

The Effects of High-frequency Positive-pressure Ventilation on Intracranial Pressure and Brain Surface Movement in Cats

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Compared to traditional, low-frequency ventilation (LFV), mechanical ventilation at high frequencies (60-200 breaths/min) and low tidal volumes (HFV) is known to: 1) eliminate respiratory-synchronous variations in blood pressure; 2) minimize ventilatory effects on the cardiovascular system; 3) reduce peak airway pressures; and 4) suppress spontaneous respiratory efforts. Since these and other properties may make HFV useful in patients with acute intracranial pathology, we studied the effects of HFV on intracranial pressure (ICP) in cats. Compared with LFV (rate 11/min, tidal volume = 15 ml/kg), HFV (rate 100/min, $V_T = 3.3$ ml/kg) had little effect on mean arterial pressure, heart rate, right atrial pressure, mean ICP or mean cerebral perfusion pressures, even if baseline ICP was raised using an epidural balloon. However, HFV effectively eliminated ventilator-linked fluctuations in both blood pressure and ICP, and at all levels of mean ICP studied (4.8, 15, and 30 torr), significantly reduced the peak ICP seen during a single respiratory cycle. The reduction in ICP fluctuation and peak pressure was more pronounced as intracranial compliance fell. However, the physiologic significance of such a change in the ICP pressure waveform is unknown.

Because of the observed influence of HFV on ICP fluctuations, we also examined its effects on the physical movement of the exposed brain, using a non-contact, inductive displacement measuring device. During LFV, the cortical surface moved "in and out" by 0.36 ± 0.1 (\pm SD) mm, a distance sufficient to make microscopic focusing difficult. Changing to HFV reduced surface movement to 0.05 ± 0.01 mm, producing a very stable surface. These results suggest that HFV may play a very important role in the intraoperative management of patients undergoing certain neurosurgical procedures, particularly those requiring microsurgical techniques where reduced brain movement may facilitate surgery. (Key words: Anesthesia; neurosurgical. Brain; intracranial pressure; surface movement. Surgery; neurologic. Ventilation: high frequency; mechanical, tidal volume.)

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HIGH-FREQUENCY POSITIVE-PRESSURE VENTILATION (HFPPV or HFV) was introduced approximately 10 years ago as an experimental method of eliminating the respiratory-synchronous variations in blood pressure that occurred during traditional mechanical ventilation.¹ These systems have used gases intermittently insufflated into the trachea at rates of 60 to 200 times/min, with very small tidal volumes.^{2,3} During the ensuing years, this has been shown to be a satisfactory form of ventilation, producing normocapnic oxygenation without complicating factors such as microatelectasis or lung damage and one which has minimal detectable effects on the cardiovascular system.^{2,4-8} Added benefits include lower peak airway pressures, and an apparent reflex inhibition of spontaneous respiratory drive even in the face of normal P_{aCO_2} values.^{2,5,9} § These latter factors have made HFV particularly useful in patients with pulmonary gas leaks¹⁰ and, since the need for sedation and muscle relaxants seems to be reduced, may be useful in conscious patients requiring mechanical ventilation. A variety of clinical reports have now appeared concerning its use in both the ICU and in the operating room.¹¹⁻¹⁵ More recently, a form of HFV has appeared which uses very high frequency oscillations (up to 40 Hz) applied to the airway, which can adequately ventilate both animals and humans at even lower airway pressures.¹⁶⁻¹⁸

To date, most work on HFV has been devoted to

§ Jonzon A: High-frequency positive-pressure ventilation and carotid sinus nerve stimulation. Acta University Upsaliensis (thesis) 138, 1972.

ABBREVIATIONS

HFV	High-frequency ventilation
LFV	Low-frequency ventilation
V_T	Tidal volume
HR	Heart rate
BP	Arterial blood pressure
RAP	Right atrial pressure
ICP	Intracranial pressure
CPP	Cerebral perfusion pressure
P_{AW}	Proximal airway pressure
ARF	Acute respiratory failure

understanding or applying its beneficial circulatory or pulmonary properties, and little has been done to define its effects on other body systems. Because of ongoing interests in our laboratory, we have looked at the influence of both traditional low-frequency, large-tidal volume ventilation (LFV) and HFV on intracranial pressure. The results obtained led to an evaluation of the effects of both forms of ventilation on brain movement in animals with craniotomies and an open dura, ($ICP = 0$), such as would occur in the operating room. Our observations indicate that HFV may have a valuable role in future neurosurgical and neuroanesthetic practice, particularly during microsurgical procedures, and may also prove useful in a neurointensive care setting.

Materials and Methods

Twelve adult cats of either sex, weighing 3–5 kg were used for all studies. Anesthesia was induced with pentobarbital, 35–40 mg/kg, given intraperitoneally (ip), and supplemented with additional 10 mg doses given iv to maintain a stable level of anesthesia, as assessed by heart rate, pupil size, blood pressure, and the electroencephalogram (EEG). Atropine, 0.08 mg/kg, was given intramuscularly (im) following induction. After paralysis with pancuronium bromide (0.1 mg/kg, iv), the trachea was intubated with a 5.0-mm (i.d.) cuffed endotracheal (ETT) and the cats ventilated with O_2 :air ($FI_{O_2} = 0.4$) at a tidal volume (V_T) of 15 ml/kg and a rate sufficient to maintain normocarbica ($Pa_{CO_2} = 30$ torr). To minimize the anatomical dead space contributed by the upper airways, the tip of the tube was placed as close to the carina as possible. Esophageal temperature was maintained at $37.5^\circ C$ using a servocontrolled heating lamp (Yellow-Springs Instruments). After infiltration of the skin with bupivacaine 0.25 per cent, catheters were introduced into the femoral artery and vein, and threaded into the abdominal aorta and right atrium, respectively. When the lines were secure, the animal was placed in a prone position and the head fixed in a stereotaxic frame (David Kopf Instruments) with the intra-aural line approximately 10 cm above the mid-chest. The scalp was infiltrated with bupivacaine and the skin and temporalis muscles widely resected over one or both sides of the skull depending on the particular experiment.

Monitored variables in *all* cats included heart rate (HR), phasic and electrically meaned arterial blood pressure (BP and \overline{BP}), right atrial pressure (\overline{RAP}), proximal airway pressure (P_{AW} , measured at the ETT connector), lead II of the electrocardiogram (ECG, using subcutaneous electrodes), expired CO_2 (F_{ETCO_2}) (Beckman[®] LB-2 infrared analyzer), FI_{O_2} (Instru-

mentation Laboratories Model 406) and arterial blood gases (Radiometer BMS-3MK2, using 0.3–0.4 ml samples). The EEG was recorded from brass screws in the skull, using lead positions described in the sections below. Data were recorded on a Hewlett Packard[®] 7758 polygraph, using Gould[®] P231D transducers. In ICP experiments, BP and ICP transducers were zeroed at head level to permit accurate assessment of cerebral perfusion pressure ($CPP = \overline{BP} - \overline{ICP}$). For brain movement studies, transducers were balanced at mid-chest.

All mean pressures (\overline{BP} , \overline{ICP} , \overline{RAP}) were obtained by electrical damping. However, due to limitations of the polygraph amplifiers, this value represents an average over the entire respiratory cycle, although both BP and ICP varied widely around this electrical mean during the course of a single respiratory cycle (particularly during LFV). Since this variation was important for these studies, some added numbers were needed. The variation in arterial pressure over the course of a single breath was defined as the difference between the highest and lowest *systolic* blood pressure noted during a breath (and averaged over at least 5 breaths). High and low pressure were identified by inspection of the polygraph tracings. *Maximum* ICP was taken as the highest *peak systolic* pressure seen at any time during a single breath, while minimum ICP represented the lowest end-diastolic value.

No attempt was made to define the variation in cerebral perfusion pressures during a single ventilator cycle, and all values for CPP are obtained from electrical means ($CPP = \overline{BP} - \overline{ICP}$).

INTRACRANIAL PRESSURE MEASUREMENT

For ICP studies, the scalp was reflected from both sides of the skull, and oblong burr holes ($\approx 1 \times 0.7$ cm) were drilled bilaterally over the parietal areas. On the right side, a catheter (PE-90) was placed into the subarachnoid space via a small dural nick, and used to record ICP. The dural incision was sealed with Eastman[®] 910 cement. To allow controlled elevation of baseline ICP, a double-walled latex balloon was laid against the dura on the left side and covered with a foil "roof". EEG screws were placed to allow recording of a single biparietal lead (bipolar) and the entire exposed skull was covered with dental cement.

BRAIN MOVEMENT MEASUREMENTS (5 CATS)

The basis of the measurement technique used has been described elsewhere.¹⁹ Briefly it consists of continuously recording the distance between a thin metal target lying on the surface of the exposed brain, and a sensor-transducer coil mounted nearby

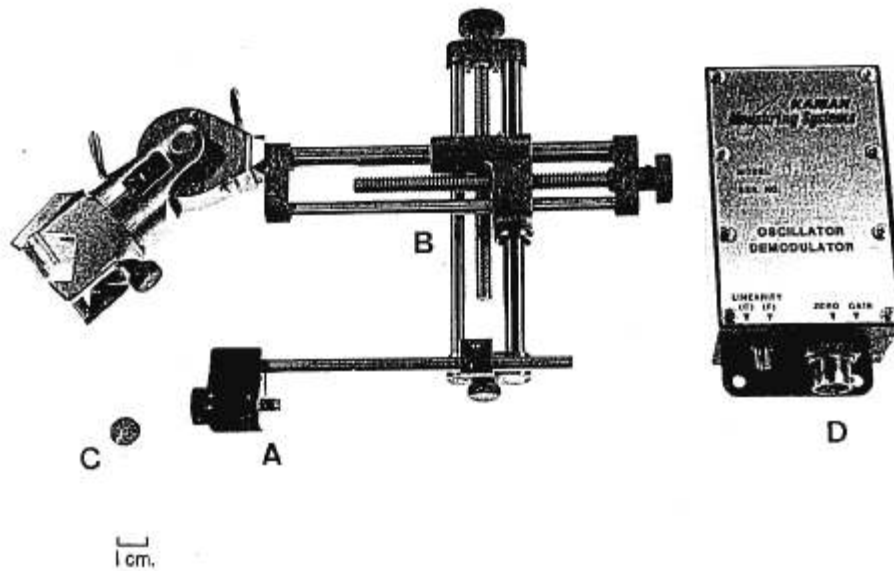


FIG. 1. Displacement measuring device. The system consists of a sensor (A), which has been mounted on a stereotactic manipulator (B) so that it can be carefully brought into proximity with an aluminum foil target (C) which has been laid on the surface of the brain. Output from the oscillator-demodulator unit (D) is recorded on the polygraph. The connecting cable from A to D is not shown. The system is calibrated by mounting the target and sensor on micrometers. For purposes of comparison, note that the diameter of the target (C) is 1.2 cm.

on a stereotaxic manipulator. The method is based on the induction of oscillating currents in the target and continuous measurement of the strength of the secondary electromagnetic field which is thus established. The system is commercially available,[¶] and includes a sensor with a 1.4-cm diameter face, a connecting cable, and an oscillator-amplifier-linearizer unit (fig. 1). It is capable of recording fluctuations in the target-sensor distance of as small as 6×10^{-4} mm, with a response time of 10 μ s. There are no connections to the target or between the target and sensor.

In practice (fig. 2) the scalp was reflected over the right side of the skull, and a large (1.5×2.5 cm) craniotomy performed over the right parietal area. The dura was carefully removed, and a 1.2-cm diameter aluminum foil target disc (0.05 mm thick) was laid on the exposed cortex. The target weighed about 16 mg, moved passively with the brain surface, and produced no apparent compression of the cortical surface. The sensor was moved into place with its face approximately 2 mm from, and coplanar with the target. System output was recorded on the polygraph as millimeters between the target and sensor. Calibration was performed prior to the experiment using a target and sensor mounted on micrometers, and remained stable for weeks. Cortical drying was prevented by a slow continuous drip irrigation with saline, and the target was removed every 30–40 min

to prevent any drying under the contact area. For these experiments, brain temperature was not recorded.

Over experimental times of up to 3 hours there were no signs of cortical damage, such as significant hyperemia, swelling or discoloration. A single lead EEG recorded across the craniotomy (anterior to posterior margins) remained unchanged over the course of the experiments, and movement measurements taken were unchanged over a similar period (hours) when made under identical conditions.

EXPERIMENTAL PROTOCOL AND VENTILATOR SYSTEMS

All experiments were designed to directly compare the effects of the two forms of ventilation over short time periods under otherwise identical circumstances, and involved relatively quick changes from one form of ventilation to the other. LFV was performed with a standard small animal ventilator (Harvard® Apparatus) at a fixed tidal volume of 15 ml/kg and whatever rate was required to maintain normocarbica [11 ± 1 (SD) breaths/min]. The circuit was constructed of plastic tubing (Tygon®) with a compression volume of 0.2 ml/torr of airway pressure. Tidal volume was delivered via the endotracheal tube. HFV used a rodent ventilator with a 30-ml piston (Harvard® Apparatus). The inspiratory limb consisted of an approximately 15-gauge (i.d.) catheter that ended inside the distal tip of the endotracheal tube. Compression volume was negligible. Exhalation was passive (through the ETT) but was valved by the ventilator. Tidal volume and rate were adjusted to yield

[¶] KD-2310-611 Displacement Measuring System. Kaman Sciences Corporation, P.O. Box 7463, Colorado Springs, Colorado 80933. Interested persons should also request application note number 108 for technical details.

the lowest possible airway pressure compatible with normocarbica, with $V_T = 3.3 \pm 0.79$ (SD) ml/kg and rate = 100 ± 5 /min. No added end-expiratory pressure was used. Airway pressures with LFV were 8.0 ± 1.9 over 0 ± 0 torr (end expiration), and $3.8 \pm 1.1/0.8 \pm 0.5$ torr with HFV. I:E ratio for LFV was 1:1, and was 1:2 for HFV. $F_{I_{O_2}}$ was kept at 0.4 on both systems by adjustment of O_2 flow rates.

To facilitate rapid changeover from one mode of ventilation to the other, a Y-shaped connector was constructed that allowed switching from one ventilator to the other within 1–2 s, without the need to disconnect or open the airway (fig. 3).

ICP EXPERIMENTS

ICP was measured before and 5 min after changing the pattern of ventilation. To eliminate any effect of order, both LFV to HFV and HFV to LFV changeovers were performed. In addition to circulatory variables, we specifically noted the maximum and minimum ICP values reached during a respiratory cycle, as well as the electronic mean (see above). Comparisons were done at a normal baseline ICP (balloon empty) and again after ICP had been elevated, first to 15 torr, and finally to 30 torr. The balloon was inflated with saline, using an infusion pump set at a rate of 0.05 ml/min or less. Measurements were performed when ICP had been stable at the desired level for at least 5 min.

Comparisons of measurements taken during two forms of ventilation were disregarded if Pa_{CO_2} varied by more than 3 torr.

BRAIN MOVEMENT

Movement was measured before and 5 min after a change in the mode of ventilation (both orders of change). Since the dura was open, ICP = 0 in all studies. CO_2 restrictions were the same as above.

Values are reported as the mean \pm SD and comparisons between the two modes of ventilations were made using a paired *t* test.

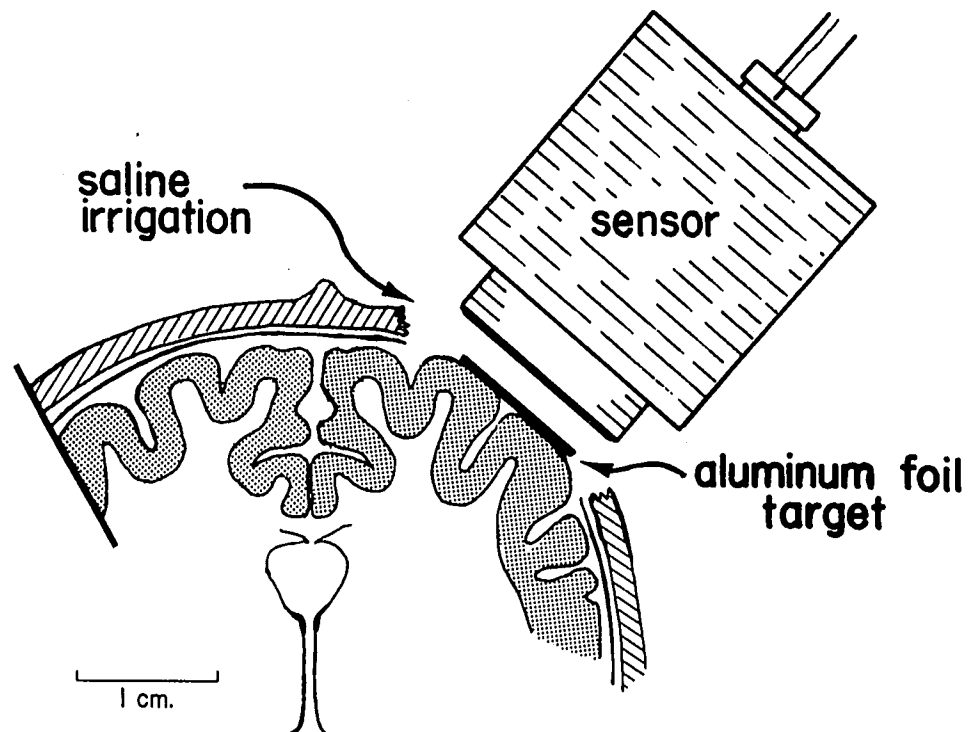
Results

The order of ventilator changeover, (*i.e.*, LFV to HFV or vice versa) had no effect on any measured variable, and therefore all data were combined and will be expressed "as if" all changes from LFV to HFV. There were no EEG changes attributable to the method of ventilation.

INTRACRANIAL PRESSURE EXPERIMENTS

A total of 40 paired comparisons were made in seven cats: eighteen at control ICP values, ten at ICP = 15 torr, and twelve at 30 torr (table 1). HFV had no effect on \overline{BP} , \overline{RAP} , heart rate or blood gases, and had no effect on ICP or CPP except for a 0.4 torr increase in ICP at control levels (4.8 *vs.* 5.2 torr,

FIG. 2. Drawing of a coronal section of the cat's head, showing the relative positions of the sensor and the foil target during movement measurements. The dura has been removed, and the exposed cortical surface kept moist by slow saline irrigation. Sizes are to scale, with the diameter of the target being 1.2 cm, although target thickness has been exaggerated for clarity.



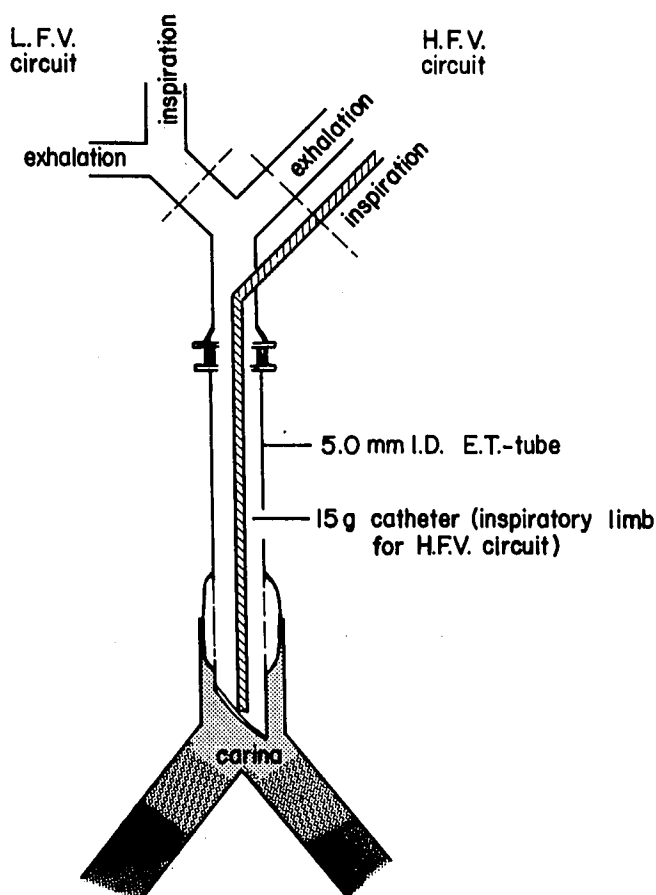


FIG. 3. Diagram of the animal end of the ventilator circuit. The inspiratory limb of the HFV system ends inside the distal tip of the endotracheal tube, which has been placed as close to the carina as possible (to minimize dead space). During both LFV and HFV, expiration is passive, but is valved by the ventilator. Change-over from one system to the other is done by simply occluding or opening the tubing at locations indicated by the dashed lines.

$P < 0.05$). Inflation of the epidural balloon resulted in a gradual fall in \overline{BP} and heart rate under both forms of ventilation.

Phasic Changes. During LFV at all levels of \overline{ICP} , beat-to-beat BP fluctuated widely around \overline{BP} (fig. 4), with the difference between maximum and minimum systolic pressure during a single ventilator cycle being 32.0 ± 9.9 torr. Changing to HFV had no effect on BP, but eliminated any ventilator-synchronous variation (fig. 4). HFV also reduced peak inspiratory RAP in approximate proportion to the reduction in peak airway pressure [2.1 ± 0.9 (HFV) vs. 3.6 ± 0.8 torr (LFV), $P < 0.05$].

Like BP, ICP fluctuated during the LFV-ventilator cycle (fig. 4). The magnitude of this variation increased as \overline{ICP} was raised (see fig. 5). At control levels, with $\overline{ICP} = 4.8 \pm 0.6$ torr, instantaneous ICP during a single ventilator cycle changed from a minimum of 3.7 ± 1.1 to a maximum of 6.6 ± 1.1 torr. At $\overline{ICP} = 14.7 \pm 0.6$ torr, the range was from 10.4 ± 1.4 to 17.9 ± 0.8 torr, and at the highest level studied ($\overline{ICP} = 30.2 \pm 2.3$ torr) the variation was from 24.7 ± 3.7 to 35.2 ± 2.5 torr, with peak ICP exceeding 40 torr in two cases. Changeover to HFV had no effect on ICP except as noted above, but eliminated ventilator-synchronous fluctuations and significantly reduced the magnitude of variation (fig. 4). It significantly reduced peak ICP compared to LFV, but, of course, raised minimum ICP levels. This effect was more pronounced as baseline \overline{ICP} was increased (fig. 5). During HFV, almost all variation in ICP was related to transmitted arterial pulsations, although close inspection of the ICP trace revealed some small fluctuations in pressure coincident with airway and right atrial pressures.

BRAIN MOVEMENT EXPERIMENTS

A total of nine paired comparisons were made in five cats. The effects of changing the mode of ventilation on circulatory parameters and blood gases in these experiments were identical to those noted above and will not be discussed separately.

TABLE 1. Summarized Data for Both Ventilatory Modes at the Three ICP Levels Studied (ICP Experiments). All Values (Except Heart Rate (HR) and pH) are in Torr, and are the Electrical Means \pm SD. See Text for Abbreviations. $\overline{CPP} = \overline{BP} - \overline{ICP}$. $FiO_2 = 0.4$.

	Control ICP		Elevated ICP (Epidural Balloon Inflated)			
	4.8 torr (n = 18)		15 torr (n = 10)		30 torr (n = 12)	
	LFV	HFV	LFV	HFV	LFV	HFV
\overline{ICP}	4.8 ± 1.2	$5.2 \pm 1.3^*$	14.7 ± 0.6	14.8 ± 1.0	30.3 ± 2.3	30.1 ± 2.0
\overline{BP}	167 ± 12	168 ± 12	$152 \pm 7^\dagger$	152 ± 10	$139 \pm 17^\dagger$	$138 \pm 15^\dagger$
\overline{CPP}	162 ± 14	163 ± 11	$137 \pm 8^\dagger$	$137 \pm 9^\dagger$	$111 \pm 16^\dagger$	$110 \pm 13^\dagger$
RAP	1.4 ± 1.0	1.6 ± 1.2	1.2 ± 1.1	1.0 ± 0.7	1.3 ± 1.2	1.2 ± 0.7
HR	236 ± 19	234 ± 17	230 ± 19	228 ± 23	$184 \pm 20^\dagger$	$183 \pm 19^\dagger$
Pa_{O_2}	170 ± 20	168 ± 18	167 ± 14	165 ± 19	161 ± 19	160 ± 18
Pa_{CO_2}	30 ± 3	31 ± 2	29 ± 2	28 ± 2	28 ± 2	28 ± 2
pH	7.36 ± 0.03	7.36 ± 0.03	7.37 ± 0.02	7.37 ± 0.02	7.37 ± 0.02	7.37 ± 0.04

* $P < 0.05$ LFV vs. HFV (paired t test).

$^\dagger P < 0.05$ LFV or HFV vs. control for the same mode of ventilation (unpaired t test).

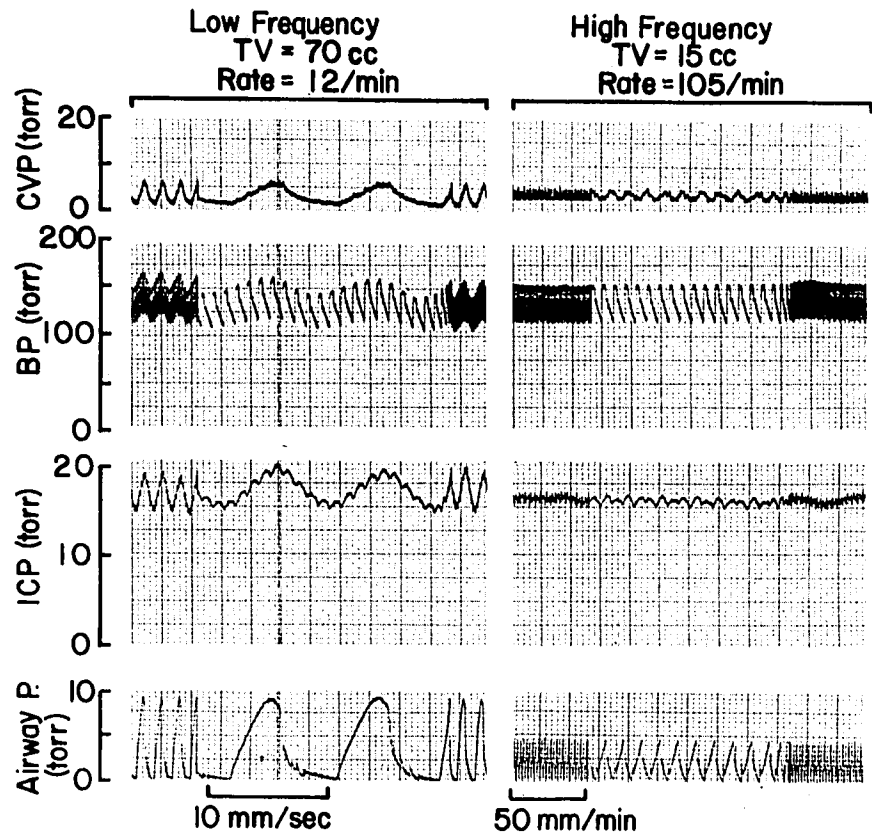


FIG. 4. Polygraph recordings of CVP, BP, ICP and airway pressures during LFV (left) and HFV (right). Change-over to HFV has eliminated ventilation-linked variations in blood pressure, and markedly reduced fluctuations in ICP. Baseline ICP has been elevated by inflation of a contralateral epidural balloon.

During LFV, the cortical surface moved "in and out" by 0.36 ± 0.1 mm. A change to HFV had no effect on the mean position of the brain relative to the sensor, but reduced surface movement to 0.05 ± 0.01 mm ($P < 0.001$) (fig. 6).

In two additional cats, microscopic observation of the brain revealed obvious movement during LFV, with the surface moving in and out of focus with each breath. By contrast, during HFV, the surface was remarkably stable, with no evident ventilation-related movement. Arterial pulsations were apparent with both forms of ventilation, and did not interfere with adequate focusing.

Discussion

High-frequency, positive-pressure ventilation was originally developed as an experimental tool to eliminate respiratory-synchronous fluctuations in blood pressure during mechanical ventilation, and only recently has this technique found its way into clinical practice. Its major advantages compared with traditional modes of ventilation, *i.e.*, large tidal volumes at low rates, are: 1) lower peak airway pressures and thus a lower potential risk of barotrauma; 2) minimal interference with circulatory parameters such as blood pressure, cardiac output, filling pressures (right

atrial and pulmonary artery pressures), heart rate, etc.; and 3) some reflex suppression of spontaneous respiratory efforts. All of these properties have been summarized in two recent collections of articles.^{20,21} Even if respiratory drive is not suppressed, some HFV systems allow spontaneous respiration to continue simultaneously with mechanical ventilation. This particular property may make weaning easier, and in at least one study, made emergence from anesthesia smoother since coughing and "bucking" on the endotracheal tube was minimized by the use of HFV.¹¹ One last benefit seems to be the enhanced clearance of respiratory secretions, possibly making pulmonary toilet easier and more effective.¹⁵

All of these properties make HFV a theoretically attractive alternative mode of mechanical ventilation, both in the ICU and in the operating room, and various workers have described its use in the treatment of acute respiratory failure (ARF) or during bronchoscopy or operative laryngoscopy.¹⁰⁻¹⁵ These same attributes make it a particularly attractive possibility in the neuro-intensive care unit and in a neuroanesthesia setting. For example, if coughing and bucking on the endotracheal tube can be minimized during HFV, it should be possible to avoid their deleterious effects on ICP without resorting to large doses of sedatives or muscle relaxants which can obscure use-

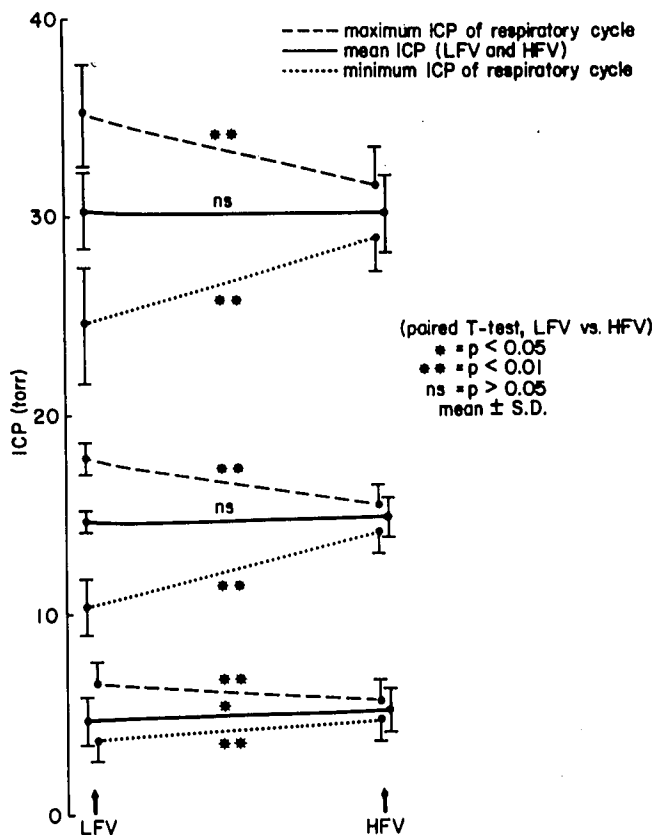


FIG. 5. Summary of the effects of LFV and HFV on ICP. "Maximum" and "minimum" values for ICP are defined in the text and the difference between the two (*i.e.*, the distance between the dashed and dotted lines) reflects ventilation-induced variations in pressure. Mean ICP refers to the electrical mean, taken at end-expiration. At all levels of ICP studied, change-over to HFV had little or no effect on mean ICP (see table 1), but significantly damped respiratory-linked variations, significantly reducing peak ICP, but at the same time increasing minimum ICP. During HFV, fluctuations in ICP are due to some small variations related to CVP and airway pressure.

ful neurologic information. Such a reduction in required sedation has been described for two patients with ARF receiving HFV.¹⁴

The current work began as an examination of the effects of LFV and HFV on ICP at various levels of intracranial compliance. The results clearly showed that compared to traditional LFV, HFV had little effect on BP, RAP, HR, or ICP. However, HFV dramatically reduced phasic variations in both BP and in ICP, and significantly reduced peak ICP seen during a single respiratory cycle. This damping effect on ICP was more pronounced as intracranial compliance was reduced by inflation of the intracranial balloon.

The mechanism by which HFV damps the oscillations in arterial pressure is unknown, although there

are various suggested possibilities.^{2,7} Similarly, the clinical or physiologic importance of the changes in ICP wave-form during HFV is totally unclear. Close inspection of the polygraph traces indicated that peak ICP during LFV generally coincided with, or was only slightly delayed behind the point of maximum BP. While we have not attempted to calculate beat-to-beat values for CPP, this observation suggests that the phasic rise in ICP, even at higher baseline levels (15 and 30 torr) was not accompanied by a drop in CPP, but rather may actually have been caused by a rise in perfusion pressure. It also implies that the effects of HFV on ICP may be mediated entirely via its effects on the peripheral circulation.

It is well known that pathologic variations in ICP can result from fluctuations in cerebral blood volume.²² When the skull is open, such as occurs during a neurosurgical procedure, such volume changes are immediately seen as expansion or contraction of the cortical surface. An example might be the bulging seen during hypercarbia and the phasic pulsation of the brain in synchrony with the arterial pulse wave and with respiration. Until recently, this respiratory-linked movement has been regarded as a curiosity or as a mild neurosurgical nuisance, but with the introduction of increasingly delicate microsurgical techniques, it has been recognized as a real impediment to certain surgical procedures, particularly microvascular anastomosis.²³ Our studies clearly show that a change to HFV can effectively eliminate ventilation-linked brain movement. However, close inspection does show some small fluctuations in the brain occurring simultaneously with inspiration (at the time of peak airway or atrial pressure), suggesting that some direct transmission of intrathoracic pressure is still present. This may indicate that a further reduction in peak airway and intrathoracic pressure may improve the situation even more. One potential means of accomplishing this might be to use higher frequencies, with low volumes and ventilatory oscillations at rates of up to 40 Hz.¹⁶⁻¹⁸

The usefulness of brain motion studies as a physiologic tool deserves some comment. If the dura is open, and changes in CSF volume are eliminated from consideration, changes in the volume of the brain (as reflected by cortical swelling and contraction) must be the result of changes in either cerebral blood volume of tissue volume. Very rapid changes (such as seen during a single respiratory cycle), almost certainly reflect rapid variations in cerebral blood volume.²⁴⁻²⁶ These measurements cannot be used to accurately quantitate changes in whole brain volume, but it should be possible to use such a method to compare the relative influence of various drugs,

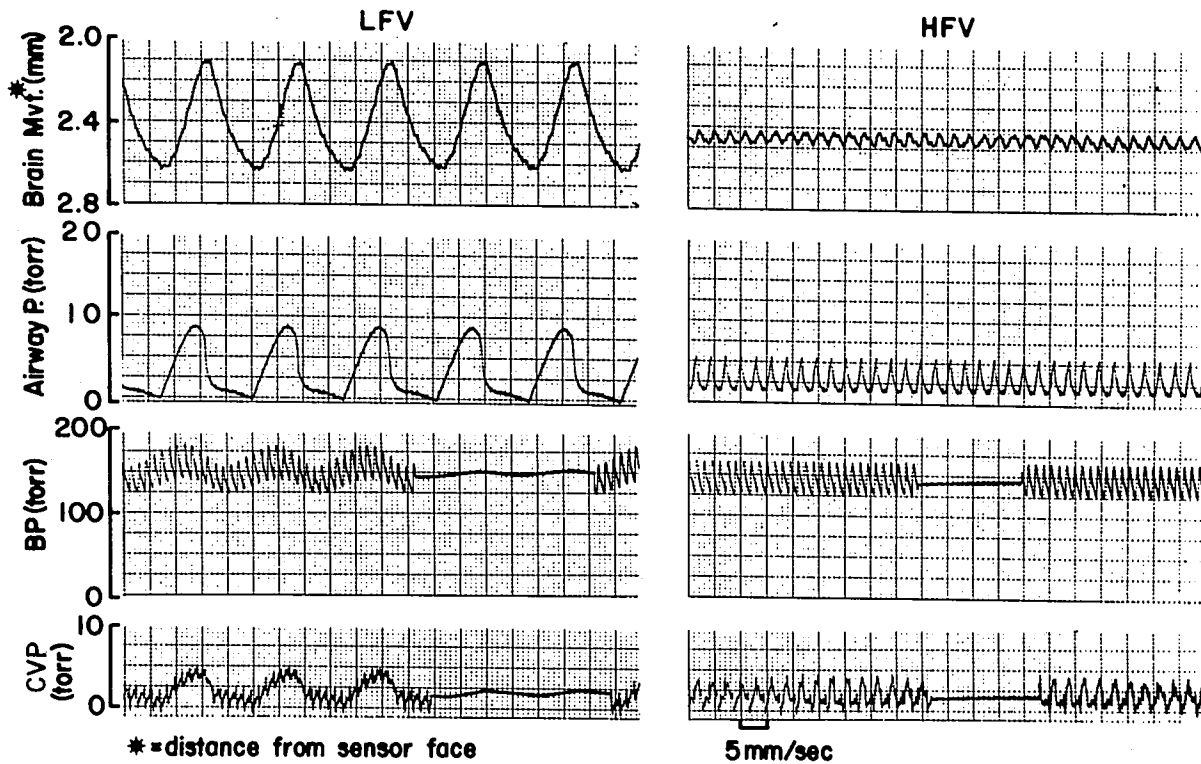


FIG. 6. Brain surface movement during LFV (left) and HFV (right). Brain movement (top trace) is defined as the variation in the distance of the brain surface (foil target) from the sensor face. Upward deflection of the pen indicates movement of the surface toward the sensor, *i.e.*, upward, out of the skull. Full scale on the movement trace is 0.8 mm. Note that change-over to HFV markedly reduced the measured surface motion. The reduction was easily seen with the naked eye. Residual motion during HFV was linked to fluctuations in airway pressure and CVP.

respiratory and circulatory maneuvers, etc., on blood volume, without the complicating factors of intracranial pressure and compliance. It may also allow insights into the mechanisms by which changes in respiratory-circulatory patterns influence CBV and CBF. Of course, it cannot replace but only supplement direct measurements of ICP, cerebral blood flow and volume. At the very least, however, the data from such studies may have direct application in the selection of agents and techniques (*e.g.*, volatile anesthetics) for use during neurosurgery, when the head is open.

As noted, the clinical importance of the effects of HFV on the ICP wave-form and on peak ICP is unknown, and it is impossible to firmly state that HFV will prove beneficial in a neurologic intensive care setting. Nevertheless, if no detrimental effects of HFV on the central nervous system are found, the various cardiovascular and respiratory properties discussed earlier may make it a useful tool. However, the observed reduction in brain movement produced by HFV may have a much more immediate applicability. If clinical trials confirm that some form of HFV can indeed improve operating conditions during microsurgical procedures, then this mode of

ventilation could come to play a very important role in clinical neuroanesthesia and neurosurgery.

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