Transmission of Intrathoracic Pressure to the Intracranial Space during Cardiopulmonary Resuscitation in Dogs

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SUMMARY. Elevation of intrathoracic pressure during cardiopulmonary resuscitation generates carotid pressure and flow, but also increases intracranial pressure. This increase in intracranial pressure may limit cerebral blood flow. Therefore, we performed studies designed to quantify the extent of this transmission and to identify the mechanism of transmission of intrathoracic pressure to the intracranial space during cardiopulmonary resuscitation in dogs. Intracranial pressure increased during the chest compression phase of all modes of cardiopulmonary resuscitation tested. During simultaneous compression-ventilation cardiopulmonary resuscitation, change in intracranial pressure (mm Hg) = 0.33 change in intrathoracic pressure (mm Hg) + 2.02 (r = 0.86) and was not significantly different from the relationship observed during conventional cardiopulmonary resuscitation. The magnitude of transmission of intrathoracic pressure to the intracranial space was increased by binding the abdomen and by raising the baseline intracranial pressure. No single route accounted for transmission of intrathoracic pressure to the intracranial space during cardiopulmonary resuscitation. Intracranial pressure fluctuations were unrelated to either carotid arterial or jugular venous pressure, and were found instead to be the result of pressure transmission by blood in non-valved veins and by cerebrospinal fluid. This was determined by three maneuvers. First, obstruction of cerebrospinal fluid flow by ligation of the cervical spinal cord reduced intracranial pressure (P < 0.001) and made the change in intracranial pressure equivalent to pressure changes at the confluence of the intracranial venous sinuses, without affecting pressure changes at the confluence of the intracranial venous sinuses. Second, ligation of the cervical spinal cord and one of the two longitudinal vertebral veins adjacent to the cervical cord reduced the pressure changes in the intracranial space and at the confluence of the intracranial venous sinuses to about 60% of the levels observed when the cervical cord alone was ligated. Thus, the non-valved longitudinal vertebral veins appear to be the vascular channels of critical importance to pressure transmission. Finally, pressure changes in the thoracic cerebrospinal fluid were increased (P < 0.05) by cord ligation, even after exsanguination minimized pressure transmission via blood-filled channels, indicating direct transmission of intrathoracic pressure through intervertebral foramina to the cerebrospinal fluid. Thus, although non-valved veins and cerebrospinal fluid account for transmission of intrathoracic pressure to the intracranial space during cardiopulmonary resuscitation, this pressure transmission is modest except with abdominal binding or under conditions of increased intracranial pressure. (Circ Res 56: 20–30, 1985)
Previous studies of the relationship of intracranial pressure fluctuations to intrathoracic pressure have been confined to the intact circulation and, in general, assess transmission to the intracranial space of arterial pulse pressure or airway pressure during positive pressure ventilation with positive end-expiratory pressure (PEEP). Carotid pulse pressure ordinarily produces only very small changes in intracranial pressure (usually less than 5 mm Hg); these fluctuations in intracranial pressure are increased under conditions of increased intracranial elastance (Lofgren et al., 1973a; Guinan, 1975) but not by PEEP. PEEP produces changes in jugular venous pressure which correlate closely with (and seem to determine) changes in intracranial pressure (Luce et al., 1982). As in the case of carotid pulse pressure, the resulting intracranial pressure fluctuations are influenced by intracranial elastance and are usually small.

Insofar as CPR can be defined as a system characterized by rapid and wide fluctuations in intrathoracic pressure, the observations of Hamilton et al. (1944) on lumbar cerebrospinal fluid pressure during coughing may be more relevant to the subject of intracranial pressure fluctuations during CPR. They reasoned that jugular and carotid pressure fluctuations during cough were not responsible for the observed increase in spinal canal pressure. During cough, spinal pressure increased by more than twice the increase in jugular pressure, a relationship that ruled out jugular pressure changes as the sole determinant of changes in intracranial pressure. Whereas increases in peak arterial and spinal canal pressure were of similar magnitude during cough, Hamilton and colleagues argued that these pressures were unrelated to each other, because only a tiny fraction of arterial pulse pressure is normally reflected in the cerebrospinal fluid pressure. Augmentation of arterial pulse pressure by coughing should not alter this relationship. They concluded by postulating direct transmission of intrathoracic and intraabdominal pressure through the intervertebral foramina to the cerebrospinal fluid.

The present studies were designed to assess the extent of transmission of changes in intrathoracic pressure to the intracranial space during CPR in dogs. We also determined the influence of different modes of CPR, abdominal binding, and the level of intracranial pressure itself on the magnitude of pressure transmission. Finally, we examined the route of transmission of intrathoracic pressure to the intracranial space in terms of the contributions to intracranial pressure fluctuations made by the cerebrospinal fluid and arterial, jugular, and other venous blood-filled channels.

Methods

Forty-eight large mongrel dogs (22–35 kg) were anesthetized with ketamine (15–20 mg, im) and pentobarbital (15 to 20 mg/kg, iv) and intubated through a tracheostomy. Fluid-filled catheters were advanced to the right atrium and thoracic aorta via femoral cutdown, and to the carotid artery and jugular vein through side branches in the neck. Catheter position was confirmed by characteristic prearrest pressure tracings and by postmortem examination. Zero pressure was obtained by exposing the catheter tip to air in situ at the end of each experiment. Intracranial venous pressure was measured through a 16-gauge cannula inserted into the lateral ventricle via a Burr hole. When necessary, this cannula was flushed with mock cerebrospinal fluid prepared by adding 2.4 mEq of sodium bicarbonate to 100 ml of 0.9% saline. Intracranial venous pressure was measured through a 16-gauge cannula inserted into a drill hole at the confluence of the sagittal and transverse sinuses. Cannula position was confirmed by the free return of venous blood and by the observation that elevation of intracranial pressure by infusion of mock cerebrospinal fluid had no effect on pressure at the confluence, whereas elevation of pressure at the confluence by large saline bolus raised intracranial pressure. Pressures were measured with Statham P23Db and P23ID transducers.

All pressures and flows were recorded on a Brush Mark 600 recorder. In these studies, we did not measure intrathoracic pressure directly, because we wanted to avoid any possibility of modification of intrathoracic pressure fluctuations during CPR by instrumentation of the pleural space. Instead, changes in right atrial pressure were taken as equivalent to changes in intrathoracic (pleural) pressure based on previous studies which showed an extremely close correlation between these pressures during CPR (Chandra et al., 1981a, 1981b).

All dogs received heparin, 2 mg/kg, prior to arrest. In some animals (see below), a Biotronix BL-2045 cannulating electromagnetic flow probe was inserted in the carotid artery, and flow was measured with a Biotronix BL-613 sine wave flowmeter.

After measurement of control pressures and flows, ventricular fibrillation was induced by passing 60 Hz alternating current through a right ventricular pacing wire. External chest compression was initiated within 30 seconds of fibrillation with a Michigan Instruments pneumatic chest compressor (Thumper). During conventional CPR, the chest was compressed 2.5–8.0 cm at a rate of 60 compressions/min. Compression duration was 0.6 second. After every fifth chest compression, the lungs were inflated to a pressure of 33 mm Hg. Simultaneous compression-ventilation (SCV) CPR consisted of chest compression with the Thumper and synchronized ventilation with a pressure-limited ventilator, as previously described (Chandra et al., 1981b). Compression and ventilation were maintained for the first 50% of each compression-release cycle. Compression force and ventilation pressure were varied so that we might explore intracranial pressure fluctuations over a wide range of intrathoracic pressures. Except where otherwise noted, SCV-CPR was performed at a rate of 40/min. All animals were ventilated with room air using a Harvard volume cycled ventilator prior to arrest and with 95% oxygen and 5% carbon dioxide during CPR in order to prevent arterial Po2 from falling to very low levels during SCV-CPR (when there is marked hyperventilation). The possibility of increased cerebrovascular tone due to profound hypocarbia was in this way avoided.

Determinants of the Magnitude of Transmission of Intrathoracic Pressure to the Intracranial Space

The extent of transmission of intrathoracic pressure to the intracranial space was evaluated sequentially in three
Mechanism of Elevation of Intracranial Pressure

Role of Cerebrospinal Fluid

To examine the role of thoracolumbar cerebrospinal fluid in the transmission of intrathoracic pressure to the intracranial space, laminectomies were performed in seven animals at the level of the fourth cervical vertebral body. Heavy silk suture was placed loosely around the spinal cord and fed into a snare. CPR was performed at variable chest compression force and airway pressure to establish baseline intrathoracic-intracranial pressure relationships and then repeated with the snare drawn tight. Pressure in the thoracic cerebrospinal fluid was measured in six of these animals by inserting an 18-gauge plastic cannula into the subarachnoid space on the caudal side of the snare.

Role of Vascular Channels

The role of vascular channels in the transmission of intrathoracic pressure to the intracranial space was examined in eight dogs. Animals were instrumented as described above, and baseline intrathoracic-intracranial pressure relationships were established during a brief period of CPR. The animals then were defibrillated and exsanguinated by opening additional large bore arterial catheters to atmosphere. Upon cessation of cardiac activity, CPR was reinstituted. Intracranial pressure was maintained at baseline by infusing mock cerebrospinal fluid into the lateral ventricle, and intrathoracic-intracranial pressure relationships were studied again. We assume that, under these conditions of acute, profound hypovolemia, with carotid and jugular pressure near zero, the vascular contribution to intrathoracic-intracranial pressure relationships was minimized.

The contribution of arterial pressure to intracranial pressure was examined in five animals by advancing a balloon-tipped catheter to the ascending aorta under fluoroscopy. When inflated, this balloon completely occluded the aorta. This method permitted study of the effect of abolition of extrathoracic arterial pressure on intracranial pressure fluctuations.

The role of the jugular vein in the transmission of intrathoracic pressure to the intracranial space was examined in two ways. The first consisted of rapid fluctuation of airway pressure during SCV-CPR performed at slow rates (10/min). This maneuver permitted careful inspection of phase and amplitude relationships in the jugular and intracranial pressures. In a second approach, large bore plastic tubing was inserted into the external jugular vein and advanced across the venous valves at the thoracic inlet in the course of resuscitative efforts in four dogs. In this way, the influence of the ensuing marked increase in jugular pressure fluctuations on intracranial pressure was studied.

Finally, the role of venous channels other than the jugular was examined in seven dogs by measuring pressure at the confluence of the sagittal and transverse venous sinuses. Laminectomies were performed at the level of the fourth cervical vertebral body. A snare was passed around the cervical spinal cord and one of the two longitudinal venous sinuses which lie on the ventral side of the spinal cord. Pressure was measured in the lateral ventricle and in the confluence of the sinuses before and after tightening the snare.

Data Analysis

Data were analyzed on CLINFO (sponsored by NIH Division of Resources Grant 5 M01 RR 35-20). Regression lines were constructed by least-squares analysis. Significance levels for correlation coefficients of regression equations follow each equation in parentheses. Standard error of the estimate (Sy-x) also follows each equation, under the abbreviation SEE. Slopes of regression lines were compared by the method of Glantz (1981). Paired data were compared by t-test.

Results

Transmission of Intrathoracic Pressure to the Intracranial Space

Intrathoracic pressure was transmitted to the intracranial space in all animals studied. Figure 1 shows tracings obtained in one animal during conventional CPR and SCV-CPR. Carotid flow and pressures in jugular vein, aorta, right atrium, and lateral ventricle can be seen to rise and fall together in phase with each chest compression. Higher intrathoracic pressures were accompanied by increased intracranial pressure fluctuations during both forms of CPR. The relationship between changes in intrathoracic pressure and intracranial pressure during conventional CPR and SCV-CPR with the abdomen bound and unbound is plotted in Figure 2. The relationship between changes in intrathoracic pressure (ΔITP) and intracranial pressure (ΔICP) with the abdomen unbound was fit by the following linear equation:

\[ ΔICP (\text{mm Hg}) = 0.33 \times ΔITP (\text{mm Hg}) + 2.0 \]  \hspace{1cm} (1)

where \( r = 0.86 \) (\( P < 0.001 \)), \( \text{SEE} = 4.74 \), and \( ΔITP \) ranged from 10 to 90 mm Hg.

Abdominal binding, previously shown to increase carotid pressure and flow during CPR (Rudikoff et al., 1980), increased the extent to which intrathoracic pressure was transmitted to the intracranial space. With the abdomen bound, the relationship was fit by a linear equation with a significantly different (\( P < 0.01 \)) slope:

\[ ΔICP (\text{mm Hg}) = 0.52 \times ΔITP (\text{mm Hg}) + 2.7 \]  \hspace{1cm} (2)

with \( r = 0.84 \) (\( P < 0.001 \)), and \( \text{SEE} = 7.45 \).

During conventional CPR, the relationships for intracranial and intrathoracic pressure changes were similar.

Transmission of Intrathoracic Pressure to the Intracranial Space

The Role of Baseline Intracranial Pressure

To examine the influence of baseline intracranial pressure on the extent of transmission of intrathor-
Intracranial pressure to the intracranial space, mock cerebrospinal fluid was infused into the lateral ventricle, and SCV-CPR was performed with both normal and elevated baseline intracranial pressure in seven dogs. Elevation of baseline intracranial pressure increases the extent of transmission of intrathoracic pressure to the intracranial space. For baseline intracranial pressure between 12 and 16 mm Hg, the relationship between fluctuations in intrathoracic and intracranial pressure was fit by the equation:

\[ \Delta \text{ICP (mm Hg)} = 0.40 \Delta \text{ITP (mm Hg)} + 5.9 \]  

where \( r = 0.91 \) (\( P < 0.001 \)), and \( \text{SEE} = 3.71 \).

Elevation of baseline intracranial pressure to the range of 22–30 mm Hg changed the relationship (\( P < 0.001 \) vs. Equation 3) to:

\[ \Delta \text{ICP (mm Hg)} = 0.77 \Delta \text{ITP (mm Hg)} + 2.4 \]  

where \( r = 0.95 \) (\( P < 0.001 \)) and \( \text{SEE} = 5.21 \).

**Mechanism of Transmission of Intrathoracic Pressure to the Intracranial Space**

**Role of the Cerebrospinal Fluid**

To evaluate the role of the cerebrospinal fluid as a medium by which intrathoracic pressure is transmitted to the intracranial space, pressure was measured in the thoracic subarachnoid space (\( \Delta \text{CSF}_T \)) and lateral ventricle before and after spinal cord ligation in seven animals. Prior to cord ligation, pressure fluctuations were similar in the thoracic and intracranial cerebrospinal fluid:

\[ \Delta \text{CSF}_T \text{ (mm Hg)} = 0.33 \Delta \text{ITP (mm Hg)} + 2.9 \]  

where \( r = 0.88 \) (\( P < 0.001 \)) and \( \text{SEE} = 3.49 \), and

\[ \Delta \text{ICP (mm Hg)} = 0.32 \Delta \text{ITP (mm Hg)} + 3.2 \]  

where \( r = 0.91 \) (\( P < 0.001 \)) and \( \text{SEE} = 2.67 \).

Following cord ligation, these relationships were no longer equivalent (\( P < 0.002 \)):

\[ \Delta \text{CSF}_T \text{ (mm Hg)} = 0.40 \Delta \text{ITP (mm Hg)} + 3.2 \]  

where \( r = 0.76 \) (\( P < 0.001 \)) and \( \text{SEE} = 6.34 \),

\[ \Delta \text{ICP (mm Hg)} = 0.22 \Delta \text{ITP (mm Hg)} + 2.4 \]  

where \( r = 0.82 \) (\( P < 0.001 \)) and \( \text{SEE} = 2.78 \).

Intracranial pressure fluctuations thus decreased...
substantially (slope of equation 6 ≠ slope of equation 8, P < 0.01) following spinal cord ligation, indicating that cerebrospinal fluid is one medium by which intrathoracic pressure is transmitted to the intracranial space.

Mechanism of Transmission of Intrathoracic Pressure to the Intracranial Space

Role of Vascular Channels

The possibility of transmission of intrathoracic pressure to the intracranial space along vascular channels was investigated in several ways. First, eight dogs were fibrillated and SCV-CPR was performed briefly with the abdomen unbound to establish baseline intrathoracic-intracranial pressure relationships. The animals were then defibrillated, exsanguinated, and CPR was resumed. Prior to exsanguination, the relationship between intracranial and intrathoracic pressure fluctuations was

\[ \Delta \text{ICP} (\text{mm Hg}) = 0.31 \Delta \text{ITP} (\text{mm Hg}) + 1.5 \]  

where \( r = 0.95 \) (P < 0.001) and see = 1.36.

After exsanguination, baseline intracranial pressure fell almost to zero and intracranial pressure fluctuations were virtually abolished. Because of the previously described dependence of intracranial pressure fluctuations on baseline pressure, mock cerebrospinal fluid was infused into the lateral ventricle until the original intracranial pressure was restored. Under conditions of acute hypovolemia with normal baseline intracranial pressure, the relationship was:

\[ \Delta \text{ICP} (\text{mm Hg}) = 0.06 \Delta \text{ITP} (\text{mm Hg}) + 1.8 \]  

where \( r = 0.56 \) (P < 0.01) and see = 1.55.

This slope is less than that of Equation 9 (P < 0.001). Thus, even after restoration of normal baseline intracranial pressure, transmission of intrathoracic pressure to the intracranial space was markedly reduced by removal of blood from vascular channels. This indicates that blood-filled channels are necessary for the majority of pressure transmission.

To examine the contribution of extrathoracic arterial pressure fluctuation to changes in intracranial pressure, a balloon-tipped catheter large enough to occlude the aorta completely, when inflated, was advanced to the ascending aorta under fluoroscopy in five animals. In all animals, carotid pressure and flow fell immediately to zero with balloon inflation, but intracranial pressure fluctuations were not affected. Figure 3 shows a tracing during balloon inflation in one of the five animals. Intrathoracic pressure is not transmitted to the intracranial space along arterial channels.

The jugular veins were also examined as possible routes for transmission of intrathoracic pressure to the intracranial space during CPR. The rate of jugular pressure rise often lagged behind the rate of rise in intracranial pressure (Fig. 3) and marked discrepancies between peak jugular and intracranial pressures were occasionally observed. Because these findings suggested that jugular pressure changes did not determine intracranial pressure, two maneuvers were studied in an attempt to clearly dissociate intracranial and jugular pressure fluctuations.

The first maneuver consisted of rapid fluctuation of airway pressure (and, thus, intrathoracic pressure) during SCV-CPR performed at slow rates. In seven animals with the abdomen bound and high baseline intracranial pressure (21.6 ± 5.0 mm Hg), rapid fluctuation of airway pressure produced changes in intrathoracic pressure of 37.7 ± 5.5 mm Hg. During this same maneuver, intracranial pressure changed by 23.3 ± 8.4 mm Hg, whereas jugular pressure changed by only 2.4 ± 2.7 mm Hg (P < 0.001) (Fig. 4).
In the second maneuver, large bore plastic tubing was inserted into the external jugular vein and advanced across the venous valves at the thoracic inlet in the course of resuscitative efforts in four dogs. This maneuver resulted in a rise in jugular venous pressure to the level of right atrial pressure but had no effect on intracranial pressure (Fig. 5). Postmortem dissection of the jugular veins of these animals revealed a more distal set of venous valves at the level of the mandible in each animal, as previously described by Hagedes and Shackleford (1965).

Because jugular and intracranial pressures were not closely correlated and could be easily dissociated, pressure transmission along venous channels other than the jugular was examined in an additional seven dogs during SCV-CPR. Pressure fluctuations were measured at the confluence of the sagittal and transverse sinuses (ΔCon) as well as in the lateral ventricle and thoracic subarachnoid space before and after cord ligation. Prior to cord ligation, the relationships between ΔITP, ΔICP, and ΔCon were fit by the equations (Fig. 6a):

\[ ΔICP (\text{mm Hg}) = 0.32 \times ΔITP (\text{mm Hg}) + 3.2 \quad (6) \]

where \( r = 0.91 \) (\( P < 0.001 \)) and \( S\bar{E}E = 2.67 \).

\[ ΔCon (\text{mm Hg}) = 0.21 \times ΔITP (\text{mm Hg}) + 3.5 \quad (11) \]

where \( r = 0.81 \) (\( P < 0.001 \)) and \( S\bar{E}E = 2.88 \).

Although quantitatively less than intracranial pressure changes (slope of Equation 6 \( \neq \) slope of Equation 11, \( P < 0.001 \)), the phase and direction of pressure changes at the confluence of the sinuses were always similar to intracranial pressure changes. This contrasts with the previously noted discrepancies between jugular and intracranial pressure changes and suggested that fluctuations at the confluence of the intracranial sinuses reflected the vascular contribution to intracranial pressure changes. After cord ligation, (Fig. 6b) with ΔITP again ranging from 10 to 80 mm Hg, the relationship was fit by a new set of equations:

\[ ΔICP (\text{mm Hg}) = 0.22 \times ΔITP (\text{mm Hg}) + 2.4 \quad (8) \]

where \( r = 0.82 \) (\( P < 0.001 \)) and \( S\bar{E}E = 2.78 \).
\Delta \text{Con (mm Hg)} = 0.22 \Delta \text{ITP (mm Hg)} + 2.2 \quad (12)

where \( r = 0.84 \) (\( P < 0.001 \)) and \( \text{SEE} = 2.41 \).

These equations do not differ significantly from each other. Thus, once pressure transmission via cerebrospinal fluid was abolished by spinal cord ligation, intracranial pressure changes were dependent on and equivalent to changes in pressure at the confluence of the sinuses (Fig. 7).

Although pressure fluctuations in the thoracic cerebrospinal fluid did not increase significantly after cord ligation with the abdomen unbound, they did increase (\( P < 0.01 \)) following cord ligation with the abdomen bound. This finding suggested the possibility of direct transmission of intrathoracic pressure to the thoracic subarachnoid space (in addition to pressurization of thoracic cerebrospinal fluid by blood in paraspinal veins which anastomose with the intercostal and azygous systems). To examine this process, pressure fluctuations were measured in the intracranial and thoracic subarachnoid spaces and at the confluence of the venous sinuses after exsanguination. Following exsanguination and prior to cord ligation,

\[ \Delta \text{CSF}_T \text{ (mm Hg)} = 0.06 \Delta \text{ITP (mm Hg)} + 2.7 \quad (13) \]

where \( r = 0.73 \) (\( P < 0.001 \)) and \( \text{SEE} = 1.0 \).

After cord ligation pressure changes in the thoracic cerebrospinal fluid were increased (\( P < 0.05 \)) to:

\[ \Delta \text{CSF}_T \text{ (mm Hg)} = 0.11 \Delta \text{ITP (mm Hg)} + 3.3 \quad (14) \]

where \( r = 0.91 \) (\( P < 0.001 \)), \( \text{SEE} = 1.1 \), and \( \Delta \text{ITP} \) ranged from 10 to 80 mm Hg.

Because exsanguination virtually abolished pressure changes at the confluence of the venous sinuses, thoracic cerebrospinal fluid pressure fluctuations in the absence of vascular pressure fluctuations
indicate direct transmission of intrathoracic pressure to the spinal canal.

Finally, to determine whether the longitudinal vertebral veins participate in pressure transmission, a snare was passed around one of the two veins and the spinal cord. Before the snare was tightened,

$$\Delta \text{ICP (mm Hg)} = 0.31 \Delta \text{ITP (mm Hg)} + 3.0 \quad (15)$$

where $r = 0.94 \ (P < 0.001)$ and $\text{SEE} = 2.2$.

$$\Delta \text{Con (mm Hg)} = 0.23 \Delta \text{ITP (mm Hg)} + 3.6 \quad (16)$$

where $r = 0.89 \ (P < 0.001)$ and $\text{SEE} = 2.2$.

After the snare was tightened around the cervical cord and one of the longitudinal vertebral veins adjacent to the cord,

$$\Delta \text{ICP (mm Hg)} = 0.10 \Delta \text{ITP (mm Hg)} + 4.8 \quad (17)$$

where $r = 0.66 \ (P < 0.01)$ and $\text{SEE} = 2.3$.

$$\Delta \text{Con (mm Hg)} = 0.09 \Delta \text{ITP (mm Hg)} + 5.0 \quad (18)$$

where $r = 0.62 \ (P < 0.01)$ and $\text{SEE} = 2.5$.

Once again, intracranial venous sinus and lateral ventricular pressure changes were equivalent after obstruction of cerebrospinal fluid flow. However, with simultaneous ligation of one of the longitudinal vertebral veins, these pressure changes were significantly less than intracranial venous sinus pressure prior to ligation. The result obtained by cord ligation alone, after which pressure fluctuations in the lateral ventricle and the confluence of intracranial pressure changes at transmission of pressure were increased by cord ligation, suggesting direct transmission of intrathoracic pressure to the thoracic subarachnoid space.

Discussion

These studies demonstrate that elevation of intracranial pressure is a consistent feature of CPR. At each of the baseline intracranial pressures studied, the proportion of intrathoracic pressure transmitted to the intracranial space was relatively constant over a wide range of changes of intrathoracic pressure during both conventional and SCV-CPR. In animals with normal baseline intracranial pressure, about one-third of the change in intrathoracic pressure was transmitted to the intracranial space during CPR with the abdomen unbound and about one-half of the intrathoracic pressure change was transmitted to the intracranial space during CPR with the abdomen bound.

Baseline intracranial pressure is a critical determinant of the extent to which intrathoracic pressure is transmitted to the intracranial space. When baseline intracranial pressure was raised from a range of 12 to 16 mm Hg to a range of 22 to 30 mm Hg by
infusing a mock solution of cerebrospinal fluid into the lateral ventricle, transmission of intrathoracic pressure to the intracranial space nearly doubled (Fig. 3). The fact that transmission of intrathoracic pressure to the intracranial space is increased by raising intracranial pressure is in keeping with current understanding of intracranial elastance in both animals and humans in the non-arrest situation (Lofgren et al., 1973; Lofgren and Zwetnow, 1973; Guinane, 1975; Avezaat et al., 1979; Burchiel et al., 1981). Increased intracranial elastance is also a likely explanation for the marked increase in intracranial pressure reported by Rogers and colleagues (1979) during CPR in a patient with cerebral edema.

Vascular channels are responsible for the bulk of transmission of intrathoracic pressure to the intracranial space, as demonstrated by studies in which exsanguination reduced intracranial pressure fluctuations to low levels despite maintenance of baseline intracranial pressure. Ligation of the cervical spinal cord also decreased intracranial pressure fluctuations, but by a lesser amount than exsanguination. Because this maneuver eliminated pressure transmission by the cerebrospinal fluid, it indicates that cerebrospinal fluid also contributes to intracranial pressure fluctuations during CPR. In addition, thoracic cerebrospinal fluid pressure, in equilibrium with intracranial pressure before cord ligation, was increased following cord ligation (Fig. 7). The increase in thoracic cerebrospinal fluid pressure produced by cervical cord ligation, present even during acute, profound hypovolemia, demonstrates that intrathoracic pressure is transmitted to the spinal canal directly through intervertebral foramina as well as via vascular channels.

A major goal of these experiments was the identification of the specific vascular routes of transmission of intrathoracic pressure to the intracranial space. Intracranial pressure fluctuations were unaffected by ablation of carotid (and all other cephalic arterial) pressure changes during aortic occlusion. Clearly, then, the large arteries of the neck do not transmit intrathoracic pressure to the brain. The dissociation of jugular venous pressure and intracranial pressure can be explained by the presence of competent venous valves at the thoracic inlet and mandible (Hegedus and Shackleford, 1965; Niemann et al., 1980; Fisher et al., 1981), which prevented retrograde pressure transmission.

That neither the extrathoracic arteries nor the jugular veins account for transmission of intrathoracic pressure to the intracranial space does not rule out other vascular channels as routes of pressure transmission. As noted above, when CPR was performed at constant baseline intracranial pressure, exsanguination eliminated approximately 80% of intracranial pressure fluctuations. This result demonstrates that blood-filled vascular channels are essential for transmission of intrathoracic pressure to the intracranial space. Direct evidence in support of non-valved veins in the transmission of intrathoracic pressure to the intracranial space was provided by studies in which pressure was measured at the confluence of the transverse and sagittal sinuses. Before cord ligation, pressure changes at the confluence accounted for most of the intracranial pressure change (Eq. 11). After cord ligation, pressure changes at the confluence were unchanged and accounted for all of the intracranial pressure fluctuation (Eq. 12).

We did not examine directly blood pressure or flow in the vertebrobasilar system or the longitudinal vertebral veins. Nevertheless, these veins, which are not known to contain valves, must be regarded as loci for transmission of intrathoracic pressure to the intracranial space during CPR. The longitudinal vertebral veins are particularly attractive in this regard. Situated inside the vertebral column, the longitudinal vertebral veins communicate extensively with the azygous and intercostal veins and have direct access to the intracranial space via the foramen magnum (Reinhard et al., 1962) Indeed, when one of the two longitudinal sinuses were ligated, pressure fluctuations at the confluence of the sinuses and in the lateral ventricle fell.

In summary, at normal baseline intracranial pressure, a relatively constant proportion of intrathoracic pressure is transmitted to the intracranial space. The majority of pressure transmission occurs along venous channels other than the jugular. The longitudinal vertebral veins are the most important of several possible channels. Substantial pressure transmission is also mediated by cerebrospinal fluid as a consequence of transmission of intrathoracic pressure ITP to the thoracic CSF, most likely via the intervertebral foramina. Increased pressure transmission during abdominal binding is presumably due to the fact that abdominal binding increases lumbar spinal fluid pressure as well as intraabdominal pressure. Caudal run-off of blood and spinal fluid during chest compression is thereby impeded.

Increases in intracranial pressure during ventilation with positive end-expiratory pressure (PEEP) in dogs have been attributed to passive elevation of jugular venous pressure (Luce et al., 1982). These findings do not necessarily conflict with our own observations. Unlike PEEP, which is applied at low levels for long periods of time in animals with normal carotid and jugular flow, CPR consists of the application of high pressure for short time intervals with low blood flows. In relation to the brain, the jugular vein during CPR is a downstream segment, compartmentalized by valves, with a compliance great enough so that the small amount of antegrade and retrograde blood flow is accommodated with a pressure rise which may be considerably less than intracranial pressure fluctuations. The brevity of increases in intrathoracic pressure during CPR may
not permit equilibration of jugular pressure with pressure in those non-valved veins which do transmit intrathoracic pressure to the intracranial space. This contrasts sharply with studies of PEEP, in which increases in intrathoracic venous pressure are so modest and so slow that the jugular valves may not close. If so, the jugular vein would remain in equilibrium with other craniovertebral veins during PEEP. As these studies demonstrate, this equilibrium is not necessarily reached during CPR.

The adequacy of cerebral blood flow during CPR has been an issue of major concern ever since the development of modern resuscitative techniques. Two recent papers (Koehler et al., 1983, Michael et al., 1984) show a clear correlation between cerebral perfusion and cerebral perfusion pressure gradients (mean carotid artery pressure—mean intracranial pressure) over a wide range of pressures and experimental conditions. Peak intrathoracic pressure ranged from 56 to 101 mm Hg during conventional and SCV-CPR in these studies, and mean carotid pressure varied from as little as 17 mm Hg during SCV-CPR with carotid collapse, to 71 mm Hg during SCV-CPR after collapse, had been reversed by epinephrine. In these studies, intracranial pressure fluctuations were minimally affected by carotid collapse for reasons made clear by Figure 3: intracranial pressure changes during CPR are unrelated to carotid arterial pressure. Intracranial pressure fluctuations were substantially increased by abdominal binding in the study by Michael et al. (1984), but this phenomenon did not alter the fundamental relationship between cerebral perfusion pressure and actual cerebral blood flow.

The major goals of CPR are maintenance of cerebral and coronary perfusion at levels compatible with cell survival until spontaneous effective circulation is restored. Viewed from the perspective of generation of cerebral perfusion pressure gradients, elevation of intrathoracic pressure is a two-edged sword. Transmission of intrathoracic pressure to the intracranial space, which limits cerebral flow, is ever present and increased by abdominal binding and under conditions of elevated baseline intracranial pressure. Nevertheless, elevation of intrathoracic pressure is a sword which may be wielded to advantage. Intracranial pressure fluctuations are proportional to intrathoracic pressure changes but unrelated to carotid artery pressure during CPR. Therefore, efforts designed to maintain peak carotid pressure at or near peak intrathoracic pressure should maximize the cerebral perfusion pressure gradient for any given change in intrathoracic pressure. Furthermore, to the extent that the efficiency of transmission of intrathoracic pressure to the carotid artery can be maximized, efforts aimed at the safe elevation of intrathoracic pressure to levels above those attained during conventional CPR will be rewarded by increased cerebral blood flow.

INDEX TERMS: Cardiopulmonary resuscitation • Resuscitation • Intracranial pressure
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