Letters to the Editor of Clinical Workshop

Anesthetic Considerations in Patients on LSD-25

To the Editor: The growing use of LSD-25, lysergic acid diethylamide, is receiving wide publicity. Although its hallucinogenic properties have been described vividly, and the public has become alarmed about its acute and chronic ill effects, few know that some physicians are using it to alleviate the mental anguish in terminal cancer patients to the extent of obtunding consciousness. Some anesthesiologists [1] have found that LSD-25 caused a marked potentiation of the analgesic effects of meperidine and dihydromorphinone and also that it possesses some analgesic effect. The potentiation of analgesia by LSD-25 is not surprising, since this compound was found to have marked anticholinesterase activity [2] and anticholinesterase agents have long been known to potentiate analgesic drugs.

The widespread use of LSD-25 in addicts and by physicians emphasizes the need for anesthesiologists to be cognizant of the possible adverse influence of the compound on the course of anesthesia. One should know that the anticholinesterase activity of LSD-25 is approximately one-tenth that of hexafluorenium (Mylaxen) [3] used for the potentiation of the muscle relaxant effect of succinylcholine. Consequently, some prolongation of apnea and neuromuscular block from succinylcholine may be expected in chronic LSD-25 users. Since the ester-types of local anesthetics are detoxified primarily by plasma cholinesterase, a similar potentiation of local anesthetic toxicity may result from LSD-25. These complications are more likely to occur in cancer patients who may also be receiving anti-cancer drugs [4] and narcotic-analgesics with anticholinesterase effects.[5] In the differential diagnosis of prolonged apnea, therefore, an additional iatrogenic factor, LSD-25, should be taken into consideration.

Furthermore, LSD-25 was shown to cause inhibition of monoamine oxidase (MAO).[6] Therefore, an augmentation of the effects of sympathomimetic amines may occur, resulting in hypertension and hyperpyrexia. Since serotonin and, to some extent, histamine are metabolized by MAO, a potentiation of these endogenous substances may be induced by LSD-25, leading to hypotension and bronchospasm.

The disturbances of autonomic nervous system activity may further complicate anesthetic and postanesthetic management. Hyperthermia may lead to hyperpyrexia during prolonged anesthesia. Pupillary dilatation may obscure the eye signs of anesthetic depth. Bronchospasm, hypotension and bradycardia, especially after the combination of a rapidly administered sequence of thiopental, halothane, and succinylcholine, may precipitate cardiac arrythmias and/or arrest in these patients. Motor irritability and hallucinations may also present a problem before induction of anesthesia and in the recovery phase. I realize, of course, that clinical experience not yet available is needed to assess the validity of these assumptions.

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