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Superficial thrombophlebitis

Sir,-The paper by Dr P P Mehta and others (13 September, p 614) shows that unparalled resolution of signs and symptoms of superficial thrombo-
phlebitis developing after continuous intra-
venous infusion. It also shows that 68 patients had infusions in situ for three days or more. No wonder the authors were able to collect 100 patients with “infusion arms.”

Superficial thrombophlebitis developing after intravenous infusion is largely prevent-
able by ensuring that infusions are taken down each night. This is more physiological than running them continuously for 24 hours, relieves night nurses of the particularly diffi-
cult task of keeping to infusion schedules in a dark ward, and enables the patients to have a less disturbed night’s sleep. Such a policy does require the housemen to insert intra-
venous cannulae each morning, but as the in-
fusions last no more than 16 hours small needles can be used. These can be put into small veins with relative ease, and blood can be taken through them for analysis before setting up the infusion. In addition it ob-
viates the need to disturb the houseman when an infusion is required at the bedside.

Since this policy was started on the professorial surgical unit at Guy’s three years ago “infusion arms” have been a rare event, and in each case cannulae have been left in the veins for more than 24 hours at a time.

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An aetiological agent in Whipple’s disease?

Sir,-I read with interest the article by Dr R L Clancy and others (6 September, p 568) on the isolation of an aetiological agent from a patient with Whipple’s disease. I should like to make two comments.

Firstly, I had difficulty in deciding whether the authors considered the agent to be a cell-
wall-deficient (CWD) form of Streptococcus dysgalactiae or the bacterium itself. My con-
fusion arose because, on the one hand, jejunal biopsy and lymph node culture materials were interpreted as shown by electron microscopy, while on the other hand the authors reported “the isolation of a CWD bacterium from a pro-
longed lymph node culture with features that strongly suggested that this organism was an aetiological agent.” The authors showed the value of using hypertonic medium, but the growth of a CWD form in such medium was considered less important since it existed in this state in the patient. It cannot be excluded that the streptococcal organisms were in the bacterial form, perhaps in small numbers so that isolation by con-
ventional means was not possible, and that they were converted to the CWD form in vitro by the use of hypertonic medium.

Secondly, even if the isolated streptococcus is the same as that observed in the diseased tissues of the patient, it does not follow that it is necessarily causative. If it is an aeto-
logical agent, then the interesting observations made by the authors are very unlikely to prove unique, even if more than one agent is responsible for causing Whipple’s disease. Thus, while in no way detracting the work, I feel that without confirmation it is premature to consider the isolated streptococcus as a cause of the disease on the basis of observations made on a single case.

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\* \* We showed this letter to Dr Clancy and his colleagues, whose reply is printed below.

ED, BMJ

Sir,—Two points are made by Dr Taylor-
Robinson and are well taken.

Firstly, the nature of the isolated organism.

The organism was isolated only under cultural conditions that were “characteristic” of those under which cell-wall-deficient (CWD) organisms grow. The initial varia-
tion in morphology and staining of the bacterial and the eventual stable growth in our blood agar are more consistent with enhanced cell wall stability following isolation than conversion in vitro to CWD forms in the presence of hypertonic medium. The electron micrographic appearance of numerous bacteria in the tissues suggests that the hypertonic medium did not have its effect by allowing growth of organisms present in small numbers. The presence of apparent bacterial cell walls in the electron micrographs neither supports nor excludes functional “cell wall deficiency.”

Secondly, is the organism the prime aetiological factor in Whipple’s disease?

Clearly no definite statement can be made from this study on one patient. Our studies to define the characteristics of this organism, the dramatic clinical improvement following tetracycline therapy associated with a dis-
appearance of bacteria from the tissues, and a reduction in antibody titres to the isolated organism following antibiotic therapy (un-
published) support but do not prove an aetiological role for the organism in our patient. The production of lesions similar to those of Whipple’s disease in a rabbit in-
fected with the organism provides further support to this argument.

The questions raised by Dr Taylor-
Robinson are critical, and for definitive answers further data are required. We certainly do not intend to convey the impression that the organism isolated in our study is necessarily the cause of Whipple’s disease but hope we can stimulate study of more patients with this interesting and com-
plex disorder, to understand better its aetiology.

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Diagnostic criteria in the 131I-fibrinogen test

Sir,—Mr V C Roberts’s paper (23 August, p 455) is a timely and valuable statement of problems encountered with the Pitman rate-
meter and we strongly support his plea for standardization of diagnostic criteria in the

131I-fibrinogen test. The need for standard-
isation is clearly illustrated by our figures. A total of 314 patients were scanned...