

Colloid Shift Following Trauma

TO THE EDITOR: We are pleased to read the recent article on colloid shift following trauma by Briggs and Amberson (1). We have studied on the same subject over the past few years and the resulting report is in press (2). It is indeed exciting that their findings are very similar to ours. We also used visual evaluation method essentially same as theirs in assessing the colloid distribution, but only posterior views were included in the study. The areas of greatest density in each organs were visually compared. When splenic density was greater than liver we called it grade III (Colloid shift), liver density greater than spleen grade I, and when they were equal density we called it grade II. Forty-three of 73 trauma patients (59%) showed colloid shift, while 12 of 87 our control patients (14%) showed colloid shift ($p < 0.01$). The colloid shift following trauma in the young adults and children was even more frequent when they were looked at separately; 17 of 20 (85%) had colloid shift. We agree that the severity of trauma was not related with the high incidence of colloid shift since the prognosis of all our trauma patients were excellent. Truly, the mechanism of the colloid shift in trauma patients is unknown. While their suggested explanation of reticuloendothelial system stimulation is a good possibility, we were more attracted to the speculation that our patients were likely to have had alterations in their splanchnic blood flow following trauma (3).

References

1. Briggs RC, Amberson SM. Colloid shift following blunt trauma. *J Nucl Med* 1987; 28:188-190.
2. Lee HK, Kajubi SK. Colloid shift in 99mTc-sulfur colloid liver-spleen scans of trauma patients. *Clin Nucl Med*: in press.
3. Gottlieb ME, Sarfeh JI, Stratton H, et al. Hepatic perfu-

sion and splenic oxygen consumption in patients post injury. *J Trauma* 1983; 23:836-843.

H.K. Lee
S.K. Kajubi
Mount Sinai Services—City Hospital
Center at Elmhurst
Elmhurst, New York

REPLY: We thank Lee and Kajubi for their comments and are gratified to read that their observation of colloid shift after trauma is similar to ours. We look forward to their report on this subject (1).

We find their speculation that only alteration in splanchnic blood flow following trauma explains colloid shift to be unsatisfactory in light of the data we reported (2). We believe that a complex set of factors must be involved in the production of (a) colloid shift immediately after trauma and (b) colloid shift as late as six months after the traumatic event. Indeed we included changes in splanchnic arterial flow as one of the possible factors that might lead to colloid shift within 24 hr of injury. Would this mechanism explain colloid shift at 6 days or 6 mo post-trauma? We think not, and the further investigation of other possible explanations for this phenomenon seems worthwhile.

References

1. Lee HK, Kajubi SK. Colloid shift in 99m Tc-sulfur colloid liver-spleen scans of trauma patients. *Clin Nucl Med*: in press.
2. Briggs RC, Amberson SM. Colloid shift following blunt trauma. *J Nucl Med* 1987; 28:188-190.

Russell C. Briggs
Steven M. Amberson
Maine Medical Center
Portland, Maine