repeatedly emphasized that the cardinal advantage of the selective technique was anatomic assurance of accomplishing a complete gastric vagotomy, this advantage was overshadowed by the great debate about diarrhea and, judging from what appears in the current literature, is now largely lost and not appreciated by the majority of surgeons. Only a minority (e.g., Sawyers, J. L., Scott, H. W., Edwards, W. H., Shull, H. J., and Lay, D. H.: Comparative studies of the clinical effects of truncal and selective vagotomy, Am. J. Surg. 115: 165, 1968) recognizes the advantage of consistently successful results of adequate vagotomy by dissecting at the gastric cardia and avoiding the anatomic pitfalls at the hiatus.

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Reply: The vagaries of the vagus

To the Editors:

Thank you for the opportunity to reply to Dr. Griffith's discussion of our paper, "Identification of vagal structures at the esophageal hiatus."

First, we reported what we actually found. The discrepancy is not entirely a matter of "semantics and failure to define the exact anatomic region in question." At the hiatus, in our material, 91 percent of anterior vagal trunks and 86 percent of posterior vagal trunks were to the right of the esophageal midline (Fig. 2). An abdominal esophagus of variable length was always present; this was taken into consideration.

We made no attempt to discuss the topographic anatomy of the vagi distal to the hiatus. Since selective vagotomy is currently so important, the authors will discuss identification of vagal structures distal to the hiatus in a future paper.

Second, we do not understand Dr. Griffith. We can only repeat that the posterior trunk, at the hiatus, was always closer to the aorta than to the esophagus.

Third, we are in error here. We cited Dr. Griffith's 1964 paper and we read, but unfortunately failed to refer to his chapter in Maingot's *Abdominal operations,* ed. 2, chap. 16. In Fig. 2, p. 240, "various levels of esophageal hiatus" are shown. Perhaps we misunderstood this figure. If so, we sincerely apologize for both the misunderstanding and the failure to refer to it. We believe we have no disagreement.

To sum up, we agree with Dr. Griffith without quarreling with Dr. Alexander-Williams. The vagaries of the vagus are many and we do not wish to muddy the waters.

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Phagocytosis

To the Editors:

The inference made by Olcay and associates' that depression of phagocytosis is "due exclusively to Kupffer cell dysfunction"[1] is tenuous. It would seem that further information on hepatic blood flow is necessary to draw this conclusion. Clearance rates and total organ uptake of materials eliminated from the blood by phagocytosis are dependent upon the function of the phagocyte, on the rate of blood flow, and on the dose of the material used.

The Kupffer cell population constitutes an estimated 70 to 90 percent of the blood-clearing phagocytic population of cells,[2] hence even small changes in blood flow through the liver may be reflected in altered clearance rates. Clearance studies under controlled conditions of blood flow can be performed, however, by applying the isolated perfused rat liver technique.[3-6]

In the reported experiments, considerable manipulation of hepatic bloodflow took place six hours preceding the actual clearance study.[1] Hence, the conclusion that the observed changes are exclusively due to Kupffer cell dysfunction is misleading in the absence

of information concerning hepatic blood flow. What evidence is there to indicate that the period of Kupffer cell dysfunction would exceed that of altered hepatic blood flow? As a consequence, some of the conclusions drawn by the authors may not be valid.

Also, the Limulus lysate test is not what it once was thought to be; it has serious limitations that deserve emphasis here,[7, 8] in view of its current popularity. It is less specific for endotoxin, since it has been found to react also with thromboplastin, nucleoproteins, and thrombin.[8]

Evidence for the enteric origin of clotting and fibrinolytic agents has previously been provided.[9, 10, 11] In addition we noted, while doing portacaval shunts in rats that the mortality rate is considerably reduced if the animals are heparinized prior to cross-clamping the portal vein, also suggesting that a thrombinlike material escapes from the intestine into the portal blood to exert its deleterious effect.[12] Whether the authors measured endotoxin activity or thrombinlike activity remains to be established, although its absence in germ-free rats would suggest that both activities may well be identical.

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Reply

To the Editors:

Dr. Gans is obviously correct in that vascular clearance of particulate materials can be influenced by hepatic blood flow, and indeed, under certain conditions, small changes in blood flow are reflected in altered clearance rates. This statement is, however, correct only when a relatively minute particulate load is employed and hepatic extraction of the colloid is 100 per cent. Under these conditions liver blood flow becomes the rate-limiting factor in colloidal clearance and the fractional clearance rate × blood volume is, therefore, a measure of sinusoidal liver blood flow.

The studies of the influence of transient portal vein occlusion on reticuloendothelial (RE) function in the rat[6] followed an extensive series of studies on the effect of total normothermic hepatic ischemia on hepatic parenchymal and RE function in the baboon. In these studies, liver blood flow was measured and correlated with changes in vascular clearance of the RE test lipid emulsion.[1] By employing differential doses of the colloid in which the dose for measuring liver blood flow was below the "critical colloid dose," it was observed that sinusoidal blood flow was decreased 50 to 60 per cent at the three-hour postocclusion interval. When phagocytic function was ascertained by employment of a particulate load which was above the "critical colloidal dose," it was noted that despite the pronounced alteration in liver blood flow, vascular clearance of the lipid emulsion was normal. Indeed, at the 24 hour postocclusion interval when phagocytic dysfunction, as reflected by an approximate sevenfold impairment in vascular clearance of the emulsion, occurred in the group which ultimately succumbed to the insult, liver blood flow was normal. These studies