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Vive Pascal but Kilo Newton?

To the Editor:—Regarding de Jong's note[1] helping to enlighten readers about SI, the atmospheric pressure is not about 100 N/m^2, but about 100 kN/m^2 (101,325 N/m^2 is the standard atmosphere). Newton "kil"ed by a typo—even the teachers get confused! Perhaps de Jong was suggesting that Newton was worth 1,000 Pascals, an Anglic counterbalance to the Gallic assault on that little millimeter of mercury. Perhaps he was.

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REFERENCE

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Mechanism of Epidural Lidocaine Reversal of Tachyphylaxis to Epidural Morphine Analgesia

To the Editor:—Recently, McCoy and Miller reported in a single patient the efficacy of epidural lidocaine injections in reversing the analgesic tachyphylaxis associated with epidural morphine.[1] My response is to not discourage or object to this practice, since epidural local anesthetic injection can provide both temporary relief and prognostic information concerning the pain syndrome in question.[2] Rather, my primary interest in this report is twofold. First, I wish to report my inability to consistently reverse morphine tachyphylaxis with epidural local anesthetics. In this regard, a representative case is instructive. In February 1981, a 60-year-old man with squamous cell carcinoma of the lung and associated brachial plexus invasion was treated with thoracic epidural morphine injections after increasing doses of methadone (up to 40 mg/day) and hydromorphone HCl (up to 24 mg/day) failed to control his pain. Initially 6 mg of epidural morphine produced 12 h of nearly complete pain relief. Subsequently, a subcutaneous reservoir of the Ommaya® type was placed on the chest wall in series with a silastic thoracic epidural catheter to facilitate epidural narcotic delivery. During the next two weeks, progressive dosage increases were required, while the frequency of injection increased from once to twice daily. Injection of epidural local anesthetic (either 8 ml 1 1/2% lidocaine or 1/4% bupivicaine at T6) produced complete relief of pain, but this effect was sustained for only 2–3 h, and the tachyphylaxis to epidural morphine was not affected. More disconcertingly, attempts to control pain with parenteral morphine resulted in rapid tolerance to even massive doses (up to 175 mg/h). He died after 1 month, at which time he was receiving 100 mg of morphine as an iv bolus every 2–3 h in addition to the continuous iv infusion. Seemingly, this patient was already significantly tolerant to opiates (prior to epidural morphine) at least at supraspinal opiate receptor sites. Aggressive opiate activation of spinal cord opiate receptors (spinal receptors were initially responsive, since epidural morphine analgesia initially was achieved) then occurred consequent to bolus epidural morphine thus, apparently leading to generalized opiate receptor indifference to even astronomic doses of parenteral morphine. In this regard, the case is similar to that described by Woods and Cohen.[3] In selecting intraspinal narcotic therapy, one thus assumes that tolerance at the spinal cord opiate receptor is less pronounced than at supraspinal receptors, in spite of substantial conventional narcotic exposure. Thus the "multiplicative" antinociceptive interaction resulting from both spinal and supraspinal opiate receptor activation (see Yeung and Rudy[4]) may be lost more completely than if only one opiate receptor site has been activated substantially and continuously. This may explain a related phenomenon; namely, spinal opiate tolerance appears to occur more rapidly during continuous intrathecal morphine use as opposed to chronic epidural administration. Alternately, since different opiate receptors may predominate at the spinal cord level (for example the delta opiate receptor subtype as proposed by Pasternak), agonists with lesser affinity at this receptor may become more effective with direct intraspinal application.

Secondly, I am interested as to the mechanism re-