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SUPPLEMENT

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CORRESPONDENCE

Correspondents are asked to be brief

Treatment of Anaphylactic Shock

SIR,—The Therapeutic Conference by Professor A. G. Macgregor and others on treatment of drug allergy (3 April, p. 37) contains some inaccurate and misleading statements.

Death in anaphylactic shock is caused by circulatory collapse due to loss of fluid from the intravascular compartment, severe bronchospasm, and, occasionally, laryngeal oedema.[1 2] Parenteral antihistamines undoubtedly have an important place in the treatment of anaphylaxis, but Dr. R. A. Wood's assertion that in anaphylactic shock administration of chlorpheniramine on its own should rapidly improve the blood pressure and thereby ensure recovery is misleading. As Dr. Wood himself suggests, other autacoids besides histamine are involved in anaphylaxis. These include kinins,[3] slow reacting substance,[4] and possibly prostaglandins.[5] Antihistamines will obviously not antagonize the effects of these mediators. Furthermore, as Dale has pointed out[6] antihistamines are usually ineffective in antagonizing responses to locally liberated or "intrinsic" histamine. For these same reasons antihistamines are notoriously unsuccessful in relieving bronchospasm, which is a feature of anaphylactic shock and which was present in the patient described at the conference.

While it is true that glucocorticoids do not inhibit either antigen-antibody combination or release of the mediators of anaphylaxis we disagree with the Aberdeen speakers' claim that there is no theoretical basis for giving corticosteroids and that corticosteroids are useless in anaphylactic shock. Corticosteroids should work theoretically, since they reduce vascular permeability, thus correcting loss of fluid into the extravascular compartment, and since they relieve allergic bronchospasm. There is also experimental evidence of their effectiveness in anaphylactic shock[7] and we believe that intravenous corticosteroids coupled with parenteral fluid replacement may be life-saving in this situation. Adrenaline may be useful as a bronchodilator, but care must be exercised in its administration because of the risk of potentially fatal cardiac arrhythmias especially in the presence of ischaemic heart disease.

We agree that skin testing with penicillin is dangerous in patients with a history of anaphylaxis, but in their discussion Professor Macgregor and his colleagues omitted mention of the newer in vitro tests including the radio-allergosorbent test,[8] the use of passively sensitized lung tissue,[9] and histamine release from leucocytes,[10 11] which might have been valuable in establishing the diagnosis of penicillin allergy in their patient.—We are, etc.,

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Decline of the Necropsy

SIR,—Your leading article (24 April, p. 181) analyses some of the reasons for the fall from grace of this most important part of medical work.

Perhaps, however, part of the decline in importance may lie in the routine nature of the technique used, which has little if at all changed in this country over the last two decades. Part of this may lie in faulty contact between the clinician requesting and the pathologist undertaking the necropsy. A closer collaboration, as your leading article implies, would mean that confirmation or refutation of a clinical diagnosis could be established more quickly without a long wait while the routine necropsy is performed.

Equally, with regard to technique it is disappointing to see that newer postmortem rooms built have no facilities for radiology of the cadaver, a procedure that is used in some American institutions and which would contribute considerably to the facility and accuracy of necropsy studies.—I am, etc.,

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