Arterial blood gases during raised intracranial pressure in anesthetized cats under controlled ventilation

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In anesthetized paralyzed cats ventilated with air, blood gases were analyzed repeatedly before and during episodes of raised intracranial pressure (ICP). The ICP was raised by infusion via the lumbar subarachnoid or intraventricular route, and increases were maintained for at least 30 minutes. A minor degree of hypoxemia commonly developed, but was always associated with hypercapnia; normoxia was restored by increasing the ventilation sufficiently to restore normocapnia. Relative under-ventilation is thus liable to develop if the minute volume is maintained constant when ICP is raised, probably because of increased metabolic rate which may be associated with a rise in temperature; there is no evidence to implicate more obscure causes of hypoxemia in this circumstance. Pulmonary hemorrhage and edema were found post mortem in nine of 20 animals, but only two of these had developed greater hypoxemia than could be accounted for by under-ventilation. Phrenic electroneurograms were recorded, and respiratory activity was shown to persist during prolonged periods of cerebral ischemia.

KEY WORDS • intracranial pressure • hypoxia • pulmonary edema • cerebral ischemia

PULMONARY hemorrhage and edema may be associated with brain damage induced clinically or experimentally by a variety of agents; also some degree of arterial hypoxemia is common in patients with brain damage. Some experimental evidence suggests that not only edema but also varying degrees of ventilation/perfusion (VA/Q) imbalance may result from a direct neural effect on the lungs. Both factors could contribute to hypoxemia.

Raised intracranial pressure (ICP) commonly accompanies brain damage. Some investigators have linked experimentally maintained increases in ICP in dogs with progressive arterial hypoxemia or intra-pulmonary shunt, but other workers have not confirmed this relationship. A previous study in spontaneously breathing cats, in which the ICP was increased for 10-minute periods, indicated that hypoxemia developed only to an extent accounted for by ventilatory depression. It was suggested to us that the development of hypoxemia might require more prolonged exposure to raised ICP, although a neurally-mediated effect on the lungs could be expected to develop as rapidly as, and concurrently with, the effect on systemic circulation.

Accordingly, in the experiments described here increases in ICP were maintained for at least 30 minutes to establish whether or not
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hypoxemia developed during a longer period when ventilation was controlled with air as the inspired gas. In order to estimate brainstem respiratory function in the absence of spontaneous breathing, phrenic nerve activity was recorded.

Materials and Methods

Two series of experiments were conducted separately, the first by both authors, the second by J.H. alone.

Animal Groups

Series 1. Ten cats, weighing 2 to 4 kg, were anesthetized with halothane and nitrous oxide, and anesthesia was maintained with intravenous pentobarbitone (30 mg/kg, with later supplementary doses of 5 to 10 mg/kg hourly). For the period of controlled ventilation, the cats were paralyzed with 2 ml gallamine (Flaxedil), repeated after about 2 hours as necessary. Air was inspired throughout.

After tracheal and femoral arterial and venous cannulation, a lumbar laminectomy was performed and a cannula passed up alongside the spinal cord intrathecally for several centimeters. It was secured and sealed in place by an extradural circumferential ligature. A parietal burr hole was made, and a cannula with multiple punctures at the tip was inserted into the subdural plane. The skull was sealed with dental acrylic. This cannula was attached to a pressure transducer and the output checked for normal pulsations; the presence of these, and an immediate rise of ICP in response to a small infusion, allowed repeated confirmation of free communication and patency, indicating valid ICP measurements.

The following continuous measurements were established on an ultraviolet chart recorder: pressures from the intracranial cannula (ICP) and from the femoral artery (BP) measured by photoelectric transducers, calibrated in parallel against a mercury manometer; %CO₂ in inspired-expired air from the tracheal cannula, for calculation of end-tidal pCO₂ by rapid infra-red analyzer calibrated by gas mixtures analyzed on the Lloyd Haldane apparatus; integrated inspired volume (V₁) by pneumotachograph with a flow head attached to the tracheal cannula calibrated for flow rates of 2 to 6 liters per minute by Rotameters, and being within 5% of linearity at the extremes of this range; instantaneous heart rate (HR), with the BP transducer output to trigger a ratemeter via an oscilloscope.*

For the phrenic electroneurogram, the right phrenic nerve was exposed in the neck and divided; the distal end was stripped and placed on electrodes under paraffin for audio-monitoring and to record integrated electrical activity by means of an AC integrator assembled for the purpose (amplifier, integrator, and a low-pass filter). The peak output obtained in each burst of inspiratory impulses measured by this instrumentation correlates well with tidal volume during spontaneous breathing.

In addition to these continuous records, arterial blood was sampled intermittently, and immediately analyzed for pH, pCO₂, and pO₂ on the Radiometer system, calibrated by gas mixtures from a Wosthoff pump. Measurement temperature was 37°C, and the values were corrected to the animal's rectal temperature at the time of each sample, using the Severinghaus blood gas calculator.†

Rectal temperature was recorded at intervals of 15 minutes throughout, and the heated operating table was adjusted to correct deviations from normal. Mock CSF* at 37°C was infused to raise ICP; infusion was controlled


†Severinghaus blood gas calculator manufactured by Radiometer A/S 72 Emdrupvej, DK 2400, Copenhagen, Denmark.
Fro. 1. Example of changes in one animal in Series 1, during the sequence of alterations in intra-cranial pressure (ICP). Open symbols = PaO₂; closed symbols = PaCO₂. Between the vertical lines, ICP (shaded) was held at 100, 150, and 200 mm Hg, respectively. Broken line = mean arterial blood pressure (BP). Mean BP and ICP are shown graphically as a constant average: the values below show the range of fluctuations of cerebral perfusion pressure (CPP = BP − ICP) during each run. At each arrow, the ventilation was increased so as to restore the PaCO₂ as nearly as possible to the value before the ICP was raised. During the run at 200 mm Hg, BP fell below ICP and the experiment was terminated.

manually during pressure increases and a rate of rise of 1 mm/sec was achieved by reference to the display of the ICP transducer on the recording apparatus; thereafter the raised pressure was maintained by means of a pressurized Woulff bottle.‡

Series 2. Ten cats, weighing 2.5 to 4 kg, were anesthetized and monitored according to the method described for the animals in Series 1. Mock CSF was infused not into the lumbar subarachnoid space, but into the right lateral ventricle through a No. 20 needle placed stereotaxically and secured with dental acrylic. Phrenic activity was not recorded.

Procedure

All recording systems were established and the anesthetic level was stabilized with the animal breathing spontaneously, so that its arterial blood gases were within the normal range for the cat.⁷ The animal was then paralyzed and the level of mechanical ventilation adjusted until the end-expired pCO₂ was close to the previous level during spontaneous breathing. When the end-tidal pCO₂ and ventilation had remained steady for at least 5 minutes, a baseline arterial sample was analyzed; intracranial pressure was then raised.

Pattern of Intracranial Pressure Increase

Series 1. A sequence of ICP elevations was imposed starting at 80 to 100 mm Hg. This level was chosen because it had been shown earlier to be associated with effects on respiration and on arterial blood pressure,¹ and with increased sympathetic activity.¹⁰ Raised ICP was maintained for 30 minutes; pressure was then lowered and all recorded variables were allowed to return to normal; ICP was then raised to about 150 mm Hg, and again maintained for 30 minutes; recovery was allowed once more, after which ICP was raised to 200 mm Hg, and held there for as long as the arterial BP remained elevated. If BP began to decrease, indicating imminent circulatory failure, CSF infusion was stopped, ICP was allowed to fall, and the animal was immediately killed by an overdose of barbiturate.
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Series 2. These animals were subjected to a single increase of ICP of 150 mm Hg lasting 30 to 60 minutes (six cats) or of 200 mm Hg lasting 30 minutes or as long as the animal tolerated it (four cats). A 30-minute recovery period followed and the animal was then killed by an overdose of barbiturate. The volume of mock CSF required to maintain ICP elevations never exceeded 40 cc/kg/hr, an amount shown in other studies to have no effect on arterial blood gases.11

Ventilation and Blood Gas Measurements

Cumulative inspired volume and tidal CO₂ were recorded continuously. Arterial blood gases were measured immediately before each increase of ICP (in a period of stable ventilation and end-expired pCO₂) and at 10-minute intervals during raised pressure. When the period at 200 mm Hg was less than 10 minutes, a final sample was taken immediately before lowering ICP.

During the first minutes of raised ICP, the end-expired pCO₂ usually increased despite constant ventilation. In Series 1 experiments, the pump-stroke volume was increased sufficiently to restore the end-expired pCO₂ to its previous value before the final blood sample was taken at any given ICP. In Series 2, ventilation was not adjusted when pCO₂ increased.

The lungs were examined macroscopically post mortem.

Results

Arterial Blood Gases and End-Expired pCO₂

When ICP was raised, a small increase in the end-expired pCO₂ usually began within the first minute and was progressive, while the recorded inspired minute volume was unchanged. The arterial blood sample taken at 10 minutes showed a parallel small rise in the arterial CO₂ and a small decrease in PaO₂ compared with the baseline measurement (Fig. 1). The final sample at raised pressure showed that the increased ventilation which corrected the hypercapnia had, in most instances, also corrected the hypoxemia. This was true for seven of nine animals at 150 mm Hg ICP (Fig. 2), and suggested that the changes had been due to relative under-ventilation. In three of the seven, there was a small and inconsistent rise in body temperature during raised ICP; in the other four animals, temperature was steady. The two exceptions showed hemorrhagic edematous lungs post mortem; in one, the hypoxemia progressed until the end of the experiment but the other regained normoxia before and during the subsequent period at an ICP of 200 mm Hg.

Figure 3 contrasts these results for Series 1 with those for animals at 150 mm Hg ICP in Series 2 that were exposed to the same ICP for the same period, but without the adjustment to normocapnia. The latter group showed a persistent, usually progressive, minor degree of hypercapnia and hypoxia, which resolved after release of pressure; the most marked changes occurred in three animals which developed a rise in body temperature. The mean changes in PaO₂ from baseline to 30 minutes at ICP 150 mm Hg were significantly different between the two groups; there was no significant mean change in either PaCO₂ or PaO₂ from the baseline measurements to that at 30 minutes of ICP 150 mm Hg in Series 1 where ventilation was adjusted (Table 1).

The graphic data refer only to periods at ICP 150 mm Hg because full data were available for every animal after 30 minutes at

<table>
<thead>
<tr>
<th>Variables</th>
<th>Series 1 Ventilation Adjusted</th>
<th>Series 2 Ventilation Not Adjusted</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n = 9 (mean ± SE)</td>
<td>n = 7 (mean ± SE)</td>
</tr>
<tr>
<td>PaO₂ (torr)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>initial</td>
<td>88.9 ± 2.6*</td>
<td>99.7 ± 5.1*</td>
</tr>
<tr>
<td>final</td>
<td>87.4 ± 2.7</td>
<td>85.3 ± 5.8</td>
</tr>
<tr>
<td>change</td>
<td>-1.5 ± 2.6†</td>
<td>-14.7 ± 2.6†</td>
</tr>
<tr>
<td>PaCO₂ (torr)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>initial</td>
<td>32.6 ± 0.9</td>
<td>35.3 ± 1.1</td>
</tr>
<tr>
<td>final</td>
<td>30.2 ± 0.8</td>
<td>40.9 ± 1.4</td>
</tr>
<tr>
<td>change</td>
<td>-2.4 ± 1.2‡</td>
<td>5.6 ± 1.1‡</td>
</tr>
</tbody>
</table>

*The difference in initial PaO₂ between the two groups is not statistically significant: it suggests, however, a better state of pulmonary gas exchange in "normal" cats in San Francisco than in Glasgow.
†Difference between series: p < 0.001.
‡Difference between series: p < 0.01.
FIG. 2. Changes in \( P_{aO_2} \) and \( P_{aCO_2} \) for each animal of Series 1, during the period at ICP 150 mm Hg. Ventilation increased at the arrow. Same data as Fig. 3 left. The two animals represented below right, were those in which normoxia was not restored at normocapnia. These and one other showed frank pulmonary edema (E).

TABLE 2
Values for intracranial pressure (ICP), mean arterial blood pressure (MBP), and cerebral perfusion pressure (CPP) in Series 1

<table>
<thead>
<tr>
<th>Run</th>
<th>ICP Range (mm Hg)</th>
<th>MBP Mean (mm Hg)</th>
<th>MBP Range (mm Hg)</th>
<th>CPP Mean (mm Hg)</th>
<th>CPP Range (mm Hg)</th>
<th>Duration (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>80–100</td>
<td>120–170</td>
<td>45</td>
<td>25–80</td>
<td>30</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>140–160</td>
<td>155–210</td>
<td>38</td>
<td>10–70</td>
<td>30</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>180–220</td>
<td>190–220</td>
<td>10</td>
<td>0–25</td>
<td>3–25</td>
<td></td>
</tr>
</tbody>
</table>

In all instances arterial blood pressure increased promptly as ICP was raised (Figs. 4, 5, 7), and remained steadily elevated. Extent of increase varied from animal to animal, so that any one level of raised ICP represented a different cerebral perfusion pressure (CPP = mean BP – mean ICP) in each animal (Table 2). In many instances, a CPP of only 10 to 25 mm Hg was maintained for 30 minutes without a sign of deterioration in cardiovascular function (Fig. 5). This is considerably less than the value for CPP below which cerebral vasodilatation can no longer main-
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Fig. 3. Changes in blood gases over 30 minutes at an ICP of 150 mm Hg, compared between Series 1 animals in which ventilation was adjusted to correct the PaCO₂ (left), and Series 2 animals in which ventilation was kept constant. Closed circles and broken lines = PaCO₂; open circles and unbroken lines = PaO₂. On left, the broken lines show range of PaCO₂ changes, which were on average nonsignificant. Mean ΔPaO₂ and ΔPaCO₂ were significantly different between the two series (Table 1).

Respiratory Activity

The phrenic electroneurogram was recorded successfully in seven Series 1 animals and was of interest in the following three respects:

1. During the rise in ICP, with mechanical ventilation of the lungs maintained constant, inspiratory activity in many instances increased (Fig. 7), a phenomenon which confirms the earlier finding that respiration is stimulated in spontaneously breathing animals in otherwise similar conditions.¹²

2. As CPP decreased, there were various irregularities in phrenic activity, including a Cheyne-Stokes pattern. Such periodicity in the spontaneously breathing animal might be thought to be related to fluctuating blood gas levels but in this instance PaO₂ and PaCO₂ were constant and normal. The relationship

<table>
<thead>
<tr>
<th>Cat No.</th>
<th>Discharge Present at CPP (mm Hg)</th>
<th>after (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>20</td>
<td>10</td>
</tr>
<tr>
<td>4</td>
<td>25-35</td>
<td>20</td>
</tr>
<tr>
<td>5</td>
<td>20-25</td>
<td>30</td>
</tr>
<tr>
<td>6</td>
<td>25-30</td>
<td>30</td>
</tr>
<tr>
<td>7</td>
<td>0</td>
<td>10</td>
</tr>
<tr>
<td>9</td>
<td>5-20</td>
<td>30</td>
</tr>
<tr>
<td>10</td>
<td>0</td>
<td>30</td>
</tr>
</tbody>
</table>
FIG. 4. Example of recorded variables, during a period at ICP approximately 100 mm Hg. Reading from top down: Airway CO₂; heart rate (HR); arterial blood pressure (BP); intracranial pressure (ICP); cerebral perfusion pressure (CPP) = 25 mm Hg (difference between mean BP and mean ICP); cumulative minute volume ("staircase" trace — constant ventilation); cerebral venous pressure (CVP); integrated phrenic electroneurogram, showing "breaths" that are irregular both in size and frequency.

FIG. 5. Same recorded variables as in Fig. 4. An intracranial pressure (ICP) of approximately 150 mm Hg had been maintained in this animal for 20 minutes at the time of this trace. The ICP was falling below the required level and was deliberately increased twice during the period shown (at A and at B); the immediate blood pressure (BP) response can be seen, and also a stimulation of "breathing."
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![Graph showing CO2%, BP, ICP, and CVP over time]

Fig. 6. Example of low perfusion pressure (varying between zero and 30 mm Hg) maintained for 30 minutes at ICP 150 mm Hg. The blood pressure (BP) and intracranial pressure (ICP) traces overlap and each was separately interrupted at the dotted lines. The phrenic activity shows an irregular pattern of "breaths" with a tendency to periodicity. The peak activity coincides with the lowest perfusion pressure.

of respiratory activity to cerebral ischemia was not clear, because in some cases maximal activity appeared to coincide with minimal perfusion pressure, rather than the reverse (Fig. 6). However, when animals were virtually "apneic," the occasional "breath" occurred only when BP exceeded ICP.

3. Inspiratory activity survived remarkably prolonged low CPP in several instances (Table 3). In such conditions spontaneously breathing animals would be expected to become apneic.

Appearance of Lungs

Series 1. The lungs appeared normal immediately after death in seven of the 10 animals. The remaining three showed gross edema and patchy hemorrhage; two of these had been slightly hypoxemic (PaO2 82, 77 torr, respectively) and the third was normoxic at normocapnia 5 minutes before sacrifice, after 30 minutes with ICP at 200 mm Hg.

Series 2. The lungs were grossly normal in four of the six animals which had a sustained ICP of 150 mm Hg for 30 minutes; the other two showed gross edema and hemorrhage. All four animals exposed to an ICP of 200 mm Hg for 20 to 30 minutes had gross hemorrhagic change with edema.

Discussion

Pulmonary Edema and Hypoxemia

Increased sympathetic outflow with systemic vasoconstriction can shift blood into the pulmonary circulation; this shift, in conjunction with alterations in cardiac function, could indirectly affect oxygenation by altering the rate of fluid extravasation and/or the distribution of V_\text{A}/Q ratios. In addition, sympathetic stimulation or autonomic imbalance might act directly on the lungs: pulmonary vasoconstriction could increase the capillary pressure; the V_\text{A}/Q distribution might be affected so as to cause an effective increase in shunting and thence hypoxemia; surfactant might be depleted,
resulting in decreased compliance and a tendency to extravasation.

Grossly evident pulmonary edema was found in three animals out of 10 in Series 1; a minor degree of hypoxemia occurred in two of these, and the other was normoxic after ventilation was adjusted. Six animals in Series 2 showed similar changes but in no instance did \( \text{PaO}_2 \) decrease below 70 torr during the 30 minutes after ICP elevation was stopped and before sacrifice.

Our results provide no particular evidence for or against a direct neural effect on the lungs causing congestion and edema, as opposed to the alternative explanation in terms of the consequences of systemic effects. Elevation of blood pressure in all animals was severe and prolonged, implying that there must have been widespread systemic vasoconstriction and some shift of blood into the lesser circulation. The late and inconstant development of edema is compatible with an earlier increase in extravasation rate, which would not necessarily exceed the capacity of lymphatic drainage, or if it did, would take time to cause accumulation of fluid to a grossly recognizable extent. It is quite possible that lesser degrees of interstitial edema took place in the other animals in our two series, and could have been detected by appropriate methods for estimating lung water.

It is known that considerable increases in interstitial fluid can occur without impairing diffusion, so the absence of hypoxemia in this series, except in association with frank lung changes, is understandable. In another series of experiments using a similar preparation for raised ICP, lung water has been measured and a significant increase was usual; again hypoxemia did not occur. The persistent normoxia (after correction of the mild hypercapnia) in the face of prolonged increase in ICP, in arterial BP, and presumably in sympathetic outflow, is evidence against any direct or indirect pulmonary hemodynamic effect causing \( \dot{V}_A/Q \) maldistribution. Correction of \( \text{PaO}_2 \) might have been achieved in such circumstances but only by hyperventilation to hypocapnia.
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Fig. 8. Diagram showing the interrelationship between $P_aO_2$ and $P_aCO_2$ for cats under pentobarbitone anesthesia. The regression lines refer to an earlier series of spontaneously breathing animals; the solid line relates $P_aO_2$ to $P_aCO_2$ in different animals at different levels of anesthesia, at normal intracranial pressure (ICP); the broken line relates values in the same group at raised ICP, showing varying degrees of ventilatory depression. Blood gas data from the present experiments on ventilated animals have been superimposed. Open symbols represent estimations at raised ICP, triangles with, and circles without adjustment of ventilation; and closed symbols, at normal ICP.

The existing evidence in favor of such an effect derives from experiments in dogs breathing 100% oxygen. Berman and Ducker reported an increase in alveolar-arterial pO2 difference during raised ICP, and Maxwell and Goodwin showed an increase in the calculated percentage shunt, which was related to the severity of applied ICP. The magnitude of the shunt increase in the latter study (7% to 11%) was such that, if an animal were breathing air, the PaO2 could decrease from, say, 100 to 75 torr. Some cats in our present series showed a comparable decrease in arterial pO2. Had this hypoxemia been due to shunt, it would not have been corrected by ventilating to normocapnia. Thus our results do not require the hypothesis that raised ICP, by virtue of increasing sympathetic outflow, induces shunting in the lungs. The reason for the difference between these and other findings is not clear.

Increased Metabolic Rate During Raised Intracranial Pressure

The mild hypoxemia and hypercapnia that occurred with unchanged minute volume, suggested an increase in metabolic activity and therefore relative hypoventilation. In some animals this could be accounted for by an increase in body temperature, but in others in which temperature was better controlled the phenomenon still occurred. This would be compatible with an increase in catecholamine release.

A possible alternative explanation of the rise in pCO2 might have been that effective alveolar ventilation was reduced because of an increase in alveolar dead space; this would have caused a widening of the end-expired arterial difference for pCO2, which did not occur. Rise in body temperature could increase the ventilation requirement, and it is perhaps

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significant that those animals that developed the greatest degree of hypoxia and hypercapnia were those in which the temperatures increased noticeably during the relevant period. Conversely, two animals which showed no change in blood gases during a period at 200 mm Hg ICP had a fall in body temperature at that time.

Data from previous experiments in spontaneously breathing cats plotted on an O₂–CO₂ diagram, served as the basis for predicting the likely change in PaO₂ relative to the change in PaCO₂, when ventilation was depressed either by raised ICP or by incremental barbiturates; in that study, the ΔPaCO₂/ΔPaO₂ relationship was similar in the two circumstances, making it unnecessary to postulate a specific effect of raised ICP on the lungs. In the present series, the data fitted this same relationship (Fig. 8).

Respiratory Activity During Severely Decreased Cerebral Perfusion Pressure

The persistence of inspiratory impulse activity during prolonged increases in ICP, when the spontaneously breathing animal would almost certainly have been apneic, requires explanation. Hypercapnia and hypoxemia develop when respiration begins to diminish in the breathing animal. A decrease in PaO₂ in the presence of cerebral ischemia will further threaten the activity or respiratory neurons. Thus, a positive feedback mechanism is operative and tends to worsen the situation. In the artificially ventilated animals, however, arterial blood gases were normal. It remains remarkable that regular phrenic activity persisted for so long in some instances.

Conclusions

Anesthetized cats, breathing air, commonly develop a slight increase in PaCO₂ and a concomitant decrease in PaO₂ during periods of severely increased intracranial pressure, if the ventilation is kept constant at the level that maintained normocapnia at normal ICP. However, if the ventilation is adjusted to correct the pCO₂, the pO₂ is also corrected, implying that the alteration resulted from inadequate alveolar ventilation. This could be due to increased metabolic rate related to increased sympathetic activity, and is sometimes, but not necessarily, associated with a rise in body temperature. We conclude that, in this preparation, there are no grounds for implicating a specific effect of raised intracranial pressure on the pulmonary vascular bed that leads to hypoxemia. A proportion of animals develop pulmonary edema and hemorrhage, but even this is not necessarily associated with arterial hypoxemia, despite being sufficient to be recognizable macroscopically. Unless there is a species difference, therefore, raised ICP per se is probably not the factor in clinical brain damage responsible for hypoxemia until or unless it leads to frank pulmonary edema.

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References

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