agents *per se* play only a minor role in the observed hormonal changes seen during surgical procedures 60 to 90 min in duration.2-4 Existing evidence would suggest that anesthesia is not an important initiator of endocrine-metabolic alterations during or after surgical stress.

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### Dose Limits to Acute Nitroprusside Therapy Challenged

*To the Editor:*—We wish to comment on a letter by Professor Katz,1 Our suggestion for a maximum dose of sodium nitroprusside (SNP) of 1.5 mg/kg2 was made on the basis of reported blood cyanide levels in cases of deliberate cyanide poisoning and SNP overdosage. We concluded from these cases that plasma cyanide levels of about 10 μmol/l could be lethal and levels below this might well prove toxic during the inevitably prolonged exposure to cyanide during SNP infusion.3 Indeed, the fact that *in vitro* the terminal respiratory enzyme cytochrome oxidase is inhibited by 50 per cent by 1.5 μmol cyanide/l makes a lower toxic plasma level very likely. It is probable that there would be a concentration gradient between plasma and tissue, and an upper limit to plasma cyanide levels of 3 μmol/l was suggested. Work in patients demonstrated a correlation between total dose of SNP and plasma cyanide levels, and extrapolation of these results indicated that 3 μmol cyanide/l plasma could result from a total dose of 1.5 mg SNP/kg for hypertensive anesthesia of short duration.2 Our work in dogs, soon to be reported, indicates that this
upper limit may be too high, since infusion of SNP, 1.5 mg/kg, for one hour produces peak plasma cyanide level of 1.58 ± 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.1,2 Beta-adrenergic receptor blocking agents, however, can be most useful in the treatment of ventricular dysrhythmias,3,4 as illustrated by the following case.

Report of a Case

A 29-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 iced 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 antiarrhythmic 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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