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E. Cornelius Yale Medical School New Haven, Connecticut

REPLY: The term used by Cornelius, "need," is a teleologic one and has to be translated into more specific terms such as: stimulus, effector pathway, and controlling factors.

- 1. At one extreme, a marked decrement in splenic size follows daily removal of a small amount of blood from rabbits over a period of several months (1). Even if this were to reduce the splenic "need" for sequestering aging red blood cells, does it also reduce the requirement for filtering bacteria and other particulates? The situation has to be more properly explored function by function.
- 2. Despite the multitude of cases of splenectomy that we have followed, in only one did an accessory or splenotic spleen reach a "normal" size. Is the equilibration size of the organ less than normal because of inadequate stimuli, a damaged effector pathway, or factors (hormonal or otherwise) limiting its growth?
- 3. The term "need" may also mask the effects of "foreign" stimuli, such as invading microorganisms. These are not "needed" but an analysis of which of their products stimulated splenic growth or function might yield clues as to the underlying molecular mechanisms.

A rational approach to analyzing splenic growth and function is to steer away from "need" and to concentrate on the stimuli, the effector pathway and any controlling factors.

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Richard P. Spencer

University of Connecticut Health Center Farmington, Connecticut

REPLY: We appreciate Dr. Spencer's editorial (1) accompanying our article (2) and we thank Dr. Cornelius for his comments. We are pleased that so much interest for this topic exists. However, this can also mean that many uncertainties about the function of accidental or intentional autotransplantation after post-traumatic splenic rupture persist.

Accidental splenosis is usually discovered because of some complication, either acute abdominal pain (3) or following post-splenectomy sepsis (4). Spontaneous or therapeutic splenic autotransplants that do not cause any problems are overlooked. A false impression is thus created that splenic autotransplants are ineffective and unable to exert part of the immunologic and clearance function. A constant fear of potential acute abdominal complications accompanies splenosis.

It was not the principal goal of our prospective work to answer

the basic questions about the stimulus for autotransplants' growth nor to elucidate the complex mechanisms involved in immunologic mechanisms after removal of the primary organ. We followed the natural history of therapeutic transplants, their progressive growth and improved clearance function using splenic scintigraphy with heat damaged radiolabeled red blood cell. Most studies point out the two necessary factors to restore part of immunologic function after splenosis or autotransplantation, namely the appropriate vascular supply of the graft and adequate volume of the newly formed splenic tissue (5,6). It was also proven in animal experiments that the imposed workload has critical influence on transplant growth, but the ability to perform the required work depends on perfusion of the regenerates (7).

The size of individual transplants in our patients was measured from the surface area of grafts on the scintigrams. However, no attempt was made in our study to correlate the size of the transplants measured with the planar technique to their volume because of the unreliability of planar technique as compared to the tomographic measurements (8). The functional perfusion of autotransplants in our patients was good enough to maintain the clearance function as can be estimated from intensive uptake of the radiolabeled spherocytes. The increased intensity of tracer accumulation on the later scans was noted. Improved clearance function together with lack of serious infection or other complications allowed us to assume that partial restoration of immunologic function existed even with hypofunctioning heterotopic splenic tissue. On follow-up, the observed growth of autotransplant was not excessive. It was further pointed out that functioning heterotopic tissue per se does not guarantee the immunologic adequacy, but it is probably a valuable help in the host defense mechanisms as shown in human studies (6,9). Experiments with mice demonstrated better protection against aerosolized bacterial infection with at least partly preserved spleen when compared to splenectomized mice (10). It was shown that the "normal" weight of the spleen can range from 70 to 280 g in healthy white males and from 55 to 195 g in healthy white females (11). It remains unknown whether the size of the original spleen is related to the upper limit of the transplant growth. It cannot be predicted what will be the workload and its growth impact on a transplant. Additionally, correct histologic structure is probably one of the conditions for immunologic function restoration, which was not always the case in patients dying from sepsis. Autopsy studies were carried out in patients after overwhelming pneumococcal sepsis in the study of Millikan. No lymphocytes were found in the 20×20×3 mm splenic implants in an alcoholic patient with severe liver disease and severe reduction of immune competence (12). Poorly developed sinusoids were shown in other autotransplants after overwhelming postsplenectomy sepsis (OPSI). Splenotic nodules had a maximal weight of 3 g and the total weight of the splenic tissue in one of these patients reached 92 g (4). This report is in controversy with animal experiments in which the autotransplanted splenic particles undergo necrosis with consequent overgrowth of hystologically normal splenic tissue which is able to take over part of the normal splenic function (13).

We are not aware of the influence of the quantity of transplanted tissue on the regeneration, but it appears that the size of autotransplanted particles in patients with accidental splenosis is probably below the size of intentionally transplanted splenic particles in animal studies as well as in patients. Our patients did not have any complications after the transplantation and thus no histologic data were available. The relationship of histologic