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Letter to the Editor

Microvascular leakage of albumin in inflammatory states

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The article by Scivoletto *et al*, *Spinal Cord* 2004; **42:** 473–476, points out the inflammatory nature of pressure sores, noting the consequent anemia and hypoalbuminemia as well as other indicators of chronic inflammation.[1] The explanation for the hypoalbuminemia offered was an inhibition of hepatic synthesis. However, the cited reference that addressed this issue showed a turnover of albumin during infectious and toxic diseases no different from that of controls.[2] Furthermore, the normally rapid elimination rate of albumin in spinal cord-injured patients is accentuated during inflammatory states, suggesting increased microvascular permeability to albumin tracers.[3,4] Even during parenteral nutrition albumin levels remain low and disappearance from the intravascular space high with sepsis.[5] Direct evidence for depressed liver synthesis of albumin during an inflammatory state such as a pressure sore is lacking, but a leak to the extravascular space is a likely factor in the hypoalbuminemia of inflammatory states.

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