CHANGES IN PERIPHERAL BLOOD LEUCOCYTES FOLLOWING I.V. ANAESTHESIA AND SURGERY

Sir,—Observations of leucocytosis following anaesthesia and surgery are certainly not new. Current interest is, for the most part, centred around possible depression of cellular immune response by the various anaesthetic agents on the cancer patient treated by surgery. The high incidence of apparently “anaphylactoid” or hypersensitivity-type adverse responses to the newer i.v. anaesthetic agents is also creating an interest in the mechanism of immunological recognition (or memory) to i.v. drugs in general.

It is important to differentiate the three major factors influencing leucocytosis. These are: (a) the effect of the concentration of the i.v. agent on the chemotaxis of cells, (b) stimulation by the i.v. agents of intermediary humoral factors, such as complement and prostaglandins, which cause cell migration, and (c) the role of tissue trauma. These factors are defined both by the time sequence in which they occur and by the type of leucocytes involved. Chemotactic effects occur from the point of induction and in vivo are probably restricted in time by the pharmokinetics of the drug. These are possibly of only a few minutes’ duration unless a continuous infusion procedure is being employed. This phase is well documented in vitro by Boyden chamber experiments (Moudgil et al., 1977). The trauma factor probably develops about 2 h after anaesthesia and surgery, and is characterized by a massive influx of polymorphs into the circulation, an effect which may last for several days (Ryhanen, 1977). The intermediate humoral effect, lasting about 20–60 min, is the least understood and may even be dismissed as insignificant. This is the point, however, at which immunological phenomena appear to be initiated. We have chosen to study this particular aspect and, in view of some misunderstanding of our published observations (Watkins et al., 1976), would like to summarize the situation.

White cell counts, increasing by about 25% of the pre-induction values 5–10 min after induction, are observed in approximately 60% of all patients receiving Althesin, propanidid or methohexitone for the first time. Such changes on first exposure to a drug cannot be associated with immunological recognition (memory). Our studies indicate a degree of complement C3 activation in these patients which explains also the analogous plasma histamine release curves observed by Lorenz (1975). Although we have not yet established statistical significance of the leucocyte changes with these three anaesthetic drugs, the new hypnotic, etomidate, produced highly significant changes (P < 0.001).

A second exposure to these drugs appears to induce a degree of immune recognition in at least 5% of all patients. This is characterized by more pronounced C3 activation, a marked decrease in polymorph counts and either static, or a slight increase in, lymphocytes numbers. These reactions are essentially subclinical, but a similar pattern of leucocytosis to Althesin has been observed in an animal model by our associates. Here, the second exposure to Althesin resulted not only in a dramatic decrease in peripheral polymorphs but also in severe clinical anaphylaxis. We were recently fortunate in obtaining leucocyte data in a patient who exhibited a severe anaphylactic response to thiopentone (table 1).

The data, although far from complete, indicate the almost constant behaviour of the lymphocytes in contrast to the polymorphs which decreased dramatically initially (1 h) followed, 6 h later (as the trauma response occurs), by an equally dramatic increase.

We would suggest that the difference between subclinical response and full clinical responsiveness is essentially one of magnitude. Patients showing clinical responsiveness may do so either by a direct activation of their complement system or as a result of immunological hypersensitivity. The responding patient is likely to be genetically predisposed in a specific manner, but there is little reason to believe that patients with specific atopy are at high risk with any i.v. anaesthetic agent. Finally, it is worth noting that induction of anaesthesia with inhalation anaesthetics does not appear to produce a variation in the peripheral leucocyte count.

J. WATKINS
A. M. WARD
T. N. APPLEYARD
Sheffield

REFERENCES

AYRE’S T-PIECE AND POLLUTION

Sir,—The advantages of administering anaesthesia by means of an Ayre’s T-piece (or Y-piece) in infants and small children are well recognized. However, the use of this system for controlled respiration by intermittent occlusion of the open end of the expiratory limb with a finger tip produces considerable pollution. This problem has been solved by simple modification of the system: the rather stiff polyethylene tubing used for the expiratory limb has been replaced by a length of soft silicone rubber tubing (wall thickness 1 mm). Controlled respiration is accomplished easily by intermittent compression of the expiratory limb. Exhaust gas is scavenged through a wide-bore corrugated rubber tubing (air flow 25–30 litre.min⁻¹) by introducing the end of the expiratory limb a few centimetres into the open end of the corrugated tubing.

The system seems to work satisfactorily in clinical practice. The resistance and flow requirements of the system and the efficacy of the gas scavenging are currently under investigation.

Niels Valentin
Hvidovre, Denmark