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Insurance Incentives and the Use of Monitoring Devices

To the Editor:—Coté *et al.*[1] are convinced that the use of oximeters will save lives by facilitating early recognition of hypoxemia. They speculate that if insurance companies provided incentives such as reduced malpractice insurance costs, more individuals would use these monitoring devices.

The Joint Underwriting Association (JUA), which is the insurance consortium in Massachusetts, has already introduced such incentives. In February, 1986, Dr. Ellison Pierce suggested to the Executive Committee of the Massachusetts Society of Anesthesiologists (MSA) that if anesthesiologists in Massachusetts instituted a firm risk management program, the JUA might place MSA members in a lower risk category and thereby reduce premium costs. Dr. Joseph Beauregard, at that time President of the MSA, and Mr. Edward Brennan, Counsel to the MSA, conceived the idea of linking the verifiable use of certain monitoring instruments with a premium discount. They negotiated with the officials of the JUA and, in January, 1987, the MSA and the JUA agreed to such a discount that was then approved by the Division of Insurance (DOI) of the Commonwealth of Massachusetts. This was codified in a document entitled "Stipulation Regarding Discounts For Anesthesiologists Who Participate In Risk Management Activities." The Stipulation is 21 pages long, but its essence is found in the first paragraph: "A discount of 20% shall be granted to any anesthesiologist who certifies to the JUA that he or she shall have access to and shall use both a pulse oximeter and in all cases where physically possible an end-tidal CO_2 analyzer (capnograph) in all circumstances where their use is recommended in the Standards for Basic Intraoperative Monitoring adopted by the American Society of Anesthesiologists (ASA) on October 2, 1986 . . ." The Stipulation then makes a number of exceptions for unexpected equipment failure, emergencies, impracticalities, *e.g.*, burned patients, and in routine obstetric practice. There are also premium penalties for failure to use the devices after agreeing to do so.

The key evidence that persuaded the JUA and the DOI was the preliminary finding of the Closed Claim Study being conducted by the Professional Liability Committee of the American Society of Anesthesiologists.[2] At that time, an analysis of the first 381 claims resulting from anesthetic death or major neurological injury showed that, in the opinion of the reviewers, 113 (30%) could have been prevented if one or both devices had been used, had they been available at the time.

As part of this Closed Claims Study, the Ad Hoc Committee on Closed Claims of the MSA reviewed 151 claims that were closed from the founding of the JUA in 1975 to the end of 1984. Of the 45 cases with Severity of Injury (SOI) classification 7, 8, and 9 (major permanent neurological injury or death), 25 (56%) could have been prevented if a pulse oximeter had been in use.

One can debate the merits or otherwise of using financial incentives to influence professional behavior, but the findings of a recently completed appraisal by the JUA of the current use of the two devices are striking. Of the 78 anesthesia services in Massachusetts that the JUA insures, 70 are in full compliance and the remainder in partial compliance. There are 275 anesthesiologists practicing in Massachusetts who are insured by the JUA and, of these, 241 have been approved for the discount. Since the Stipulation only went into effect on July 1, 1987, this indicates a remarkably rapid rate of acquisition and introduction into everyday use of the two monitors. Of course, this might well have occurred even without the discount. Studies by industrial psychologists suggest that, at the professional level, control and achievement are at least as important as money in providing incentives.[3]

Only continued study will demonstrate whether or not the use of these devices will reduce the incidence of major anesthetic mishaps. The MSA and the JUA plan to do so.

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Urinary Retention following Spinal Opiates

To the Editor:—The editorial by Dray on epidural opiates and urinary retention highlights at least three issues of clinical importance.[1]

First, the editorial suggests that certain opiates produce lower incidence of urinary problems after spinal use compared with morphine. The tenet of oral and parenteral opiate studies is that side-effect incidence must be compared to equi-analgesic dosage. We would argue that until this is obeyed for spinal opiates such a conclusion is premature. Clinical trial design is complex for spinal opiates, particularly for

extradural dosing. The literature is replete with single-drug studies without controls for the parenteral uptake from that site, and with comparisons of drugs that omit sensitivity measures, such as using at least two doses of one of the drugs to see if a measurable difference results. Until these issues are addressed, side-effect incidence comparisons are misleading.

Second, the editorial suggests that use of receptor selective opiates offers the opportunity to maximize pain relief and minimize side effects. We would agree with this in principle. In practice, however, the drug doses used produce concentrations in cerebrospinal fluid (csf) which are very high, 50000 nmol/l in lumbar csf 30 min after lumbar injection of 2.5 mg morphine.[2] It may be that such high concentrations are necessary because drug is bound non-specifically, but at first glance arguing for receptor-selectivity at such levels is unrealistic.

Third, the author suggests that kappa ligands do not produce urinary problems. The difficulty with this argument is that intrathecal injection of kappa ligands may also fail to produce analgesia. In animal behavioral studies, spinal kappa ligands seem to be poor analgesics, particularly in tail-flick studies, although supraspinal analgesic effects can be observed.[3] In electrophysiological tests of analgesic effect, the net result of intrathecal kappa ligands is that they are not analgesic.[4] Until there is convincing evidence that spinal application of kappa ligands does indeed produce clinically relevant analgesia, it is premature to suggest that recourse should be made to kappa-selective drugs to minimize urinary difficulties.

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In Reply:—Drs. McQuay and Moore's comments are pertinent, but need to be balanced by the following observations. Certainly few clinical studies have critically examined the relationship between different doses of spinal opiates, the intensity of analgesia, and the occurrence of side effects. Comprehensive studies of this kind would be welcome. However, where comparisons between different drugs have been made using equi-analgesic doses, clear differences have been observed in the incidence of urinary retention.[2] This observation suggests the possibility that the mechanisms of opiate-induced analgesia and impaired urinary bladder activity are different. Several other factors may also account for apparent differences between the activities of opiates. These include differences in their pharmacokinetic properties and in their intrinsic pharmacological activity. The relationship between these factors and the relief of pain should be interpreted cautiously. In the example given, it is difficult to weight the significance of the high c.s.f. concentration of morphine, measured 30 min after spinal administration, with analgesia, because adequate pain relief is produced for many hours following a single dose of morphine. During this time, substantial redistribution and clearance of drug occurs. Indeed, a more relevant measurement would be the concentration of morphine at the active site within the spinal cord. This is likely to be a fraction of that present in the c.s.f. Finally, the specificity and receptor selectivity of spinal morphine can only be defined using a selective antagonist, such as naloxone. This has been shown in a number of studies to reverse the effects of epidural morphine.[1,3]

Finally, the efficacy of spinal kappa ligands as analgesics is indeed questionable from animal studies in which noxious somatic stimulation has been tested. Other studies, however, support the efficacy of kappa ligands against visceral nociception.[4] Clearly, such differences observed in experimental animals require further study, but these findings also invite future clinical evaluation.

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