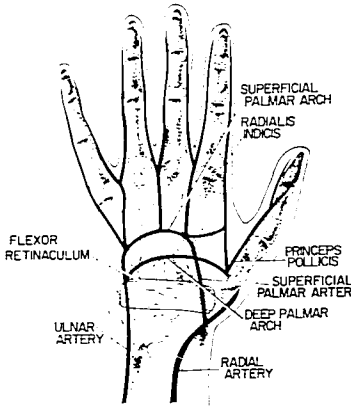

FIG. 2. Anatomy of the palmar arches.

apart. This brief anatomic description illustrates how the patency of these arches can easily be ascertained by the palpation of retrograde radial-artery pulsation following digital compression. This test should be done with all muscles in the limb relaxed, the hand slightly flexed, and the fingers kept close together to avoid compression of the arches by tensing the palmar fascia and the adductor pollicis muscle, through which the termination of the radial artery emerges into the palm. The extended arm should be placed on a flat surface.

Incomplete radial-artery occlusion will result in persistence of retrograde pulsation. If this occurs, ulnar-artery compression will not eliminate it, thus ruling out false-negative tests.

While one volunteer in 20 was found to have an incomplete arch, this should not be interpreted as suggesting that 5 percent of the population has similar anatomic aberrations.

SUMMARY

The patency of palmar arches must be ascertained before radial-artery cannulation is performed. A simple test in which digital pulsation is felt after proximal occlusion of the radial artery has been devised for that purpose.

The method is reliable, does not require active patient cooperation, and can be carried out in all clinical situations where the radial artery can be felt.

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Alterations in Ventricular Fluid Pressure during Ketamine Anesthesia in Hydrocephalic Children

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Dissociative anesthesia with ketamine hydrochloride has been recommended for neurosurgical and neuroradiologic procedures in children. However, there are reports

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Received from the Department of Anesthesiology, University Hospitals, 2065 Adelbert Road, Cleveland, Ohio 44106. Accepted for publication December 29, 1974.

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that ketamine increases ventricular fluid pressure (VFP).[1,2,5] We saw an example of this in a 4-month-old hydrocephalic child who, after receiving ketamine, 2 mg/kg, developed a bulging, tense fontanelle and respiratory arrest.[1] We, therefore, wished to explore whether this effect on VFP could be mitigated by changing the mode of ketamine injection or by premedication. The effect of intramuscular injection was compared with that of intravenous injection of ketamine and the effects of premedication with droperidol, secobarbital, and diazepam was tested.

Method

We studied 26 children, ranging in age from 4½ months to 16 years. Five children, all less than 1 year of age, had open fontanelles. All patients had hydrocephalus with shunts or external ventriculostomies that needed revision. The patients were premedicated 30 minutes before operation with atropine, 0.01 mg/kg, im, and placed supine on the operating table. Heart rate, end-expired CO_2, ECG and temperature were monitored. VFP was measured in the lateral ventricle with a Statham pressure transducer or a water manometer via a 22-gauge needle inserted through the plastic cap of a ventricular Rickham shunt. Free ventricular fluid flow and respiratory excursions in the pressure recordings were taken to indicate intraventricular placement. During anesthesia, when VFP had reached a peak value, an arterial blood sample was drawn and analyzed for *p*H, Pa_{O_2}, and Pa_{CO_2}. All patients were breathing room air spontaneously. Body temperature was monitored and maintained within normal limits. The patients were divided into three groups. Ketamine doses were those recommended by the manufacturer as average.

Six patients in Group I received ketamine, 2.5 mg/kg, iv, within 20 seconds.

Six patients in Group II received ketamine, 10 mg/kg, im, in the anterior lateral thigh.

The patients in Group III, in addition to atropine, were premedicated with droperidol, 0.1 mg/kg, im (five patients), secobarbital, 2 mg/kg im (five patients), or diazepam 0.2 mg/kg, im (four patients) one hour before anesthesia; they were anesthetized with ketamine, 10 mg/kg, im.

Results

The results are summarized in figures 1–3.

In all patients, except one who had not been premedicated, VFP increased after ketamine. The patients in Group I, who had intravenous ketamine, had two- to fivefold increases in VFP. In one patient, a 5-month-old who did not lose consciousness after injection, the pressure did not change.

All patients receiving ketamine intramuscularly developed pressure increases similar to those seen in Group I. There was no statistically significant difference between the responses in Group I and those in Groups II and III (See fig. 1). In both groups, ketamine injections were followed by VFP increases of as much as 1,600 mm H_2O, *i.e.*, two to eight times control values. In several instances we had to remove ventricular fluid when the pressures approached dangerous values.[2]

The VFP generally peaked 4 minutes (3.7 ± 1.3) after ketamine injection by either route, and the mean duration of pressure elevation was about 5 minutes (4.5 ± 3.9). The mean time to awakening with intravenous administration was 8 minutes (8.0 ± 4.0), and with intramuscular administration, half an hour (31.2 ± 16.9 minutes).

In Group III, droperidol, secobarbital, or diazepam premedication did not consistently alter the peak or the duration of the VFP increase (See fig. 2).

No significant change in respiratory rate, heart rate, or systolic blood pressure was associated with the ketamine injection when the values were compared by the signed-ranks test of Wilcoxon (see fig. 3). End-expired CO_2 at peak VFP averaged 24 torr, with 70 per cent of the values below 35 torr. Oxygen tensions at peak VFP during breathing of room air were 60 torr or more.

Discussion

Factors that increase VFP include hypercapnia, hypoxia and increased arterial pressure. In our study these variables did not change enough to explain the increases in VFP. The end-expired CO_2 tensions remained fairly constant at levels below 35 torr; arterial blood pressure did not rise significantly, and Pa_{O_2} did not fall below 60 torr. Cerebral blood flow is not affected by

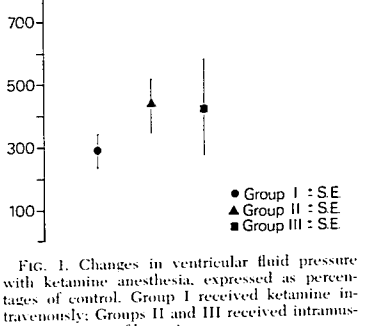

FIG. 1. Changes in ventricular fluid pressure with ketamine anesthesia, expressed as percentages of control. Group I received ketamine intravenously; Groups II and III received intramuscular injections of ketamine.

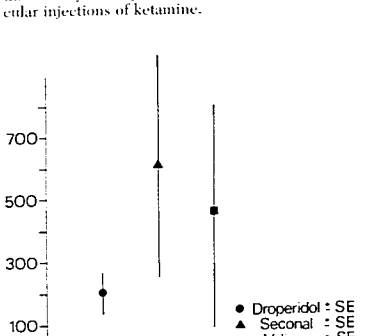

FIG. 2. Changes in ventricular fluid pressure in Group III expressed, as percentages of control. Group III received intramuscular injections of ketamine following premedication with droperidol, secobarbital, or diazepam.

arterial oxygen tensions above 50 torr, when Pa_{CO_2} is constant.[3]

The route of administration of ketamine did not change the time to peak VFP. Patients anesthetized by intramuscular administration of ketamine had higher peak VFP changes than those anesthetized intravenously. We speculate that this is a dose effect, since anesthesia after intramuscular injection lasted longer than that after intravenous injection, which implies that intramuscular injection resulted in a larger effective dose.

Dawson *et al.*[1] found ketamine to be a cerebral stimulant in dogs, as indicated by increases in EEG activity and cerebral oxygen consumption. The same authors reported an increase in cerebral blood flow after ketamine, which they attributed to reduction in cerebrovascular resistance. They also did not record any change in systemic arterial blood pressure in dogs, and stated that thiopental given just prior to ketamine alleviated the effect of ketamine and reduced cerebral blood flow and VFP. This corroborated findings of Wyte *et al.*,[5] who reported alteration of ketamine-induced intracranial hypertension with thiopental in an 8-year-old boy. Based on these findings, we wondered whether barbiturate premedication would alter VFP changes. We failed to demonstrate a decrease in VFP after ketamine with secobarbital premedication. The findings with a sleep dose of thiopental may have resulted from a decrease in cardiac output and a concomitant increase in cerebral vascular resistance.

We used droperidol for premedication in five children. Ketamine has been found to augment α-adrenergic receptor properties in dogs, and these effects have been blocked by phentolamine.[6] Droperidol has α-adrenergic blocking properties,[7] and has been reported to antagonize ketamine-induced tachycardia and hypertension.[8] The use of droperidol in clinical doses did not prevent a ketamine-induced rise of VFP.

Diazepam possesses anticonvulsant activity and produces EEG sleep patterns resembling those associated with normal sleep. We hypothesized that such a compound might counteract the cerebral effects of ketamine, which has been shown to produce CNS excitation in cats[9] and convulsions in epileptic patients.§ However, diazepam failed to prevent the ketamine-induced pressure rise.

The dosages of sedative premedicants used by us may be considered too small to protect against the ketamine effect on VFP. However, larger doses of premedicants are associated with longer and more pronounced CNS depression, the very side effect one wishes to circumvent by using ketamine as the primary anesthetic drug.

§ Jordan W.S., *et al.* Ketamine in Epileptic Patients. Presented at the Annual Meeting of the American Society of Anesthesiologists, 1973.

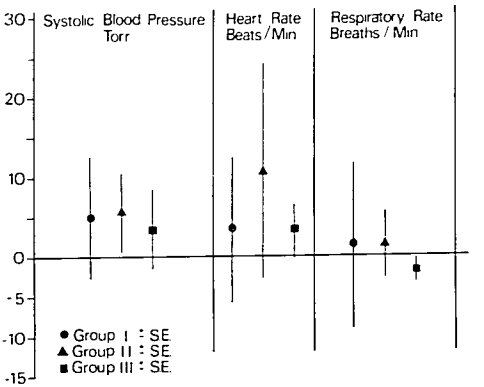

FIG. 3. Changes in blood pressure, heart rate, and respiratory rate at peak ventricular fluid pressure compared with control values.

One might ask whether a transient rise in VFP such as is seen with ketamine is clinically important. Certainly, the crying child raises his VFP. Paulson,[10] in his editorial on intracranial hypertension, discussed the reduced ability of the damaged brain to tolerate increases in intracranial pressure. A brain already compromised by an increased intracranial mass may well have areas of marginal cerebral blood flow. An abrupt rise in VFP could further decrease cerebral perfusion in these compromised areas to below acceptable levels. In addition, the ability of the brain to accommodate is limited by the bony skull and the fixed semirigid falx cerebri and tentorium. An acute elevation of VFP may increase the risk of herniation of brain tissue.

SUMMARY

We studied ventricular fluid pressure changes in 26 hydrocephalic children following administration of ketamine. The increase in VFP previously found with intravenously administered ketamine was compared with changes after ketamine given intramuscularly, and the possible alteration of this increase with sedative premedicants was studied. Changing the route of administration did not change the time to peak VFP changes or the duration of pressure elevation. There was no demonstrable alteration of the increase in VFP by premedication with secobarbital, droperidol, or diazepam in clinical dosage. We feel that acute rises of VFP may affect areas of marginal cerebral blood flow and may increase the risk of herniation of brain tissue.

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