improperly diagnosed as junctional rhythm. During periods of P wave disappearance, ID is difficult to distinguish from nodal rhythms. Furthermore, heart rate has been noted to increase on conversion from sinus rhythm to ID.*

ID may be defined as a type of A-V nodal dissociation, whereby the S-A and A-V nodes fire at almost identical rates, without conduction across the A-V node. In ID the upright P wave will be seen to gradually merge with the QRS complex, whereas in a nodal rhythm the P wave will change its configuration or be lost within the QRS complex. As noted by Sethna *et al.*[4] "(in ID,) if the moment of dissociation is missed . . . the pattern is one of QRS complexes without visible P waves and may be misread as A-V nodal rhythm." With continuous observation, the decreasing P-R interval will be noted.

There is evidence that ID may be an extremely frequent occurrence when halogenated anesthetic agents are used. In one study of normal healthy volunteers under isocapnic enflurane anesthesia without surgery, Calverley *et al.*[5] demonstrated ID in five out of 12 subjects. No case of junctional rhythm was noted. We have noted many such occurrences at our institution while using halothane, isoflurane, and enflurane. Boba[6] has noted what appears to be ID with methoxyflurane.

The etiology of ID remains unclear, as does its treatment. Breslow *et al.*[1] suggest that light anesthesia may be an implicating factor in the arrhythmia that they report. However, Laver and Turndorf[7] noted that increasing the halothane concentration from 0.3 to 1.0% leads to the development of this arrhythmia. Also, as noted above, this disturbance has been seen both in the presence and absence of surgical stimulation.

Finally, we were surprised at the suggestion of the authors that a switch from halothane to enflurane during surgery would have restored normal sinus rhythm in light of the report of Calverley *et al.*[5]

An increased awareness of the occurrence of ID during inhalational anesthesia and continuous monitoring of ECG may lead to the correct diagnosis of this common, but often misdiagnosed, rhythm disturbance.

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Action of Verapamil at the Neuromuscular Junction: Prejunctional or Postjunctional?

To the Editor:—I read with great interest, in the Correspondence section of ANESTHESIOLOGY, the controversy concerning the site of action of verapamil at the neuromuscular junction.[1,2] Durant *et al.* showed in their original report that verapamil potentiates the neuromuscular block of pancuronium and succinylcholine and state that this effect is not centered on the muscle fiber itself.[3] Foldes, however, took issue with this con-

clusion and suggested that there is considerable evidence indicating that verapamil acts primarily at the sarcolemma or the sarcoplasmic membrane.*

The controversy may be solved by one's noting the different responses to indirect nerve stimulation and direct muscle stimulation in curarized *versus* noncurarized nerve–muscle preparations. With the use of the isolated phrenic nerve–diaphragm preparation, Baraka[4] showed the following: 1) In a noncurarized preparation, both indirect nerve stimulation or direct muscle stimulation could result in a maximal twitch response when a supramaximal stimulus of 0.1–0.2 ms was used. 2) The addition of *d*-tubocurarine to the perfusion both could block completely the twitch response, whether that stimulus was applied to the nerve or directly to the muscle. 3) Following neuromuscular block, the twitch reponse to direct muscle stimulation could be completely restored by increasing the duration of the stimulus up to 1–2 ms. On the other hand, the response to nerve stimulation remained blocked, despite the increased duration of the stimulus.

It was concluded that in noncurarized nerve–muscle preparations the response to a supramaximal stimulus applied directly to the muscle may still remain indirect, resulting from the stimulation of the highly excitable nerve terminals located within the muscle. A direct muscle response can only be ensured after complete neuromuscular blockade and after increasing the duration of the stimulus about 10-fold. These criteria have been satisfied by Durant *et al.* in their additional study in the rabbit.[2] They eliminated neuromuscular transmission with a large dose of vecuronium and stimulated the muscle directly with the use of stimuli of 1 ms duration. Under these conditions, verapamil in the dose range of 0.01–1 mg/kg had no effect whatsoever on the directly elicited twitch tension. Bikhazi *et al.** used for muscle stimulation pulses of 0.2 ms duration, which may result predominantly in an indirect rather than a direct muscle response.

Interpretation of these data suggests that verapamil in the doses used does not act directly on the muscle but on the neuromuscular junction, which includes the nerve terminal, cholinoreceptors, or ionophores of the postjunctional membrane.[2] Higher doses of verapamil or other calcium channel blockade may produce an additional postjunctional effect on the muscle fiber itself. With the use of a muscle biopsy taken from a patient in whom malignant hyperthermia (MH) was developing, it has been shown that the calcium channel blocker, diltiazem hydrochloride, can both prevent and reverse the abnormal contractures produced *in vitro* by caffeine, halothane, or halothane plus caffeine.[5]

Calcium plays a fundamental role during neuromuscular transmission and subsequent muscle contraction; it acts both prejunctional at the nerve terminals affecting acetylcholine release and postjunctional at muscle coupling excitation–contraction. Ca^{++} channel blockers inhibit the normal Ca^{++} influx into cells.[6] It is therefore possible that verapamil and other calcium channel blockers can depress neuromuscular transmission[7,8] by both prejunctional and postjunctional mechanisms. The skeletal muscles have a large intracellular store of Ca^{++} and are therefore less dependent on Ca^{++} influx than the cardiac and smooth muscles, which contain relatively small amounts of endoplasmic Ca^{++}.[9–11] That is why the cardiac and smooth muscles are more sensitive than the skeletal muscles to Ca^{++} channel blockers. However, when the safety margin of neuromuscular transmission is impaired by neuromuscular blocking drugs,[3,*] the prejunctional effects of verapamil manifest and potentiate the neuromuscular block. In contrast, the direct effect of verapamil or other Ca^{++} channel blockers on the muscle might show up when used in patients having muscular disorders such as malignant hyperthermia[5] or muscular dystrophy.[12] Although verapamil is regarded as a specific calcium channel blocker, one has to keep in mind that it has a spectrum of activity including a high local anesthetic potency (1.6 times that of procaine).[6] This can affect not only "slow," but also "fast" channels and hence may contribute to its prejunctional and postjunctional effects at the neuromuscular junction.

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Computerized Anesthesia Records May Have Drawbacks

To the Editor:—To date no pursuit in anesthesiology technology has claimed more and delivered less than the search for a "computerized anesthesia record." Such again is the case in the recent letter to the editor by Rosen and Rosenzweig[1] that reports "on a system that uses proprietary software to generate an anesthesia record . . . used is the Radio Shack® Model 100, which is lightweight and portable."

The letter is often misleading and minimizes or fails to disclose drawbacks in the system as structured. It is stated that "vital signs may be entered manually or automatically through the RS-232 interface." In truth, manual entry would require multiple repetitive key strokes, a tedious and time-consuming process. No simple solution is provided either by the RS-232 interface. This is solely a mechanical design standard dealing with connector architecture and does not deal with the manner in which information is sent, received, and acknowledged. This requires special communications software. It's not enough that the plugs match!

Just this problem, data communication between monitoring equipment, has been the subject of a whole proposed standards writing effort with the Association for the Advancement of Medical Instrumentation (AAMI). The proposal was an outgrowth of a 1982 AAMI roundtable discussion that identified specifically the problem of interfacing equipment from various manufacturers. It was the consensus that entirely too much time was being taken up with software and hardware efforts to reinvent the interfacing solution while more important aspects of monitoring were not being addressed. Ultimately, the effort was tabled because of shifting priorities within AAMI and the sheer magnitude of the project itself.

The authors also did not address the issue of the time required to print the representative anesthesia record they displayed in the letter to the editor. Anyone who has watched low-cost plotters chug away knows that considerable time is required to generate the sample records depicted. The hardware and software aspects aside, the authors claim that it provides a more legible and accurate anesthesia record. This is a contention that I wholly reject. While the clarity of the characters may be improved by mechanical penmanship, the information is no more accurate or precise as to time or value than the key stroke or transducer that provided the signal. To use the old computer adage, "Garbage in equals garbage out," only this time the garbage is bagged. The authors state that "entries can be made in any order at any time before, during, or after the case." How then can random entries contribute to greater accuracy and precision in recording physiologic and pharmacologic data generated during the case?

The inference that somehow or other by using this magic box a successful defense is mounted to malpractice litigation is completely unsubstantiated. A sloppy anesthesia record may help lead the jury to the presumption of a sloppy anesthetic administration, but artful depiction of an otherwise poor anesthetic administration will not prevent malpractice judgments.

Finally, it should be noted that the Center for Medical Devices and Radiologic Health of the Food and Drug Administration considers software written for devices with microprocessors that interface with medical instruments to be classified as a medical device itself.* Assuming this to be a class II medical device, was premarket notification of the Food and Drug Administration made under regulation 510(k) of the Federal Food, Drug and Cosmetic Act?

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