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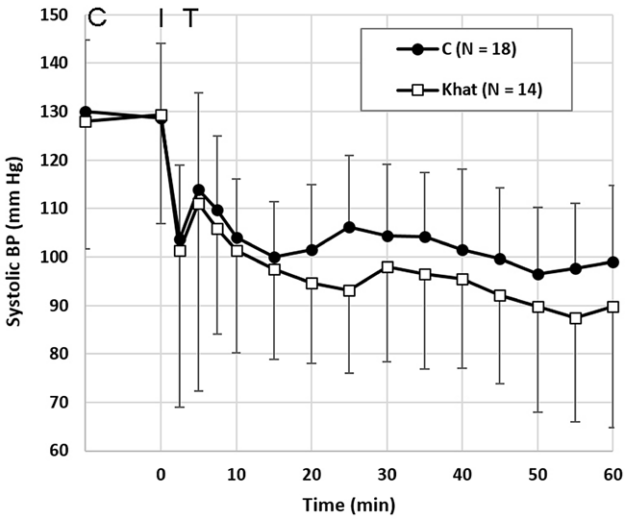

Figure. Systolic BP (mean ± standard deviation) was significantly lower in khat users (Mann-Whitney *U* test: $P < .02$), despite a similar consumption of isoflurane and a decreased administration of opioids. BP indicates blood pressure; C, control (BP at arrival in operating room); I, BP just before induction; T, tracheal intubation.

Chronic Amphetamine Users Do Not Need More Drugs During General Anesthesia

To the Editor

Studies of anesthesia in amphetamine users are scarce,[1] and the retrospective study by Kram et al[2] reporting that use of cocaine or amphetamines was not predictive of an increased need for analgesia and sedation in trauma patients is of particular interest.

A few years ago, when I was an anesthetist in Djibouti, I was warned that anesthesia in khat chewers required higher doses of narcotics and hypnotics. Khat (*Catha edulis*) leaves are legally chewed by the inhabitants of the African Horn and Arabia. They contain amphetamine alkaloids (cathine and cathinone) that cause adrenergic stimulation responsible for positive inotropic and vasoconstrictive effects. This chronic amphetamine abuse is a significant problem not only in these parts of the world where studies are infrequently performed but also in Western countries where similar drugs (ecstasy, methcathinone, or ephedrone)[3] are consumed. Moreover, "Westerners" are more and more frequently taking care of immigrants from places where khat is legal but abused (like alcohol) and are unfamiliar with khat as a source of amphetamine.

There is a popular perception that patients who chronically ingest amphetamine or amphetamine-like drugs will require high doses of anesthetic drugs to achieve adequate analgesia, sedation, and anesthesia.[4] In a work approved by the local ethnic committee of the French military hospital in Djibouti, I observed that the interaction between khat and general anesthesia was not what was commonly believed.

The preanesthetic consultation determined whether 32 consecutive patients undergoing general anesthesia were drug addicted (khat group, N = 14) or not (controls, N = 18). Induction consisted of alfentanil 0.02 mg/kg, propofol 2.5 mg/kg, and vecuronium. After endotracheal intubation, anesthesia was maintained with a mixture of oxygen (50%), nitrous oxide (50%), and isoflurane. Isoflurane end-tidal concentration was adjusted to maintain blood pressure (BP) at ±30% of its control value and alfentanil boluses to maintain heart rate at ±30% of the control value.

Biometric data, type of surgery, and induction doses were similar in both groups. During surgery, khat chewers consumed less alfentanil than controls: 1.5 ± 0.2 vs 2.1 ± 0.3 mg ($P < .05$). Although isoflurane consumption (mean end-tidal values) was not different ($P = .09$), BP was lower in the khat group (Figure; $P < .02$). Heart rate, ephedrine use, and time between extubation and first request for analgesics (145 ± 275 [khat] vs 65 ± 44 min [controls]) were not significantly different.

The lower BP in khat chewers, despite similar end-tidal concentrations of isoflurane and lower doses of alfentanil, may be related to a depletion of catecholamine receptor storage, a mechanism that has been suspected in chronic amphetamine abuse.[1] Furthermore, Johnston et al[5] demonstrated that amphetamine decreased the minimum alveolar concentration of halothane in dogs 21%.

In conclusion, acute and chronic amphetamine abuse must be distinguished. The chronic amphetamine users described here, who had not ingested their usual khat dose the day before surgery, did not need increased drug doses of either isoflurane or opioids. These findings are consistent with those of Kram et al.[2]

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High End-Tidal Oxygen Concentration Can Be a Misleading Sole Indicator of the Completeness of Preoxygenation

To the Editor

The Nimmagadda et al[1] review article and the accompanying editorial by Slinger[2] on preoxygenation bring valuable attention to this important potentially lifesaving procedure. However, both Nimmagadda et al[1] and Slinger[2] make the potentially dangerous clinical practice mistake of stating that end-tidal oxygen concentration (Eto_2) >90% indicates "maximal" and "adequate," respectively, preoxygenation. Both poor facemask seal and low-tidal volume ventilation can result in a patient having a high Eto_2 value yet be poorly preoxygenated.

If one simply flows oxygen through an anesthesia circle system at 5 L/min into a room, the Eto_2 monitor will display 98% to 100%. If the anesthesia circle system is then connected to the patient using a facemask, and there is no (zero) ventilation, then the Eto_2 monitor will continue to display 98% to 100%. If the facemask seal is unsatisfactory and there is ventilation by the patient, the sampling line can continue to mainly detect the fresh O_2 inflow and not the expired gas and the Eto_2 value will remain misleadingly high and the patient's alveolar space poorly preoxygenated. If the facemask seal is satisfactory and the patient inspires a tidal volume that is only slightly greater than the facemask and anatomical dead space (low ratio of alveolar ventilation to functional residual capacity), then the Eto_2 value will decrease a small amount during the subsequent exhalation, but the absolute value of the Eto_2 will remain misleadingly high. Then, in the presence of continuing the same hypoventilation, the Eto_2 value will very slowly and exponentially rise from the already high value that occurred after the first small breath to higher values. The early high Eto_2 level occurs because much of the exhaled gas is inspired gas residing in the anesthesia facemask and anatomical dead space and only a relatively small amount of the exhaled gas comes from the alveolar space; again, the patient's alveolar space remains poorly preoxygenated.

When using Eto_2 values as an indicator of adequate preoxygenation, each exhalation should result in a positive capnograph and the reservoir bag should expand and contract with each exhalation and inhalation, respectively. These additional findings will confirm that the Eto_2 values represent alveolar gas rather than fresh O_2 inflow or dead space gas.

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In Response

End-Tidal Oxygen Is a Reliable Indicator for Maximal Preoxygenation

We thank Drs Benumof and Herway for their comments and acknowledge Dr Benumof's many contributions to the science and practice of preoxygenation. They are correct in stating that both a poor mask seal and a low-tidal volume ventilation can result in a falsely high end-tidal oxygen (Eto_2) value despite poor preoxygenation. However, these situations should not occur if: (1) the Eto_2 measurements are interpreted accurately; and (2) the preoxygenation technique is performed correctly. A fundamental principle in interpreting the end-tidal gas measurements is that the tidal volume is sufficient to displace the alveolar dead space. Obviously, if the tidal volume is too small or the patient is apneic, the monitor will display erroneous values. The importance of a sealed system during preoxygenation has been thoroughly addressed in research reports, editorials, and book chapters.[1,2] Clinical evidence for a sealed system is an adequate movement of the reservoir bag during inspiration and expiration and a normal capnographic tracing that permits measurement of inspired and end-tidal carbon dioxide ($EtCo_2$).[1,2] As stated in our review, "the technique should be performed correctly ..." and "the absence of a normal capnographic tracing and a lower than expected $EtCo_2$ and Eto_2 should alert the anesthesiologist to the presence of leaks in the anesthetic circuit."[3] Providing that the Eto_2 measurements are accurately interpreted and preoxygenation is properly performed, an Eto_2 value ≥90% is a reliable noninvasive indicator of its efficacy.

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