at 2 weeks and 6 weeks, and 6-keto-PGF1α (a non-enzymatic degradation product of epoprostenol) production was compared at 2 weeks and 6 weeks, and 6-keto-PGF1α could be detected in control grafts at 2 weeks, but values ranged from 0–10% of aorta at 6 weeks. The site of this production is uncertain since no endothelialisation of the graft was apparent by light or electron microscopy.

The results imply that mesothelial cell seeding of dacron grafts may be a possible means of introducing PGI2 into dacron arterial prostheses soon after the time of surgical implantation. Further animal studies are essential before a human study could be considered, but this technique, in conjunction with postoperative PGI2 infusion, may have an important future application.

We thank J. G. Pollock, J. Drury, and J. M. F. Clarke for surgical expertise. A full report will be published elsewhere.

8. Herman AG, Clayson M, Monzada S, Vaze JR. Biosynthesis of prostacyclin (PGI1) and 12L-hydroxy-5,8,10,14 eicosatetraenoic acid (HETE) by pericardium, pleura, peritoneum and aorta of the rabbit. Prostaglandins 1979; 18: 439–52.

**NOMOGRAM FOR PREDICTIVE VALUES AND EFFICIENCIES OF TESTS**

Sir,-A clinician reading about a binary test, one that gives results that are either positive or negative in relation to disease in question, wants to know if the test will help his patients. He needs to have the probability that the patient has the disease when the result of the test is positive; this is known as the predictive value of a positive test result (PVpos). He also needs the probability that the patient does not have the disease when the test is negative (PVneg) and efficiency of the test, which is the proportion of all results positive or negative, that are true results. Unfortunately, PVpos, PVneg, and efficiency vary with the prevalence of disease in the clinician’s population, whereas sensitivity and specificity of a test are constants. By using complex formulae or tables, or a computer program physicians can calculate PVpos, PVneg, and efficiency from a test’s sensitivity and specificity and their knowledge (or “best guess”) of disease prevalence in their patients. However, simpler ways of doing this are needed and I propose the following equations:*  

\[
PV_{pos} = \frac{P}{P + (1-P)L_{pos}}
\]

\[
PV_{neg} = \frac{P}{P + (1-P)L_{neg}}
\]

Efficiency = \((P \times \text{sensitivity}) + (1-P \times \text{specificity})\)

*The mathematical derivation of these equations may be had from J. M. P.

**PAINFUL DIABETIC NEUROPATHY**

Sir,—We read with interest your Jan 12 editorial on pain perception in diabetic neuropathy. The pathogenesis of painful symptoms in diabetic neuropathy is complex. Whilst reduction in small myelinated and unmyelinated fibres and axonal degeneration is important, the role of hyperglycaemia cannot be understressed. Besides chronic hyperglycaemia, wide fluctuations in blood sugar (eg, after a meal), may be responsible for exacerbating painful symptoms.

At the March, 1984 meeting of the Medical and Scientific Section of the British Diabetic Association we suggested that painful neuropathic symptoms can be rapidly relieved by maintaining 24 h euglycaemia, and a full report of our findings will appear elsewhere. We use an open-loop intravenous insulin infusion system, which differs from closed-loop systems in ensuring delivery of insulin in the absence of continuous feedback control, and so is not cumbersome and is inexpensive.  

Nomogram to calculate predictive values, knowing prevalence of disease (P) and values of Lpos and Lneg.

Line from P (on right) through Lpos gives PVpos (on left). Similarly for P and Lneg to give PVneg.