CORRESPONDENCE

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Propofol in the Management of Myoclonus Syndrome Induced by Chloralose Poisoning

To the Editor—Poisoning with chloralose, sold as rodenticide, is well known to produce coma with myoclonic jerks or generalized convulsions. The prognosis usually is favorable, provided that symptomatic treatment is instituted early. Diazepam may be used, generally with success, to control myoclonus.1 We report a case of chloralose poisoning in which diazepam alone failed but adding propofol succeeded in controlling myoclonic jerks.

A 70-year-old woman with a history of depression and obesity was admitted to our intensive care unit because of chloralose poisoning. Four hours after ingestion of chloralose (Chauvin Chloral, 20 g), she was discovered at home, unconscious, with generalized myoclonic contractions. After 20 mg intravenous diazepam, the trachea was intubated and the lungs were mechanically ventilated. During transport, despite a diazepam infusion (20 mg/h), myoclonic jerks appeared. At the hospital, gastric lavage was performed after additional diazepam (40 mg bolus) and continuous infusion (40 mg/h). On admission to the intensive care unit, despite a cumulative dose of diazepam over 5 h of 140 mg (1.4 mg/kg), myoclonic jerks still occurred and were increased by minor tactile stimuli. At this time, diazepam was stopped, and propofol was added to the regimen. The patient received a loading dose of 100 mg propofol (1 mg/kg), and within 1 min, no new myoclonic jerks were seen even with the same stimuli. After 10 min, however, the myoclonic contractions were observed again. A second dose of propofol was injected and followed by an infusion of 1 mg·kg⁻¹·h⁻¹. During the subsequent 5 h, no further myoclonic jerks occurred, and the propofol infusion was discontinued, after which awakening rapidly followed. The trachea was extubated 6 h after admission, and the patient was discharged from the intensive care unit after 25 h.

Propofol has been noted to have anticonvulsant properties,2 and its use in the management of refractory status epilepticus has been reported.3 In this case, propofol controlled myoclonic jerks induced by chloralose poisoning after a large dose of diazepam was unsuccessful. Propofol seems to have a more uniform depressant action on the central nervous system compared with benzodiazepines, thus explaining its efficacy in patients resistant to conventional treatment.4 Because of its unique pharmacokinetics,5 propofol may be an especially useful antmyoclonic drug with very transient effects for treatment of central nervous system excitation related to intoxication.

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Femororadial Pressure-¢ and Thermoregulation

To the Editor—Hynson et al.,1 attempted to elucidate the mechanism of the arteriolar pressure difference (A-Rd) sometimes seen at the conclusion of cardiopulmonary bypass (CPB). They measured femoral and radial arterial pressures and finger, skin, and forearm blood flows in six healthy 20–40-year-old volunteers exposed first to cold temperature (21°C) until shivering; second, to warm air (42°C) until profuse sweating; third, to cold temperature; and fourth, to anesthesia with nitrous oxide-propofol. The authors concluded that thermoregulation and anesthesia produced the post-CPB A-Rd by increasing “upper-extremity blood flow.”2

Although thermoregulation may play an indirect role in the A-Rd during anesthesia, this study in young, healthy volunteers, does not

provide the data needed to suggest that conclusion. The typical patient affected by post-CPB A-Rd is older than 52 yr with various degrees of vascular disease, has undergone coronary artery bypass grafting (CABG), and CPB, and, in most instances, has been cooled temporarily to a core temperature of 28°C. In adolescents and young adults, the peripheral (brachial, radial, femoral) arterial pulse pressure is about 50% greater than that measured in the ascending aorta, whereas in elderly subjects, the two measurements are virtually identical. Thus, the two patient populations bear little resemblance to each other.

The authors intended to measure forearm blood flow (FBF) but do not mention whether the circulation to the hand was excluded during the FBF measurement. I suspect that the authors never measured FBF properly. This is supported by their finding that propofol/nitrous oxide anesthesia increased FBF. Other investigators, well acquainted with plethysmography, find that both anesthetics decrease FBF, but that nitrous oxide increases total hand blood flow (HBF). Failure to exclude the hand circulation during the FBF measurement also would explain the large increase in FBF during sweating. Body heating produces a large increase in HBF and only a mild increase in FBF. A second potential problem concerns the authors' technique for assessment of finger blood flow (fBF) with venous occlusion plethysmography. The authors state that the fBF was measured "as previously described." In the study quoted, two requirements to accurately measure flows by venous occlusion plethysmography were ignored: the venous occluding cuff should be in close proximity to the plethysmograph (in the study referred to by Rubinstein and Sesler), the plethysmograph covered the last phalanx, and the venous tourniquet was at the base of the finger, leaving a large venous bed to be filled before blood flow could be detected; and flow should be calculated during the first few seconds after the occlusion. Burch demonstrated, using both calculations and real flow measurements, how critical it is to include only the first three pulsations after the occlusion. However, Hynson et al. compared high and slow flows as described by Rubinstein and Sesler. The latter authors traced high flows during the first 5 s after venous occlusion and slow flows after an interval of approximately 7 s after venous occlusion.

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