improperly diagnosed as junctional rhythm. During periods of P wave disappearance, 1D is difficult to distinguish from nodal rhythms. Furthermore, heart rate has been noted to increase on conversion from sinus rhythm to 1D.*

1D 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 1D 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.1 “(in 1D,) 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 1D may be an extremely frequent occurrence when halogenated anesthetic agents are used. In one study of normal healthy volunteers under isoflurane anesthesia without surgery, Calverley et al.5 demonstrated 1D 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 enfurane. Boba6 has noted what appears to be 1D with methoxyflurane.

The etiology of 1D 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 Turndorf7 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 enfurane 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 1D 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 Postjunctonal?

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-

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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 et al. 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 bath 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 response 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. 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. Higher doses of verapamil or other calcium channel blockers 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. Ca2+ channel blockers inhibit the normal Ca2+ influx into cells.6 It is therefore possible that verapamil and other calcium channel blockers can depress neuromuscular transmission7,8 by both prejunctional and postjunctional mechanisms. The skeletal muscles have a large intracellular store of Ca2+ and are therefore less dependent on Ca2+ influx than the cardiac and smooth muscles, which contain relatively small amounts of endoplasmic Ca2+.9–11 That is why the cardiac and smooth muscles are more sensitive than the skeletal muscles to Ca2+ channel blockers. However, when the safety margin of neuromuscular transmission is impaired by neuromuscular blocking drugs,3,9 the prejunctional effects of verapamil manifest and potentiate the neuromuscular block. In contrast, the direct effect of verapamil or other Ca2+ channel blockers on the muscle might show up when used in patients having muscular disorders such as malignant hyperthermia5 or muscular dystrophy.10 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 Rosenzweig1 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 moni-
toring 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 hard-
ware 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 infor-
mation 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
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 anes-
thesia 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 instru-
ments to be classified as a medical device itself.1* 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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