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### Phlebitis vs. Cutaneous Vasodilatation

*To the Editor:*—I was most interested in the report by Miller and Stark of acute phlebitis from nitroprusside infusion,<sup>1</sup> and would like to offer an alternative explanation. Some years ago I used to give trimetaphan to patients by continuous infusion into an internal saphenous vein at the ankle. On a number of occasions I noticed the appearance of a red streak about 1 cm wide in the skin immediately superficial to the vein as it ran upwards in the subcutaneous tissues of the leg and thigh. My first impression was that I had introduced a virulent streptococcal infection at the site of the venipuncture and this was septic lymphangitis, for the appearances were substantially those of this condition. The patients' temperatures, however, were not increased, and there was no other evidence of infection. Further, the red streak disappeared within 30 min of stopping the trimetaphan. In the end I concluded that some of the trimetaphan was escaping through the wall of the vein, perhaps via the vasa vasorum, into the surrounding tissues. By this means the drug was able to reach the skin, there to exert its specific vasodilator action, thus

causing the red streak in the skin. It did not seem to be phlebitis, for there was no accompanying thickening of the vein wall. Perhaps the same phenomenon occurs with sodium nitroprusside.

Support for this concept comes from consideration of the opposite effects of vasoconstrictor solutions, such as norepinephrine administered by continuous infusion into superficial veins. Necrosis of the skin along the line of the vein was reported as a result of this procedure. More recently, the same event has occurred following the infusion of dopamine into superficial veins.

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#### REFERENCE

1. Miller R, Stark DCC: Acute phlebitis from nitroprusside. *ANESTHESIOLOGY* 49:372, 1978

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*In reply:*—We appreciate Professor Hunter's comments. It was our intention simply to report the incident, since no determination of the etiology was possible. The reaction observed was similar to that seen when there is a release of histamine along the line of the vein (a relatively common occurrence following the administration of meperidine). We would ascribe the reaction to this phenomenon if in fact the vein had not at the same time been swollen and, therefore, seemed to have the hallmarks of an inflammation. It was difficult to find a word other than phlebitis to describe what we had seen. We agree with Professor Hunter that powerful vasoactive drugs can act locally either as a result of transmural passage of the drug or, perhaps, by direct entry via the vasa vasorum. That he observed it in the internal saphenous vein of neurosurgical patients may indicate that hydrostatic pressure could also play a part. In our

case, the infusion was into an arm vein and there was no observable venous obstruction. The mechanism is unknown, but our own belief is that it was a direct effect of nitroprusside upon the vein wall.

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