover in the intestinal epithelium is higher than in any other in vivo cell population, interference with normal regeneration of villi, with resulting malabsorption, is only to
be expected in patients with folate acid deficiency (clinical or subclinical). The findings of Kaimis, Summerly, and Giles (personal communication to Marks and Shuster, 12 September, p. 618) and the demonstration of malabsorption in the proximal small bowel which is the site of absorption of folic acid, would seem to indicate that, in addition to an increased demand, there may be malabsorption of folic acid in these patients which would further exacerbate folate deficiency.

As correctable malabsorption of vitamin B₁₂ has been described in patients with folic acid deficiency, it would seem likely that the malabsorption of vitamin B₁₂ in patients with various skin diseases is also due to folic acid deficiency. I wonder whether the authors have repeated the study after a course of folic acid. It would also be interesting to see whether the small bowel dysfunction in dermatogenetic enteropathy reverts to normal on folic acid therapy alone without local treatment of the rash. If it does (as I suspect) in addition to the local treatment of dermatosis early folic acid therapy would seem to be indicated in patients with enteropathy, even when there are no obvious haematological changes.

Similar explanation probably holds true in enteropathy associated with various chronic debilitating illnesses and also in conditions where increased cellular proliferation is a predominant feature. —I am, etc.,

B. N. SOMAYAJI.

Meharry Medical College,
Nashville, Tennessee, U.S.A.

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8. Lesher, S. W., and Busman, J., Recent Results in Cancer Research, 1966, 16, 89.

Myocardial Infarction and the G.P.

SIR,—The hospital doctor (14 November, p. 433) who can include without explanation a delay of 2 days in seeing a patient with a coronary thrombosis in a discussion of factors influencing early death clearly needs to spend some time as a general practitioner's receptionist.

He would learn some of the form in which coronary thrombosis can be presented when first brought to the notice of the medical services. He might then be in a better position to inform general practitioners what information is useful to them so as to avoid once again finding that only five out of 41 of their letters contain any useful information. Those of us who work in hospital can make everyone's work easier by explanation rather than by damning comment.—I am, etc.,

JOHN L. STRUTHERS.

School of Tropical Medicine, Liverpool.