Cerebrospinal Fluid Pressure in the Anesthetized Rat

Brian S. Budgell, PhD,[a] and Philip S. Bolton, PhD[b,c]

Abstract

Objective: The primary aims of this study were to determine the major frequencies and powers of oscillations in cerebrospinal fluid (CSF) pressure in the anesthetized rat, and determine whether the CSF pressure oscillations correlated with the major oscillation frequencies in the cardiovascular and respiratory systems as proposed by some chiropractic theories.

Methods: The cardiac and ventilatory cycles, and CSF pressure were simultaneously recorded during spontaneous and positive-pressure mechanical ventilation in the anesthetized rat. Power spectra were generated from the raw data to identify the major oscillation frequencies in cardiorespiratory and CSF data sets. Entrainment of CSF pressure with ventilation was tested by mechanically pacing the ventilation over a range of frequencies.

Results: The most powerful oscillation in CSF pressure was coincident with ventilatory chest movement during both spontaneous and mechanically paced ventilation. In 22 of 26 trials, there was also a very weak oscillation in CSF pressure that was entrained to heart rate. In addition, in 21 of 26 trials, it was possible to identify a low-frequency oscillation (<0.25 Hz) in CSF pressure that was coincident with a low-frequency oscillation in the power spectrum of the cardiac cycle.

Conclusions: This study suggests oscillations in CSF pressure in the anesthetized rat are entrained to and driven by ventilation. The arterial pulse pressure makes little contribution to oscillations in CSF pressure in the immobile, anesthetized rat. This study provides normative, quantitative data on which to develop studies concerning the effects of vertebral movements and spinal posture on CSF dynamics. (J Manipulative Physiol Ther 2007;30:351-356)

Key Indexing Terms: *Rat; Cerebrospinal Fluid; Chiropractic*

The clinical application of manual therapies such as cranial and spinal manipulation is guided by certain fundamental clinical hypotheses and theories concerning the causes of disease and the physiologic impact of the involved treatments. Indeed, at times such hypotheses and theories may seem to hold more importance than objective and quantitative measures of clinical effects. Many of these clinical hypotheses and theories remain to be rigorously tested.

Among theories at the core of certain manual therapies are hypotheses that altered motion or malposition of the cranial bones or vertebrae interfere with normal neurologic function and thus result in disease.[1-3] Different iterations of these core theories may evoke somewhat different and perhaps complementary pathologic mechanisms such as direct pressure on neural structures, tension on meninges, or disturbances in normal cerebrospinal fluid (CSF) movement.[3-5] With regard to the latter, it has been proposed that rhythmic oscillations, both normal and abnormal, in CSF pressure may result in palpable movements of the cranial and vertebral bones, sometimes termed the cranial rhythmic impulse (CRI), and that this CRI may therefore have some application to diagnosis. However, little data exist concerning the normal frequencies of the CRI, and it is uncertain whether oscillations of CSF pressure actually result in a CRI that is palpable on manual examination.[6,7]

Previously, it has been possible to measure lumbar CSF pressure in anesthetized rats,[8] and to demonstrate large absolute pressure changes associated with cerebral hemorrhage. However, it is unclear whether relatively small changes in CSF pressure at the cervical level (or higher) might be detectable at the lumbar level given the normal

[a] Associate Professor, School of Health Sciences, Faculty of Medicine, Kyoto University, Kyoto, Japan.
[b] Associate Professor, School of Biomedical Sciences, Faculty of Health, University of Newcastle, Callaghan, NSW 2308, Australia.
[c] Hunter Medical Research Institute, New Lambton, NSW Australia.
Submit requests for reprints to: Philip S. Bolton, PhD, Associate Professor, School of Biomedical Sciences, Faculty of Health, University Drive, Callaghan, NSW 2308, Australia
(e-mail: *Philip.Bolton@newcastle.edu.au*).
Paper submitted September 12, 2006; in revised form February 20, 2007; accepted March 27, 2007.
0161-4754/$32.00

doi:10.1016/j.jmpt.2007.04.002

oscillations in CSF pressure at this level. In other words, there may be too much noise in the normal lumbar CSF pressure recording to permit detection of a subtle pressure signal from the more rostral spine or cranium. Furthermore, even in the prone animal, pressure measures at the lumbar level may be affected by, for example, small changes in head position,[9] and hence, in absolute terms, lumbar CSF pressure cannot be equated with intracranial pressure. Thus, in the absence of quantitative data on normal oscillation frequencies, it would be difficult to identify changes in CSF fluid dynamics associated with vertebral movement or static malposition at any spinal level, and no conclusions can be made about any relationship between lumbar CSF oscillations and the putative CRI.

Although animal models have been developed to study the effects of spinal joint fixation on vertebral morphology,[10] and the effects of vertebral motion and manipulation on paravertebral muscle spindle behavior,[11] there are currently no available animal models to study the effects of vertebral movement or position on CSF dynamics. The aim of this study was to quantify the frequencies and relative strengths (powers) of oscillations in CSF pressure in the anesthetized rat as a first step in developing a model to investigate the effects of vertebral displacements and manipulations on CSF dynamics.

Methods

Experiments were performed on 8 urethane-anesthetized adult male Wistar rats aged 8 to 12 weeks and weighing 360 to 430 g. All procedures were performed in accordance with protocols approved by the University of Newcastle animal care and ethics committee, and conformed to the Australian National Health and Medical Research Council Code for Practice for the use of animals in experiments.

Anesthesia was induced by an intraperitoneal injection of urethane (1.3 g/kg). Animals were then tracheotomized and intubated, and initially allowed to ventilate spontaneously. A carotid artery was catheterized for the continuous measurement of blood pressure (BP) and heart rate (World Precision Instruments, BP-1, Sarasota, Fla), and a jugular vein was catheterized for administration of supplementary anesthetic and fluids as necessary. Respiratory rate was monitored by a pressure transducer (MP100, PowerLab, ADInstruments, Castle Hill, Australia) placed under the chest to measure chest excursion. In trials involving positive-pressure mechanical ventilation (see later), animals were ventilated at rates of between 65 and 100 breaths per minute using a small animal ventilator (Small Animal Ventilator Model 683, Harvard Apparatus, South Natick, Mass) with a spirometer (MLT1L Spirometer, ADInstruments) placed in series with the ventilator to monitor ventilatory flow. No paralyzing agent was used in these experiments. This allowed some animals, which remained in good physiologic condition, to be returned to spontaneous ventilation. The electrocardiogram (EKG) was recorded from subcutaneous electrodes on each side of the chest. The EKG served as a backup for monitoring the cardiac cycle in the event, as sometimes happens in prolonged experiments, that the carotid cannula became obstructed. Body temperature was monitored via a rectal probe (Model 43TA; Yellow Springs Instrument Company, Yellow Springs, Ohio) and maintained at 37°C to 37.5°C by using a heating blanket (K-20-D, American Pharmaseal Company, Valencia, Calif) and infrared lamp. Adequacy of anesthesia was judged by stability of heart rate and BP, and by periodically checking for the absence of withdrawal and corneal reflexes.

A catheter (PE10, Atom Medical, Tokyo) was introduced into the subarachnoid space of the lower lumbar spine via a partial laminectomy of the L5 vertebra by using an established protocol.[8] Specifically, the caudal half of the L5 lamina was surgically exposed and removed along with the yellow ligament, leaving the dura mater intact. A small incision was made in the dura mater, and through this incision a 25-gauge needle was inserted and used to make a small puncture in the arachnoid mater caudal to the termination of the spinal cord. Under direct visualization with a dissecting microscope (OPMI-11, Carl Zeiss, Sydney, Australia), approximately 5 mm of PE10 tubing was fed into the subarachnoid space, and the incision in the arachnoid mater was sealed with tissue adhesive (Quick Fix, Shelley Pty Ltd Padstow, NSW, Australia). The catheter was filled with artificial CSF (aCSF)[12] from a reservoir in a side port and connected to a pressure transducer for continuous measurement of lumbar CSF pressure. The EKG, BP, CSF pressure, ventilatory flow, and chest excursion were recorded on a computer (G3 Apple Macintosh) and analyzed by using a dedicated data acquisition system (MacLab 8S, ADInstruments). Mean arterial pressure and mean CSF pressure were computed by using the data acquisition system and displayed in real-time to monitor the stability of the preparation.

Data were sampled at a rate of 2000 Hz (EKG, BP, and CSF pressure) or 200 Hz (chest excursion and ventilatory flow), and analyzed using a dedicated software data analysis system (Chart ver 4.1.1, ADInstruments). Data were obtained from a total of 26 trials performed in 8 animals. Each trial consisted of a 4-minute recording period with ventilation at a stable rate, during which time systolic BP remained higher than 80 mm Hg and a stable anesthetic plane was maintained. Twelve trials were performed with spontaneous breathing and 14 trials were performed in animals that were mechanically ventilated (see above) at rates of between 65 and 100 breaths per minute. Hence, in all animals, recordings were obtained during both spontaneous and mechanical ventilation, and in some animals, it was possible to obtain records during more than 1 rate of mechanical ventilation. Four-minute blocks of data were recorded for analysis of power spectra to capture a minimum

of 260 respiratory cycles even at the slowest rate of mechanical ventilation. Power spectra were generated using 64K or 128K fast Fourier transforms with a Hamming window (Chart ver 4.1.1, ADInstruments).

Data were analyzed as follows: peak frequencies in CSF pressure, revealed by power spectrum analysis, were examined for correlation with peak frequencies in the power spectra of the respiratory and cardiac cycles, thus including, but not limited to, the mean respiratory rate and mean heart rate. Furthermore, simultaneous recordings of CSF pressure waves and either chest excursion or BP were averaged (n = 500 ventilatory or cardiac cycles) using commercial software (Scope ver 3.0, ADInstruments) to determine the phase relationship between these waves.

In 6 of 8 animals, after paced respiration, the upper cervical vertebrae were exposed, a 26-gauge needle was introduced into the subarachnoid space at the occiput-C1 level, and the entry site was then sealed with surgical adhesive (Quick Fix, Shelley's). Discrete volumes (range, 0.05-0.31 mL) of aCSF were introduced at the occipital-C1 level while concurrently measuring the lumbar CSF pressure. The purpose of this protocol was to confirm that pressure changes at the upper cervical level were detectable via the lumbar catheter. In 3 rats, at the conclusion of the experiment, blue food dye was mixed (1% Solution) with aCSF and injected (typically 0.25 mL) at the occipital-C1 level to determine if the lumbar cannular was placed in the subarachnoid space.

Results

In all 6 of 8 animals tested, injection of small volumes of aCSF into the cisterna magna resulted in essentially instantaneous increases in CSF pressure measured at the lumbar level, indicating that the lumbar catheter was patent. In all 3 rats in which dye was injected into the cisterna magna, dye was found to track in the subarachnoid space at the level of the lumbar cannula and could be aspirated with the lumbar catheter, demonstrating that the catheter was within the subarachnoid space.

Frequencies of CSF Pressure Oscillations

At the commencement of data collection, the mean (±SD) CSF pressure for all spontaneously breathing rats (n = 8) was 4.18 ± 2.49 mm Hg. All 8 rats exhibited pressure waves in the CSF (Fig 1A, inset). The power spectra of CSF pressure, in both spontaneously ventilating and mechanically ventilated rats, showed peaks within 3 frequency ranges: 0.006 to 0.24 Hz (7/8 rats—in 9 of 12 trials with spontaneous breathing, and 12 of 14 trials with mechanical ventilation); 0.98 to 1.95 Hz (8/8 rats); 5.37 to 8.06 Hz (8/8 rats) (Fig 1A). The mean (±SD) of the peaks in each of the frequency ranges, for all rats, was 0.18 ± 0.07, 1.53 ± 0.36, and 6.99 ± 0.08 Hz, respectively.

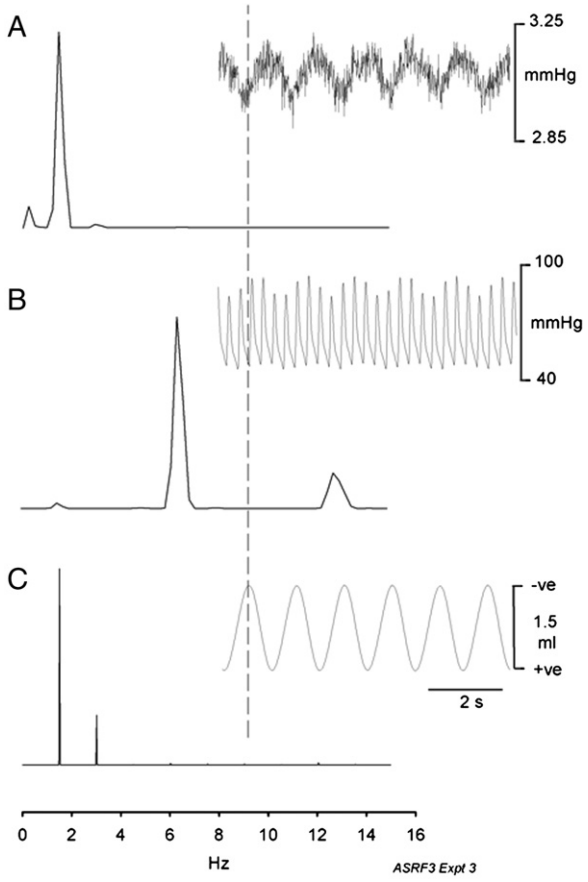

Fig 1. *Power spectra of simultaneously recorded 4-minute epochs of CSF pressure (A), BP (B), and chest excursion (C) in a spontaneously breathing anesthetized rat. The insets, top right of each panel, show 8-second epochs of raw data for the CSF pressure, BP, and chest excursion, respectively. The dashed line marks the time of peak inspiration. The principal peaks in the CSF pressure spectrum occur at 0.122 and 1.251 Hz. Principal peaks in the BP spectrum occur at 1.251 Hz (average instantaneous respiratory rate) and 6.042 Hz (average instantaneous heart rate). The larger CSF power spectrum peak (A) clearly coincides with the larger peak power spectrum of chest excursion (C). There is also a second (harmonic) peak at 2.5 Hz in the spectral analysis chest excursion (C). In this animal, the CSF peak at 0.122 Hz (A) correlated with a very small peak in the BP power spectrum, which cannot be discerned in this figure.*

Powers of CSF Pressure Oscillations

The CSF power spectra were reanalyzed with 8K fast Fourier transforms, effectively creating bins of 0.25 Hz in width. This second analysis, using a broader bin width, does not allow as precise an identification of peak frequency, but pools power over the bin width for a more efficient comparison of powers within the ranges sampled. In 25 of 26 trials, the largest peak in the power spectrum of CSF pressure was that peak within the frequency range of 0.98 to 1.95 Hz. The powers of the 3 peaks in each CSF pressure spectrum were normalized to the power of the major peak in the 0.98 to 1.95 Hz range. Within the same trials, the peaks

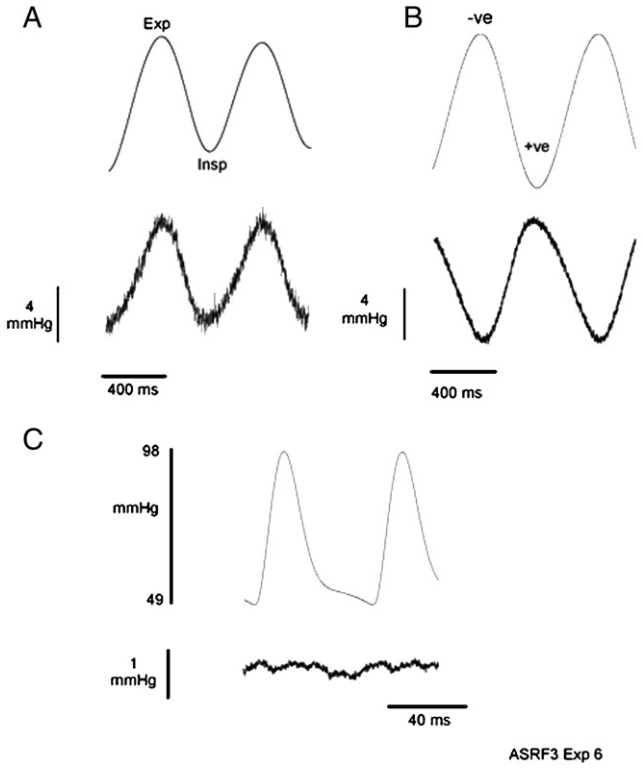

Fig 2. *This figure shows averaged records (n = 500) of chest excursion (upper panel) and CSF pressure (lower panel) over 2 cycles of spontaneous breathing (A) and mechanical ventilation (93 breaths/min) (B). Averages (n = 500) of BP (upper panel) and CSF pressure (lower panel) are shown during spontaneous breathing (C). These data are from the same animal and clearly show that CSF pressure waves are entrained to ventilatory movement. With spontaneous ventilation the peak pressure in CSF occurs at peak expiration (Exp), whereas minimum pressure occurs at peak inspiration (Insp) (A). Note that the phase of CSF pressure shifts 180° with mechanical ventilation (B) and is greatest with positive pressure ventilation (+ve) and least with negative (−ve) pressure ventilation. There was no apparent entrainment of CSF pressure (lower panel) with BP (upper panel) in this rat (C).*

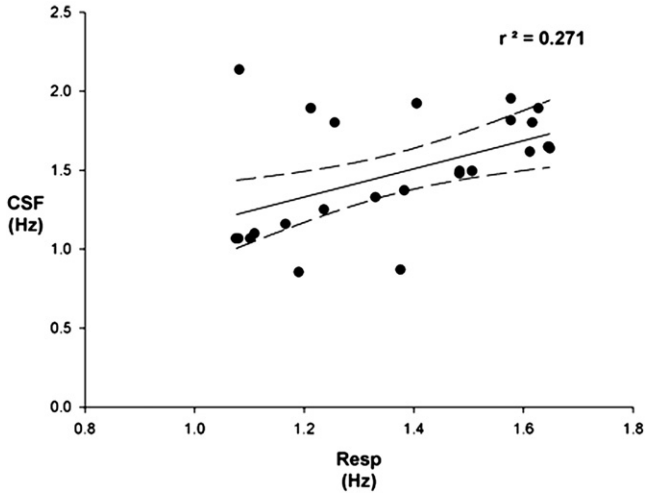

Fig 3. *Frequency of the most powerful oscillation in the CSF pressure (CSF) plotted against the frequency of ventilation (Resp) of both spontaneous and mechanically ventilated rats (26 trials in 8 rats; some data points overlap) in this study. The "linear" regression line (solid line) and 95% confidence intervals (dashed lines) have been plotted. The "linear" correlation (coefficient of determination = r^2) was 0.271.*

in the power spectra of CSF pressure within the 5.37 to 8.06 Hz range had less than 1% of the power of the peaks in the 0.98 to 1.95 Hz range. Similarly, in 21 of 26 trials, the low frequency peak (below 0.25 Hz) in CSF pressure had a power of less than 8% of the power of the major peak in the 0.98 to 1.95 Hz range. In the remaining 3 trials, the powers of these low frequency peaks (below 0.25 Hz) were 27%, 37%, and 285% of the power of the major peak in the 0.98 to 1.95 Hz range.

Entrainment of CSF Pressure Oscillations to Cardiac and Respiratory Cycles

Power spectra analysis of the EKG and BP demonstrated coincident major peaks within the frequency ranges of 5.402 to 8.392 Hz. Minor peaks also occurred at successive, higher harmonic frequencies (Fig 1B). In 4 animals, there was also a small peak in the BP power spectra within the range of 1.068 to 1.648 Hz. The BP power spectra invariably included more "noise" than the EKG power spectra, and the major peaks in the BP power spectra were relatively wide compared to the coincident peaks in the EKG spectra because of this noise. In 21 of 26 trials, it was possible to resolve a relatively small, low frequency peak below 0.25 Hz in the EKG power spectra.

The power spectra of chest excursion invariably revealed a single major peak within the range of 1.076 to 1.648 Hz, with minor peaks at successive, higher harmonic frequencies (Fig 1C). There were no lower frequency peaks in the chest excursion records in any spontaneously breathing or mechanically ventilated animals. In mechanically ventilated animals the frequencies of the major peaks in the power spectra of chest excursion were, within the limits of accuracy of the equipment, coincident with the major peaks in the power spectra of the spirometer and reflected the rate of ventilation set by the ventilator.

In 22 trials collected from 6 animals, simultaneous recordings of chest excursion and CSF pressure, averaged more than 500 respiratory cycles, showed that CSF pressure was phase-locked to chest excursion (Fig 2). In spontaneously ventilating animals, CSF pressure and intrathoracic pressure were in phase, with the maximum CSF pressure coincident at the peak of expiration. In mechanically ventilated animals, however, CSF pressure peaked at the point of maximal chest expansion. In both spontaneous and mechanically ventilated rats the frequency of the most powerful oscillation in the CSF pressure had a weak "linear" correlation (r^2 = 0.27) with rate of ventilation (Fig 3). In 9 trials in 5 animals, simultaneous recordings of BP and

CSF pressure failed to demonstrate any phase locking of these pressure waves.

Discussion

Kusaka et al[8] had reported rhythmic variations in lumbar CSF pressure in anesthetized rats that they took to be entrained to the respiratory cycle. Furthermore, Barth et al[13] reported variations in CSF pressure, measured at the cisterna magna, which they took to be coincident with respiration and the cardiac cycle. In neither instance, however, were quantitative data reported and there was no attempt to determine CSF pressure oscillation frequencies. Similar rhythmic oscillations in CSF pressure have also been reported in other species.[14,15]

In addition to rhythmic oscillations in CSF pressure, apparently coincident with the cardiac and respiratory cycles, there are numerous reports of low-frequency oscillations, the B waves or Lundberg waves, in humans. These waves have been identified in patients with intracranial pathology, and have been attributed to the low-frequency oscillations in respiratory rate that characterize brain injury.[16,17] However, these waves have also been described in artificially ventilated patients and in healthy volunteers, and so cannot be causally attributed solely to pathologic respiratory rhythms.[17,18]

Because of the superimposition of nonharmonic rhythms, including those associated with the respiratory and cardiac cycles, any given oscillation in intracranial pressure is likely to have a complex waveform,[19] and it may be difficult to recognize component CSF pressure waves without mathematical processing of the raw signal (see, eg,[20,21]). Hence, in this study, we used power spectrum analysis to search for component waves within the CSF oscillations. We particularly sought entrainment to the cardiac and respiratory cycles, but also searched at lower frequencies for phenomena such as Lundberg waves. In this regard, we note that in human beings and other animals, low-frequency oscillations in BP, sometimes called Mayer waves, have been identified and attributed to an intrinsic slow rhythm in sympathetic output.[22] In humans, Mayer waves have a duration in the order of 10 seconds. No coincident oscillation has previously been reported in CSF pressure. However, if Mayer waves were to entrain oscillations in CSF pressure, the effect in humans should be distinguishable from B waves, which have wavelengths of 0.5 to 2 minutes. Furthermore, if B waves are attributable to oscillations in the respiratory rhythm,[16] they should be effaced by paced respiration, whereas Mayer waves should be relatively unaffected.

In the current study, 3 peaks were consistently found in the CSF pressure power spectrum, suggesting that pressure oscillations at 3 different frequencies were present. In both spontaneously ventilating and mechanically ventilated animals, the power spectrum of CSF pressure invariably displayed a major peak, between 0.98 and 1.95 Hz, which was at or very close to the major peak in the power spectrum of chest excursion (Fig 1). In 25 of 26 trials, this was far and away the most powerful peak in the CSF spectrum. In mechanically ventilated animals, this major CSF power spectrum peak was perfectly coincident with the rate of ventilation as determined from power spectrum analysis of the spirometer placed in series with the tracheal cannula. Hence, in both spontaneously ventilating and mechanically ventilated animals oscillations in CSF pressure were entrained to ventilation. This was confirmed by averaging of simultaneous recordings that showed phase-locked waves in CSF pressure and chest excursion. Furthermore, with positive-pressure mechanical ventilation, wherein maximum intrathoracic pressure occurs at the peak of chest excursion, the phase of entrainment shifted 180°. This strongly suggests that not only are CSF pressure oscillations strongly entrained to ventilation, but that they are also driven by intrathoracic pressure changes.

In 22 of 26 trials, the power spectra of CSF pressure also displayed a small peak, between 5.37 and 8.06 Hz, which was at or very close to the major peak of the cardiac cycle, and so reflected an association with heart rate. The power of this peak in the CSF pressure power spectrum was invariably less than 1% of the power of the major peak, between 0.98 and 1.95 Hz, which was coincident with the ventilatory rate. Others have reported oscillations in CSF pressure that appeared to be entrained to the cardiac cycle.[14] However, our results suggest that in anesthetized rats the arterial pulse exerts only a very minor influence over oscillations in CSF pressure at the lumbar level.

In 21 of 26 trials, it was also possible to resolve a low-frequency oscillation, between 0.006 and 0.24 Hz, in CSF pressure. This peak occurred in both spontaneously breathing rats and in rats mechanically ventilated at a fixed rate. This oscillation did not correspond to any detectable peak in the power spectra of the ventilatory cycle. Rather, it was entrained to a low-frequency oscillation in the power spectra of the EKG.

Conclusion

The results of this study suggest that, in the anesthetized rat, oscillations in CSF pressure are most strongly entrained to the ventilatory cycle and are driven by intrathoracic pressure. The major frequency of the cardiac cycle (heart rate) contributes little power to oscillations in CSF pressure, suggesting that arterial pulse pressure has only a weak influence on oscillations in CSF pressure at the lumbar level. On the other hand, in some animals oscillations in CSF pressure are entrained to a low-frequency oscillation in the cardiac cycle, and in a small minority of animals this rivals the influence of the ventilatory cycle.

Changes in CSF pressure induced by injections of small volumes of aCSF at the cisterna magna were immediately detectable at the lumbar level, suggesting that this model

may be useful in studies of the effects of vertebral position and motion on CSF fluid dynamics. However, the signal from CSF pressure at the lumbar level is complex, involving at least 3 nonharmonic waves. Consequently, although pressure changes may be transmitted from the upper cervical region to the lumbar subarachnoid space, a single pressure pulse might be obscured by background noise. On the other hand, sustained changes in CSF dynamics and manifesting as a shift in the frequency or power of a component oscillation may well be detectable by power spectrum analysis even if not evident on cursory inspection of the raw CSF pressure signal.

This study has quantified the frequencies and relative strengths (powers) of oscillations in the lumbar CSF pressure in the anesthetized rat and identified a clear association with the ventilatory cycle. It has also identified constraints in the measurement and interpretation of lumbar CSF pressure in the rat that need to be considered when investigating the effects of vertebral displacements and manipulations on CSF pressure dynamics.

Practical Applications

- CSF pressure in rats fluctuates in a deterministic fashion.
- In the anesthetized rat, CSF pressure fluctuation involves superimposition of oscillations at 3 or more distinct frequencies.
- Most often, the most powerful influence on CSF pressure oscillation is ventilation. CSF pressure waves are entrained to and driven by the respiratory cycle.
- Most often, a less powerful influence on CSF pressure is a low-frequency oscillation in the cardiac cycle.
- The arterial pulse wave has only a minor influence on the oscillation of CSF pressure.

Acknowledgment

This work was supported by grant LG 2005-5 from the Australian Spinal Research Foundation.

References

1. Richards DM. The Palmer philosophy of chiropractic—an historical perspective. Chiropr J Aust 1991;21:63-8.
2. Bolton SP. The "wet specimen". Chiropr J Aust 1994;24: 147-50.
3. Pederick FO. Cranial adjusting—an overview. Chiropr J Aust 1993;23:106-12.
4. Harrison DE, Cailliet R, Harrison DD, Troyanovich SJ, Harrison SO. A review of the biomechanics of the central nervous system—part II: spinal cord strains from postural loads. J Manipulative Physiol Ther 1999;22:322-32.
5. Harrison DE, Cailliet R, Harrison DD, Troyanovich SJ, Harrison SO. A review of biomechanics of the central nervous system—part III: spinal cord stresses from postural loads and their neurologic effects. J Manipulative Physiol Ther 1999;22: 399-410.
6. Moran R, Gibbons P. Intraexaminer and interexaminer reliability for palpation of the cranial rhythmic impulse at the head and sacrum. J Manipulative Physiol Ther 2001;24: 183-90.
7. Hartman S. Cranial osteopathy: its fate seems clear. Chiropr Osteopat 2006;14:10.
8. Kusaka G, Calvert JW, Smelley C, Nanda A, Zhang JH. New lumbar method for monitoring cerebrospinal fluid pressure in rats. J Neurosci Methods 2004;135:121-7.
9. Klarica M, Rados M, Draganic P, Erceg G, Oreskovic D, Marakovic J, et al. Effect of head position on cerebrospinal fluid pressure in cats: comparison with artificial model. Croat Med J 2006;47:233-8.
10. Cramer GD, Fournier JT, Henderson CNR, Wolcott CC. Degenerative changes following spinal fixation in a small animal model. J Manipulative Physiol Ther 2004;27:141-54.
11. Pickar J, Wheeler J. Response of muscle proprioceptors to spinal manipulative-like loads in the anesthetized cat. J Manipulative Physiol Ther 2001;24:2-11.
12. McNay EC, Gold PE. Extracellular glucose concentrations in the rat hippocampus measured by zero-net-flux: effects of microdialysis flow rate, strain and age. J Neurochem 1999;72: 785-90.
13. Barth K, Onesti S, Krauss W, Solomon R. A simple and reliable technique to monitor intracranial pressure in the rat. Neurosurgery 1992;30:138-40.
14. Hamer J, Alberti E, Hoyer S, Wiedemann K. Influence of systemic and cerebral vascular factors on the cerebrospinal fluid pulse waves. J Neurosurg 1977;46:36-45.
15. Shapiro K, Fried A, Takei F, Kohn I. Effect of the skull and dura on neural axis pressure-volume relationships and CSF hydrodynamics. J Neurosurg 1985;63:76-81.
16. Steinmeier R, Bauhuf C, Hubner U, et al. Slow rhythmic oscillations of blood pressure, intracranial pressure, microcirculation, and cerebral oxygenation. Stroke 1996;27: 2236-43.
17. Lescot T, Naccache L, Bonnet MP, Abdennour L, Coriat P, Puybasset L. The relationship of intracranial pressure Lundberg waves to electroencephalograph fluctuations in patients with severe head trauma. Acta Neurochir 2005;147:125-9.
18. Friese S, Hamhaber U, Erb M, Klose U. B-waves in cerebral and spinal cerebrospinal fluid pulsation; measurement by magnetic resonance imaging. J Comput Assist Tomogr 2004; 28:255-62.
19. Morgalla MH, Stumm F, Hesse G. A computer-based method for continuous single pulse analysis of intracranial pressure waves. J Neurol Sci 1999;168:90-5.
20. Czosnyka M, Pickard J. Monitoring and interpretation of intracranial pressure. J Neurol Neurosurg Psychiatry 2004;75: 813-21.
21. Stephensen H, Andersson N, Eklund A, Malm J, Tisell M, Wikkelso C. Objective B wave analysis in 55 patients with non-communicating and communicating hydrocephalus. J Neurol Neurosurg Psychiatry 2004;76:965-70.
22. Montano N, Cogliati C, da Silva VJD, Gnecchi-Ruscone T, Malliani A. Sympathetic rhythms and cardiovascular oscillations. Auton Neurosci 2001;90:29-34.