Editorial . . .

Nutrition and Resistance to Infection

MOST of us at one time or another have attributed infection complicating surgery to the poor nutritional state of a particular patient. The existence of such a cause and effect relationship is possible and may occur at times, but clear cut proof is lacking; and in fact, there is considerable evidence to the contrary.

Frequently the obviously malnourished patient is free of infection as, e.g., the emaciated patient with esophageal stricture who remains ambulatory and working. On the other hand, streptococcal cellulitis, lymphangitis or lymphadenitis is commonly seen in well nourished individuals; and other types of surgical sepsis such as gas gangrene, acute appendicitis, septic thrombophlebitis, and peritonitis are almost certainly not related to the patient's nutritional state. Yet terminal sepsis is not uncommon in patients with wasting disease and here it is tempting to generalize about the effects of undernutrition.

Experimentally it has been shown that acute starvation may lower resistance of mice to several bacterial pathogens but this appears not to be the direct result of nutritional deficiency *per se*. It may be due to resulting metabolic disturbances such as decreased lactic acid production by cells in the area of inflammation or exhaustion of glycogen reserves of the body or the elaboration of excess ketones. Acute starvation undoubtedly has a widespread effect on cellular function and its infection-enhancing effect in animals may not only be the result of a change in host resistance but also due to the potentiation of growth of pathogens by changing the environment for bacterial growth.

There appears to be an association between acute starvation and infection in at least three clinical situations: 1) the severely burned patient, 2) patients with extensive multiple injury, and 3) patients with posttraumatic renal insufficiency. But in all these groups injury is the most prominent factor in initiating infection inasmuch as it breaks natural defense barriers to bacterial penetration. Acute starvation is undoubtedly present, but whether or not this potentiates infection is uncertain inasmuch as it is impossible to get control groups for study. Antibody synthesis and polymorphonuclear output and activity was not depressed in patients from the latter two groups when tested. Yet the fact that these measured indices of defense were normal does not necessarily mean that the patients' capacity to resist infection was not affected. Unfortunately most bacterial diseases in surgery do not have a known immunity mechanism, so that data on the antibody response may not be pertinent to the question under consideration.

As regards chronic starvation, animal experiments are somewhat conflicting. It seems likely that, under conditions of experimental starvation so complete and prolonged as to produce extreme wasting, resistance to infection may be impaired. But several groups of investigators have shown that gradual weight loss on a complete but restricted diet, *to an order comparable to the usual clinical situation,* does not increase animal susceptibility to infection; nor does the level of dietary protein influence resistance to infection.

There is considerable clinical evidence that chronically starved patients are not especially subject to infection. Virus infections are more apt to appear in well nourished individuals. Morbidity and mortality records from World War II show that infectious disease was not rampant except where sanitation control was neglected. Reports from the Nutrition Clinic in Birmingham have noted that many of their malnourished anemic patients were remarkably free of infection despite low serum

protein levels. A very low incidence of postoperative infection was reported among undernourished American prisoners in a Japanese prisoner of war hospital despite very inadequate means of asepsis. And, as indicated above, most of us have operated upon wasted patients in whom infection has not been a problem.

We have shown by quantitative methods that terminal nutritionally-depleted patients with hypoproteinemia and progressive weight loss can nevertheless synthesize large quantities of antibody, in fact more than healthy controls on the average. Such patients may also have an abundance of functioning circulating phagocytes.

Specific vitamin deficiencies may alter resistance to infection in laboratory animals but here too generalization may be unwise. Thus it has been reported that pantothenic acid deficiency renders certain genetic strains of fats susceptible to a diphtheria-like organism (*Corynebacterium*—strain 197) whereas other rat strains remained relatively resistant. This suggests that the simple concept that a given animal species requires a blanket minimum daily requirement of a vitamin in order to avoid infection is not the complete answer. It is of interest that both pyridoxine and pantothenic acid deficiency in rats suppressed antibody production, but only in the latter was susceptibility to the corynebacterium increased, so that under the experimental conditions reported, the ability to produce antibody had little relation to resistance to natural infection.

The relation between nutrition and infection is complex and still largely unsolved. One can't help but feel that to be free of infection a patient should be in an optimal nutritional state, but as we look at the evidence, this may not be so important, at least in the chronically starved. It is true that emaciated patients may have decubitus ulcers or bronchopenumonia, but pressure necrosis or inadequate ventilation or clearing of the tracheo-bronchial tree may be the primary underlying causes. Among factors which may promote infection in surgical patients are hyperglycemia, ketosis, medication with adrenal cortical hormones, the use of plastic catheters for prolonged intravenous feeding, extensive and prolonged surgery, poor wound management and careless aseptic technic. These or other circumstances may be the real culprits in promoting unexpected sepsis.

The basic principles of good pre- and postoperative management must be carefully implemented and often reviewed. When these are not found wanting, we are on safer ground in looking elsewhere for the cause of our complicating infection.

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BOOK REVIEW

Clinical Proctology. By J. Peerman Nesselrod, Assistant Professor of Surgery, Northwestern University Medical School. Philadelphia, W. B. Saunders Company, Second Edition, 296 pages, illustrated, 1957.

This is the Second Edition of a popular work in which a chapter on Anal Contracture and more specific treatment for Anal Pruritus has been added. Otherwise, it is essentially the same as the First Edition. The author emphasizes that the book is intended for medical students, interns, residents and general practitioners. Although this volume is not comprehensive, it is well written and should serve a useful purpose to those for whom it was designed.

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