Literature Briefs

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Literature Briefs were submitted by Drs. B. Buckmaster, P. J. Cohen, and C. J. Kopriva. Briefs appearing elsewhere in this issue are part of this column.

Hyperthermia

VASODILATATION AND HYPERTHER-MIA When hyperthermia developed in susceptible swine, metabolic rate increased precipitously. Initially there was a considerable loss of heat by radiation. However, this ceased after approximately 20 minutes as intense peripheral vasoconstriction developed. The authors conclude that vasodilators might have a therapeutic role when malignant hyperthermia occurs in man. (Williams CH, Houchins C, Shanklin MD: Energy metabolism in pigs susceptible to the fulminant hyperthermia stress syndrome. Br Med J 3: 411– 413, 1975.)

Endocrinology

β-STIMULATION AND THYROXINE The interrelationship of adrenergic stimulation and thyroid status is well known. β -stimulation in the rat is accompanied by vasodilatation, increased skin temperature in the tail, and hyperglycemia. The response to isoproterenol-induced β-stimulation was examined in rats made hypothyroid with aminotriazole. After 1-5 weeks of treatment, reduced responsiveness to isoproterenol was found. Response to β -stimulation returned to normal following treatment with thyroxine. These data lead to the conclusion that thyroxine plays a significant role in maintaining β -adrenergic responsiveness. (Fregly MJ, and others: Reduced β-adrenergic responsiveness in hypothyroid rats, Am J Physiol 229: 916-924, 1975.)

Intravenous Agents

INTRA-ARTERIAL DRUGS AND GAN-GRENE Although it is well known that intraarterial injection of certain drugs is extremely hazardous, the etiology of the resulting necrosis is still not perfectly understood. The authors have examined (by both light and electron microscopy) histologic changes produced by injection of diazepam into the artery of the rabbit pinna. Following this injection, there was immediate vasoconstriction, during which the pinna was dusky and cool. Edema then developed over the next 48 hours, and the pinna remained swollen for 10 days, at the end of which an area of necrosis appeared. Microscopie sections made a few hours after injection showed vasodilatation and vascular congestion; the vessels, however, were structurally normal. Although five days after injection there was interstitial edema with extravasation of erythrocytes, there were no thrombi in any vessel, even when gangrene was imminent. Examination with a Doppler flow detector showed no change in circulation until 7-10 days following injection. Electron microscopy showed distinct abnormalities in the endothelial cells of arterioles and capillaries; the cells were swollen and their membranes distorted, with a suggestion of breakage at some points. The authors propose that damage is not caused by acute obstruction of blood vessels with either crystals of the drug or thrombi. Rather, there is endothelial cell damage caused by high concentrations of a cytotoxic drug. (Knill RL, Evans D: Pathogenesis of gangrene following intra-arterial injection of drugs: A new hypothesis. Canad Anaesth Soc J 22: 637-646, 1975.)