

Cerebrospinal-fluid Pressure during Dissociative Anesthesia with Ketamine

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Since its introduction into clinical anesthesia, ketamine has been especially recommended when an endotracheal set-up would encumber the anticipated procedure. The lack of cardiovascular depression seen with this agent has also made it particularly suitable for procedures in which the patient's position could result in postural hypotension. It has, therefore, found clinical usefulness in neuro-diagnostic roentgenology.¹⁻³

Because ketamine increases blood pressure, heart rate, and cardiac output, it seemed possible that these hemodynamic changes could also have an effect on cerebral blood flow and CSF pressure, but its effect on CSF pressure had not been thoroughly investigated. This stimulated our investigation of the effect of ketamine on CSF pressure.

METHODS

Patients selected for this study were all physical status I male volunteers between the ages of 18 and 43 scheduled to undergo simple surgical procedures (herniorrhaphy and peripheral orthopedic operations) under spinal anesthesia. Informed consent was obtained during the preanesthetic visit. The patients were brought to the anesthetic induction room, unmedicated, approximately an hour before the scheduled surgical operations. An 18-gauge teflon catheter was placed in one radial artery after ascertaining that an ulnar pulse was present in that wrist. A large-bore intravenous catheter was then placed and an infusion of 5 per cent dextrose in Ringer's lactate solution was begun.

With the patient in the right lateral decubitus position, using sterile technique, a 3-inch

22-gauge needle was introduced through the L3-4 or L4-5 interspace into the subarachnoid space through a procaine skin wheal. Free flow of CSF was determined, care being taken not to lose any fluid. The subarachnoid needle and arterial catheter were connected to Stat-ham pressure transducers (P23D); these pressures were recorded directly with a Hewlett-Packard heat-writing oscillograph (model 7700). Blood gases were determined with modified Clark and Severinghaus electrodes (Instrumentation Laboratories). After baseline recordings and blood gases were obtained, a standard dose of ketamine (2 mg/kg) was administered intravenously; the patient was allowed to breathe spontaneously. Blood gases were obtained when the pressure recordings reached a plateau (usually within two minutes of the injection) and when said recordings had returned almost to baseline. The patient was then given a subarachnoid block for the operation, following which he was given diazepam intravenously to minimize the unpleasant effects which often occur with ketamine.

RESULTS

Figure 1 shows a record of the blood pressure and CSF pressure responses during an injection of ketamine. Table 1 lists the changes in CSF pressure, arterial P_{CO_2} , and arterial blood pressure seen after ketamine. Statistical analysis was made with Student's paired *t* test. CSF pressure and blood pressure changes were consistent in all patients studied; arterial P_{CO_2} values increased in eight patients and decreased in two.

DISCUSSION

Ketamine has been recommended over agents previously used in the management of patients undergoing neuro-diagnostic procedures.¹ Its advocates maintain that lack of cardiovascular

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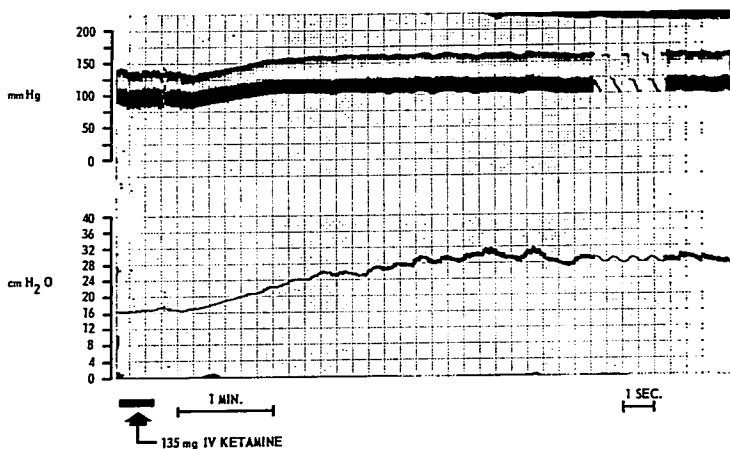


FIG. 1. Blood pressure (upper record) and CSF pressure (lower record) responses to the intravenous administration of ketamine.

depression, maintenance of an adequate airway, and preservation of protective reflexes are definite advantages of this agent^{1, 4, 5} Though many patients given ketamine for neuro-diagnostic procedures undoubtedly have increased intracranial pressure, little has been

said concerning its effect on CSF pressure. Since arterial pressure, heart rate, and cardiac output increase,^{4, 6, 7} while total peripheral resistance either decreases^{7, 8} or remains unchanged,^{4, 7} some organs must have an increased blood flow. If the brain were such an

TABLE 1. Effects of Ketamine on P_{aCO_2} , Blood Pressure, and CSF Pressure

	P_{aCO_2} (torr)		Mean Blood Pressure (mm Hg)		CSF Pressure (mm H ₂ O)	
	Control	Ketamine	Control	Ketamine	Control	Ketamine
Patient 1	31	30	95	135	150	360
Patient 1	31	36	81	86	135	230
Patient 3	23	25	75	91	144	392
Patient 4	34	35	87	107	136	476
Patient 5	38	35	93	101	144	400
Patient 6	31	37	96	139	192	490
Patient 7	34	39	104	160	150	450
Patient 8	34	39	95	150	200	633
Patient 9	38	41	87	102	160	453
Patient 10	31	32	100	120	152	304
Patient 11	—	—	101	135	160	420
MEAN CHANGE ± SE	2.4 ± 0.93 <i>P</i> < 0.05		28.36 ± 5.47 <i>P</i> < 0.001		253.18 ± 30.38 <i>P</i> < 0.001	

organ, a rise in CSF pressure might be expected. There are three separate compartments (blood, CSF, and brain) within the rigid skull; an increase in the size of any one of these is compensated for by a decrease in the sizes of the others. When compensation can no longer occur, the intracranial pressure will rise. Therefore, when an increase in blood flow occurs rapidly, there may be insufficient time for compensation, and intracranial pressure may rise.

Our data show a marked increase in CSF pressure concomitant with an increase in arterial blood pressure. No such marked change in arterial P_{CO_2} was found. In fact, in two patients arterial P_{CO_2} values decreased while CSF pressures increased dramatically. The increase in P_{CO_2} can be explained, in that we were dealing with unmedicated patients who were anxious and who tended to hyperventilate. With the induction of ketamine anesthesia, the patient's state of awareness no longer contributed to his respiratory drive, and arterial P_{CO_2} tended to rise. Furthermore, the mean increase in arterial P_{CO_2} was only 2.4 torr, which could not account for the increases in CSF pressure recorded.⁹⁻¹¹ Therefore, the cardiovascular changes produced by ketamine may have led to the increase in CSF pressure as postulated.

Further studies of patients with increased intracranial pressure who are having neurodiagnostic procedures are indicated. At present, we can only advise caution in the administration of ketamine to such patients.

SUMMARY

The effect of ketamine on CSF pressure was studied in 11 healthy male volunteers. All patients had marked increases in CSF pressure after intravenous administration of 2 mg/kg

of this drug. Until more complete documentation is available, caution should be exercised in the administration of this drug to patients with increased intracranial pressure.

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