upper limit may be too high, since infusion of SNP, 1.5 mg/kg, for one hour produces peak plasma cyanide level of 1.53 ± 0.16 μmol/l (mean ± SEM) and produced an increase in blood lactate of 2.38 ± 0.34 μmol/l, thus indicating some histotoxic effect.

Sodium nitroprusside, 1.5 mg/kg, would appear to be more than adequate for hypotensive anesthesia, but this recommended maximum dose does not apply to long-term infusion. On reviewing our clinical experience with SNP, also over a period of more than ten years, we have exceeded the dose of 1.5 mg/kg during hypotensive anesthesia on one occasion only, and our median total dose was less than 20 mg. This disparity with Professor Katz' experience (exceeding 1.5 mg/kg on more than 100 occasions) possibly reflects a transatlantic difference in anesthetic technique. We always use IPPV, d-tubocurarine, and supplemental anesthesia with halothane when hypotension is at all difficult to achieve. Tachycardia, when it occurs, is never allowed to persist, and we are quite prepared to abort an increase in pulse rate by means of small doses of propranolol (as much as 2.5 mg). It is of interest that Professor Katz has set an upper limit for SNP of 10 μg/kg/min for his infusions. This would result in a total dose of less than 1.5 mg/kg for most cases, and even when used for three hours (the limit to which we applied our recommended maximum dose), the infused amount would only be 1.8 mg/kg.

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Use of Propranolol to Control Refractory Ventricular Tachycardia upon Termination of Cardiopulmonary Bypass

To the Editor:—Anesthesiologists in the past have been wary of administering beta-adrenergic receptor blocking drugs during cardiac surgery for fear of difficulty in discontinuing cardiopulmonary bypass due to their depressant effect on myocardial performance.

Beta-adrenergic receptor blocking agents, however, can be most useful in the treatment of ventricular dysrhythmias as illustrated by the following case.

REPORT OF A CASE

A 20-year-old woman who had severe aortic regurgitation from rheumatic heart disease underwent elective replacement of the aortic valve. She had been taking digoxin, 0.25 mg, daily since the age of 8 years, but past medical history was otherwise unremarkable. The aortic valve was replaced with a Bjork Shiley tilting disc prosthesis during cardiopulmonary bypass at a temperature of 30°C, using coronary-artery perfusion. Upon removal of the aortic clamp at the termination of bypass, massive incompetence of the prosthetic valve was evidenced by a decrease in perfusion pressure to less than 20 torr and a massive increase in blood being returned through the left ventricular vent. The aorta was immediately cross-clamped, this time at normothermia, but now with no coronary-artery perfusion, as the cannulas had been withdrawn. The aortotomy was reopened, and it was apparent that the disc of the prosthetic valve had been jammed open by two sutures. Surgical correction necessitated aortic cross-clamping for 25 min, the heart meanwhile being cooled with ice saline solution. Upon attempting to discontinue cardiopulmonary bypass for the second time, it proved impossible to convert the heart from ventricular fibrillation to a stable sinus rhythm. Direct-current cardioversion was attempted 14 times without success. The serum potassium value at this time was 4.8 mEq/l. Two intravenous boluses of lidocaine, 100 mg, each had no effect upon cardioversion. Propranolol, 2 mg, was given intravenously, following which the heart converted instantaneously to a stable sinus rhythm with the next direct-current cardioversion. Cardiopulmonary bypass was discontinued. The postoperative course was uneventful.

We believe that the use of a beta-adrenergic receptor blocking agent may be helpful in open-heart surgery when ventricular fibrillation upon termination of cardiopulmonary bypass has failed to respond to the standard therapy. The judicious use of propranolol as an antidysrhythmic agent under these circumstances far outweighs any concern about the negative inotropic effect of the drug that may be seen with higher doses.

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Would Morphine in Large Doses Prevent Malignant Hyperthermia?

To the Editor:—The mechanisms leading to malignant hyperthermia (MH) have not yet been elucidated. MH could be related to an exaggerated endocrine–metabolic response to stress. Wingard has supported the hypothesis that MH is an acute stress syndrome of man, also occurring without any concomitant anesthetic procedure, and Williams has demonstrated an excess activity of norepinephrine in swine during MH.

Neurogenic impulses to the central nervous system are an essential part of the mechanisms that induce the endocrine–metabolic changes observed during and after surgical procedures. Blockade of these neurogenic impulses by epidural analgesia has been found to inhibit the response to surgical stress in man, and to prevent development of MH in susceptible swine exposed to halothane. Morphine (4 mg/kg), probably acting at the hypothalamic level, inhibits the intraoperative stress-induced increases in ACTH, cortisol, growth hormone, glucose, and cyclic AMP, and after administration to patients with severe burns, decreases in oxygen consumption, body temperature, and urinary catecholamine excretion were demonstrated.

Malignant hyperthermia in man can be induced by most anesthetics, but, to our knowledge, morphine has not been reported to be responsible. Since morphine apparently inhibits the normal response to stress, and assuming that MH is triggered by the same mechanisms, it may be possible to inhibit MH with morphine. Therefore, we administered morphine in large doses (4–8 mg/kg) intravenously to MH-susceptible swine, which then received halothane. But morphine neither modified the clinical symptoms of MH nor delayed its appearance. However, in this context there are two important differences between swine and man. First, even large doses (15 mg/kg) of morphine had an excitatory effect on both halothane-susceptible and non-susceptible swine. Second, MH appeared only 2–4 minutes after exposure to halothane. In man the latency period before MH appears is often much longer.

Because of the rarity of MH, we have been unable to test this hypothesis in man ourselves, but suggest that morphine in large doses should be considered in the treatment of patients with MH.

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