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Letter to the Editor

Pseudomembranous colitis

Price and Davies (1977) suggest that capillary microthrombi are unlikely to be the initiating cause of pseudomembranous colitis. Their evidence appears to be based on the absence of such capillary microthrombi in the 13 cases described as 'type 1 lesion' in their material.

In a recent study undertaken in our department (Bogomoletz, 1976), the presence of capillary fibrin thrombi was demonstrated in rectal biopsies from five patients with clindamycin-associated colitis, three with pseudomembranous colitis, and two with non-specific colitis (the latter form appears to be an even earlier lesion than 'type 1 lesion').

Furthermore, capillary thrombosis with vasculitis has been described in rectal biopsies of other cases of clindamycin-associated colitis (Butsch *et al.*, 1975).

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The authors comment as follows:

We do not dispute the fact that capillary microthrombosis may be involved in the pathogenesis of pseudomembranous colitis. The point at issue is whether these thrombi initiate the disease process. We think this unlikely for the reasons discussed in the paper (Price and Davies 1977) and summarised below.

1. There were no microthrombi in the early lesions we studied. This is in agreement with the recent large series by Sumner and Tedesco (1975) and also the series of Goulston and McGovern (1965). A cause of the discrepancy might be the interpretation of the eosinophilic exudate and the dilated capillaries seen just beneath the surface epithelium.
 2. Although widespread intravascular coagulation in organs may be found in patients who have died with pseudomembranous colitis (Whitehead, 1971), the typical intestinal lesion of disseminated intravascular coagulation initiated by other causes is of focal mucosal infarction (Margaretten, 1967).
 3. Careful examination of the illustrations in works on experimental vascular occlusion and low flow states (Boley *et al.*, 1965; McKay *et al.*, 1955) also show necrotic mucosal lesions and not the diagnostic (T2) appearances of pseudomembranous colitis.
- No current hypothesis satisfactorily explains the pathogenesis of these striking lesions. A toxin which could initiate mucosal damage has been found in the

faeces of some of our cases (Larson *et al.*, in press), and this supports the concept that microthrombi play a secondary role.

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