Spectral analysis of the CSF pulse wave at different locations in the craniospinal axis

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SUMMARY The frequency spectrum and the amplitude transfer function from arterial pulse pressure to the CSF pulse wave were estimated in the lateral ventricle, cisterna magna and lumbar subarachnoid space of anaesthetised ventilated cats under various conditions: (a) normal status, (b) inhalation of 5% CO_2 and (c) saline infusion into the CSF space (0·045 ml/min). The CSF pulse waveforms in the lateral ventricle and cisterna magna were almost identical in all conditions. Inhalation of CO_2 and saline infusion increased the values of the amplitude transfer function from blood pressure to the CSF pulse wave in the lateral ventricle and cisterna magna to a similar extent. The CSF pulse in the lumbar sac was remarkably damped under both normal conditions and during CO_2 inhalation, but the damping was diminished by saline infusion. During the saline infusion, the spinal canal appeared to function as a low-pass filter to the conduction of the CSF pulse.

It has been proposed that analysis of the CSF pulse waveform yields information about cerebrovascular autoregulation and the elastance of the intracranial system. Several authors have tried various methods of analysis of the CSF pulse waveform as an indicator of intracranial pathophysiology but the problem is complicated.[1-12] Before we can determine how useful it is for the management of patients, an adequate understanding of the components of the CSF pulse waveform is essential. A systematic analysis of the transmission of the spectral components of the CSF pulse waveform through the craniospinal axis, however, has not yet been reported.

This study was planned to determine the change in the spectrum of the CSF pulse waveform, the amplitude transfer function from blood pressure to the CSF pulse and the conduction of each component of the CSF pulse through the CSF space under normal conditions and abnormal conditions produced by inhalation of 5% CO_2 and saline infusion into the CSF space.

Materials and methods

Ten mongrel cats of both sexes, weighing between 3·4 and 5·4 kg, were used for these experiments. Each animal was premedicated by atropine 0·05 mg/kg subcutaneously and anaesthetised by ketamine 20 mg/kg intravenously, and half of this dose was repeated when systemic blood pressure and/or heart rate fluctuated remarkably. Tracheotomy and intubation were performed, then anaesthesia was maintained by mixed gases of 30% O_2 and 70% N_2O. A cannula for the drip infusion of Hartmann's solution was placed in the femoral vein. The femoral artery was cannulated to monitor blood gases (blood gas analyser IL system 1302 made by Instrument Laboratory, Milano, Italy). Systemic blood pressure was detected through a catheter placed in the left subclavian artery. The animal was paralysed by pancuronium bromide 0·05 mg/kg IV, and half of this dose was repeated as needed. Respiration was maintained mechanically (Harvard respirator Model 665 A made by Harvard Apparatus, South Natick, Massachusetts, USA). The animal was placed in a stereotaxic frame in the sphinx position and a laminectomy was made at L–7. A catheter and a 22-gauge spinal needle (87 mm length) were placed in the lumbar subarachnoid space for saline infusion and pressure monitoring, respectively. A 22-gauge spinal needle (87 mm length) was placed in the cisterna magna through the foramen magnum under direct vision. A spinal needle of the same size was inserted into the lateral ventricle stereotaxically through a drill hole in the skull. The sites of insertion of the catheter and needles were sealed by cyanoacrylate adhesive.

A catheter placed in the subclavian artery and the needles in the CSF space were connected to pressure transducers
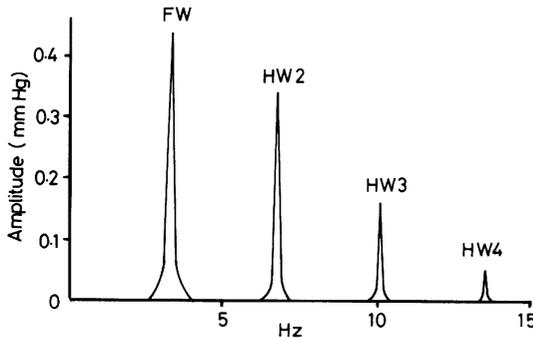
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Fig 1   *An example of spectrum of the CSF pulse wave.*
FW = fundamental wave, HW2 = 2nd harmonic wave,
HW3 = 3rd harmonic wave, HW4 = 4th harmonic wave.

Table   *Average blood pressure, CSF pressure and pCO$_2$ under each experimental condition. Mean and SD are shown*

	BP (mmHg)	Pcsf (mmHg)	pCO$_2$ (mmHg)
Normal	139·6 ± 28·6	7·0 ± 2·3	28·9 ± 5·8
	↕ N.S.	↕ p < 0·005	↕ p < 0·005
5% CO$_2$	156·2 ± 21·6	15·4 ± 5·1	48·8 ± 3·1
	↕ N.S.	↕ p < 0·005	↕ p < 0·005
Saline	132·7 ± 26·3	28·4 ± 5·0	26·4 ± 2·8

(pressure transducers made by Bentley Laboratories, Irvine, California, USA) by tubes of high pressure type (Manometer line (30 cm length) made by Portex, Hythe, Kent, England) for recording by a polygraph and FM tape recorder (FM tape recorder Model MR–10 made by TEAC corporation, Tokyo, Japan). All pressure transducers were calibrated by a water column and positioned halfway between the tip of the dorsal spine and the sternum. Pulsatile com-
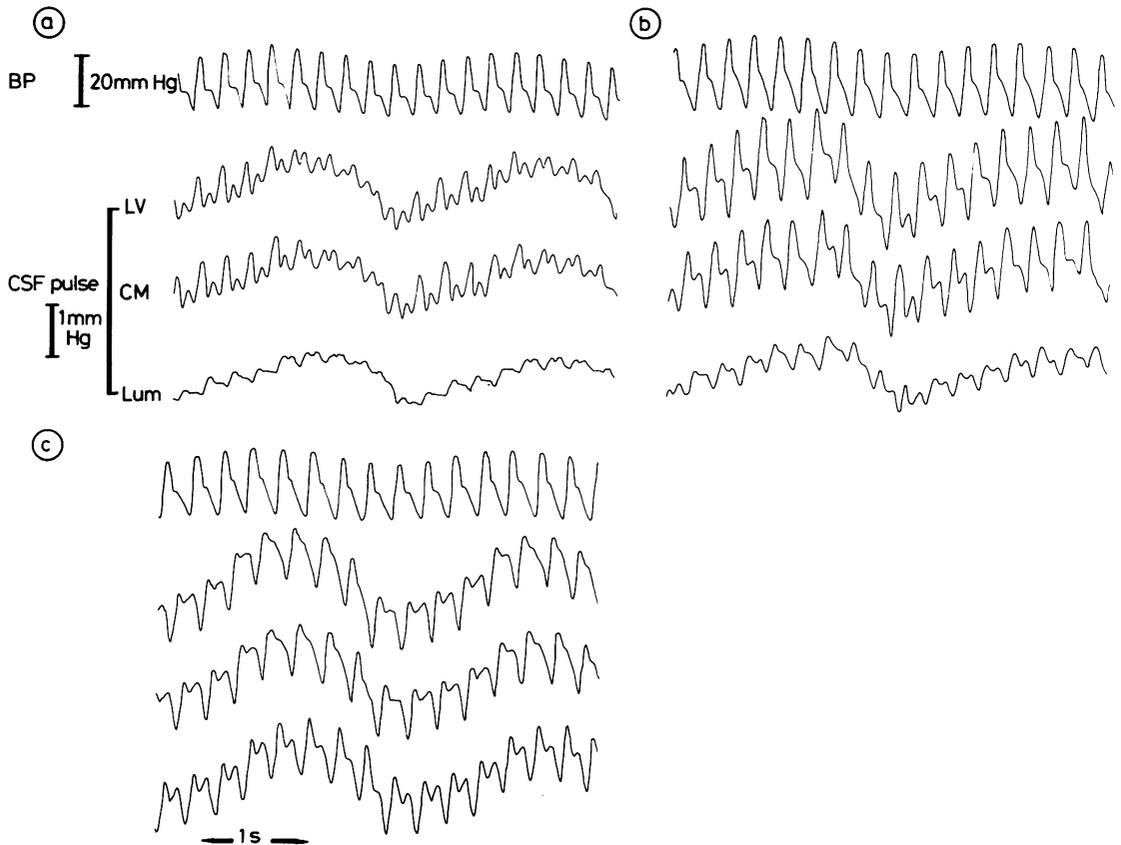

Fig 2   *Comparison of waveforms of blood pressure and the CSF pulse wave estimated at the lateral ventricle (LV), cisterna magna (CM) and lumbar sac (Lum) under various conditions; (a) normal condition, (b) inhalation of 5% CO$_2$ and (c) saline infusion (0·045 ml/min).*
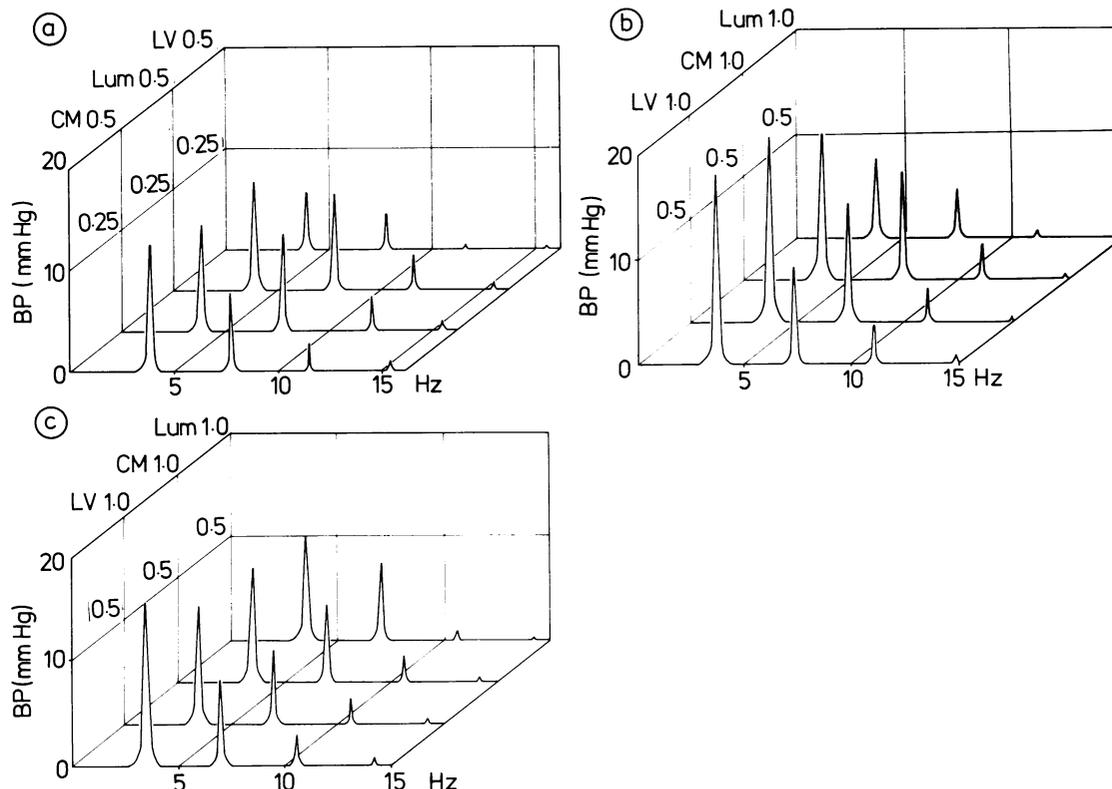*This is AC recording to show only the pulsatile component of each pulse wave.*

Fig 3 *Comparison of spectrum of blood pressure and the CSF pulse wave; (a) normal condition, (b) inhalation of CO_2 and (c) saline infusion.*

ponents of CSF pulse and blood pressure were selectively magnified by a four channel AC amplifier with preset gain from twice to 100 times, and recorded by a FM tape recorder.

After stabilisation of the animal, the CSF pulse at various sites and blood pressure under normal conditions were recorded. Then mixed gases of 5% CO_2 and 95% O_2 were inhaled for 5 minutes and all pressures were recorded. Sufficient time was allowed to recover from CO_2 inhalation, and finally saline infusion at the rate of 0·045 ml/min, was carried out to raise CSF pressure artificially.

Spectral analysis of the collected arterial and CSF pressure data was performed after the end of the experiment. The signal from the FM data recorder was sampled by a 12 bit analog-to-digital converter at the rate of 50 Hz, and the frequency spectrum of each pressure waveform was calculated by a 16 bit personal computer using an algorithm of 512 point Fast Fourier Transform, the programme for which has been reported previously.[13] The range of frequency spectrum was between 0·6 and 18·1 Hz and the frequency resolution was 0·098 Hz. In this study, we analysed only the spectral components derived from cardiac beats, ignoring the lower frequency component associated with respiration.

The frequency response of the pressure measuring system was tested and confirmed to be flat up to 20 Hz.

Nomenclature of spectral components
An example of the spectrum of the CSF pulse wave is shown in fig 1. Four sharp peaks are present; this pattern of spectrum was basically the same for both blood pressure and the CSF pulse. The definition of abbreviations for spectral components are:

    FW   =fundamental wave (or first harmonic wave)
    HW2 =2nd harmonic wave
    HW3 =3rd harmonic wave
    HW4 =4th harmonic wave

The amplitude transfer function from arterial blood pressure to CSF pulse waveform was calculated by the following equation[3]:

$$TFa(x) = \frac{\text{amplitude of spectral component (x) of CSF pulse}}{\text{amplitude of spectral component (x) of blood pressure}}$$

where TFa(x) = amplitude transfer function of spectral component (x)
(x = FW, HW2, HW3 or HW4)

The ratio of amplitude of each spectral component of the CSF pulse measured at the cisterna magna and lumbar sac was estimated to present the attenuation of the CSF pulse
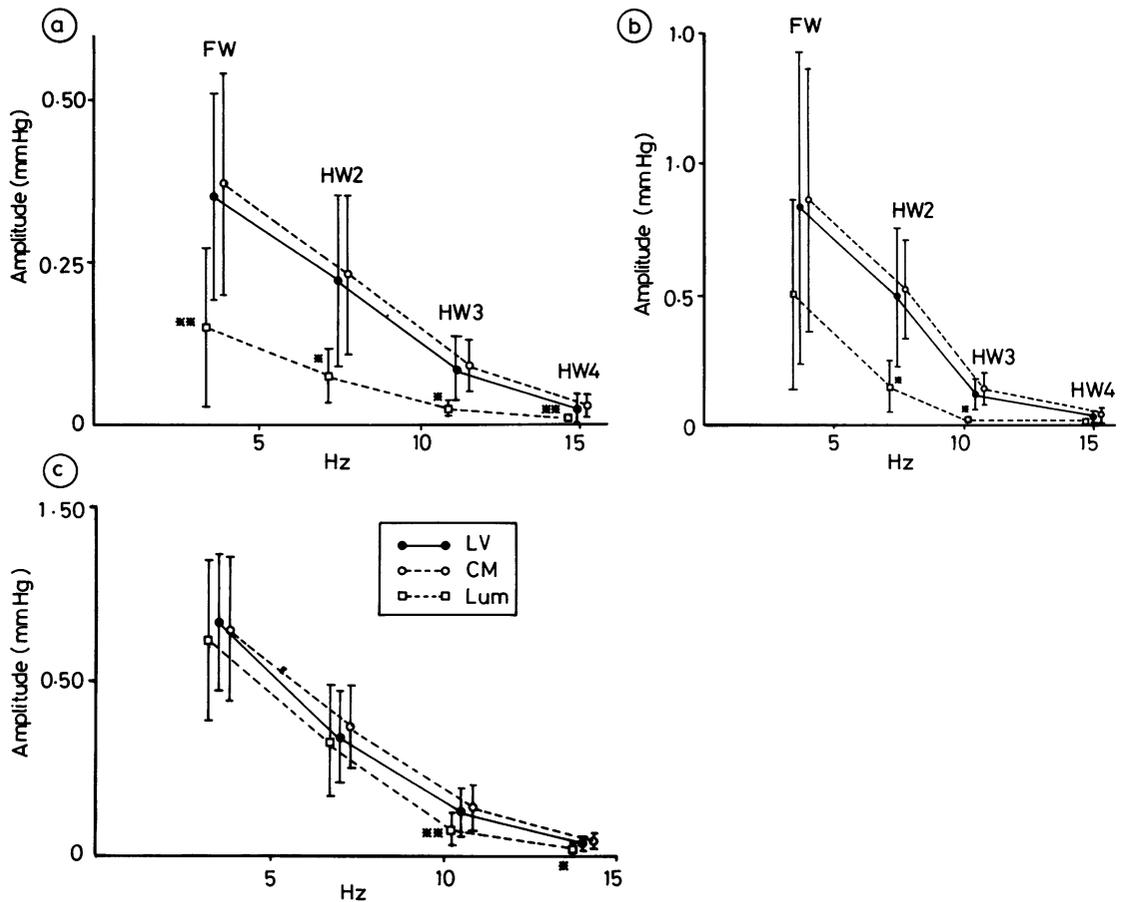
Fig 4 *Spectrum of the CSF pulse wave estimated in the lateral ventricle (closed circle), cisterna magna (open circle) and lumbar sac (square) under (a) normal condition (significant difference from values estimated in the cisterna magna; *p < 0·01, **p < 0·02 t-test), (b) inhalation of CO$_2$ (*p < 0·005) and (c) saline infusion (*p < 0·01, **p < 0·025). Mean and SD are shown.*

during its conduction through the spinal canal and was calculated by the following equation:

$$\text{Att(CM-Lum)} = \left(1 - \frac{\text{Amp(Lum)}}{\text{Amp(CM)}}\right) \times 100\%$$

where Att(CM–Lum) = attenuation of each spectral component
Amp(Lum) = amplitude of each spectral component at lumbar sac
Amp(CM) = amplitude of each spectral component at cisterna magna

Results

(1)   *Spectrum of CSF pulse wave at different locations under various conditions*
An example of the waveforms of arterial blood pres-

sure and the CSF pulse measured at the lateral ventricle, cisterna magna and lumbar sac under (a) normal condition, (b) inhalation of 5% CO$_2$ and (c) saline loading are shown in fig 2. The waveforms of CSF pulse in the lateral ventricle and cisterna magna are very similar under all three conditions. In contrast, the waveform of CSF pulse at the lumbar sac was remarkably damped under normal condition and inhalation of 5% CO$_2$, but it was much less damped while saline was infused into the CSF space. Differences in the spectral components of each waveform under various conditions are shown by the arrays of spectra in fig 3.

The spectra of the CSF pulse waveform estimated at different locations were compared under (a) normal conditions, (b) inhalation of 5% CO$_2$ and (c) saline infusion at the rate of 0·045 ml/min (fig 4). The
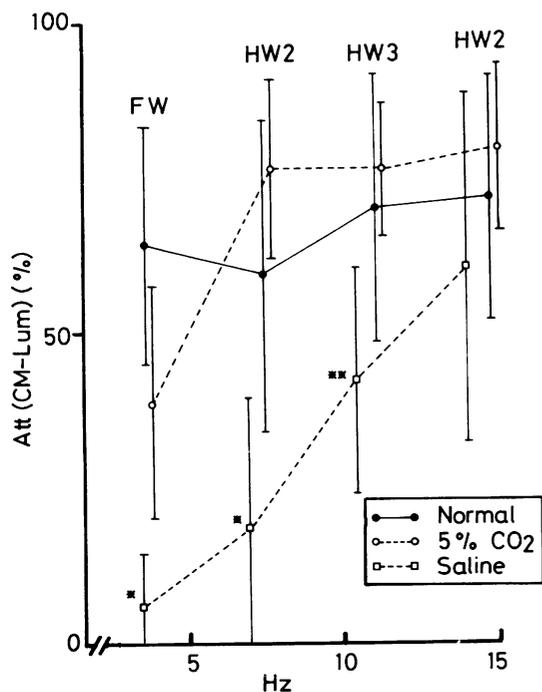
Fig 5 *Attenuation of spectral components of the CSF pulse wave during the conduction from the cisterna magna to lumbar sac under (a) normal condition ●, (b) inhalation of CO_2 ○ and (c) saline infusion □. (Significant difference from values under normal condition; *p < 0·01, **p < 0·05 t-test). Mean and SD are shown.*

levels (mean $\pm$ SD) of arterial blood pressure, CSF pressure and pCO_2 under normal condition, 5% CO_2 inhalation and saline infusion are summarised in the table. As noted in fig 4, spectral components of the CSF pulse at the lateral ventricle and cisterna magna were almost identical under all conditions. On the contrary, all components of the CSF pulse at the lumbar sac were damped or attenuated under normal conditions and during inhalation of CO_2. Saline infusion diminished the damping of the FW and HW2 components of CSF pulse wave in the lumbar sac significantly, so that the amplitude of FW and HW2 components became the same as in the lateral ventricle and cisterna magna.

(2) *Attenuation of spectral components of the CSF pulse wave during the conduction through the spinal canal.*
The attenuation of the spectral components of the CSF pulse during conduction from the cisterna magna to the lumbar sac under various conditions is summarised in fig 5. Under normal conditions, all components of the CSF pulse were almost equally at-

tenuated. Inhalation of CO_2 diminished the attenuation of the FW component and exaggerated the attenuation of other components although the difference was not significant for each component. Saline infusion diminished the attenuation of the FW, HW2 and HW3 components significantly but did not change that of the HW4 component. Thus, the spectral components of higher frequency were more attenuated.

(3) *Amplitude transfer function at different locations under various conditions*
The values of the amplitude transfer function estimated at different locations in the craniospinal axis are shown in fig 6. The amplitude transfer function at the cisterna magna was apt to be larger than that at the lateral ventricle under all conditions although the difference was not significant. The amplitude transfer function at the lateral ventricle and cisterna magna during CO_2 inhalation and saline infusion were not significantly different in spite of the differences of CSF pressure in those two conditions. The amplitude transfer function estimated at the lumbar sac showed a fairly flat pattern under normal conditions and CO_2 inhalation. In contrast, the amplitude transfer function of the FW and HW2 components of CSF pulse wave at the lumbar sac were similar to those at the lateral ventricle and cisterna magna during saline infusion.

Discussion

(1) *Spectrum of CSF pulse waveform and amplitude transfer function.*
The present results concerning the frequency spectrum and the amplitude transfer function of the CSF pulse are similar to those reported by Portnoy and co-workers although they did not compare the spectrum at different locations.[10]

The spectra and transfer functions of the CSF pulse estimated in the lateral ventricle and cisterna magna were almost identical under both normal and abnormal conditions. Some authors have reported that CSF pulse waveform in the lateral ventricle and subdural space over the convexity are similar.[14][15] These findings suggest that the CSF pulse waveform is almost the same at any location in the intracranial space.

The site or source of pulse transmission from arterial pressure to CSF has not been determined conclusively.[6][7][16-20] If the CSF pulse wave is almost the same at any location in the intracranial space, the site of pulse transmission is more likely to be a relatively large 3-dimensional structure rather than some small localised source. It would support the hypothesis advocated by Hirai that the CSF pulse is generated by
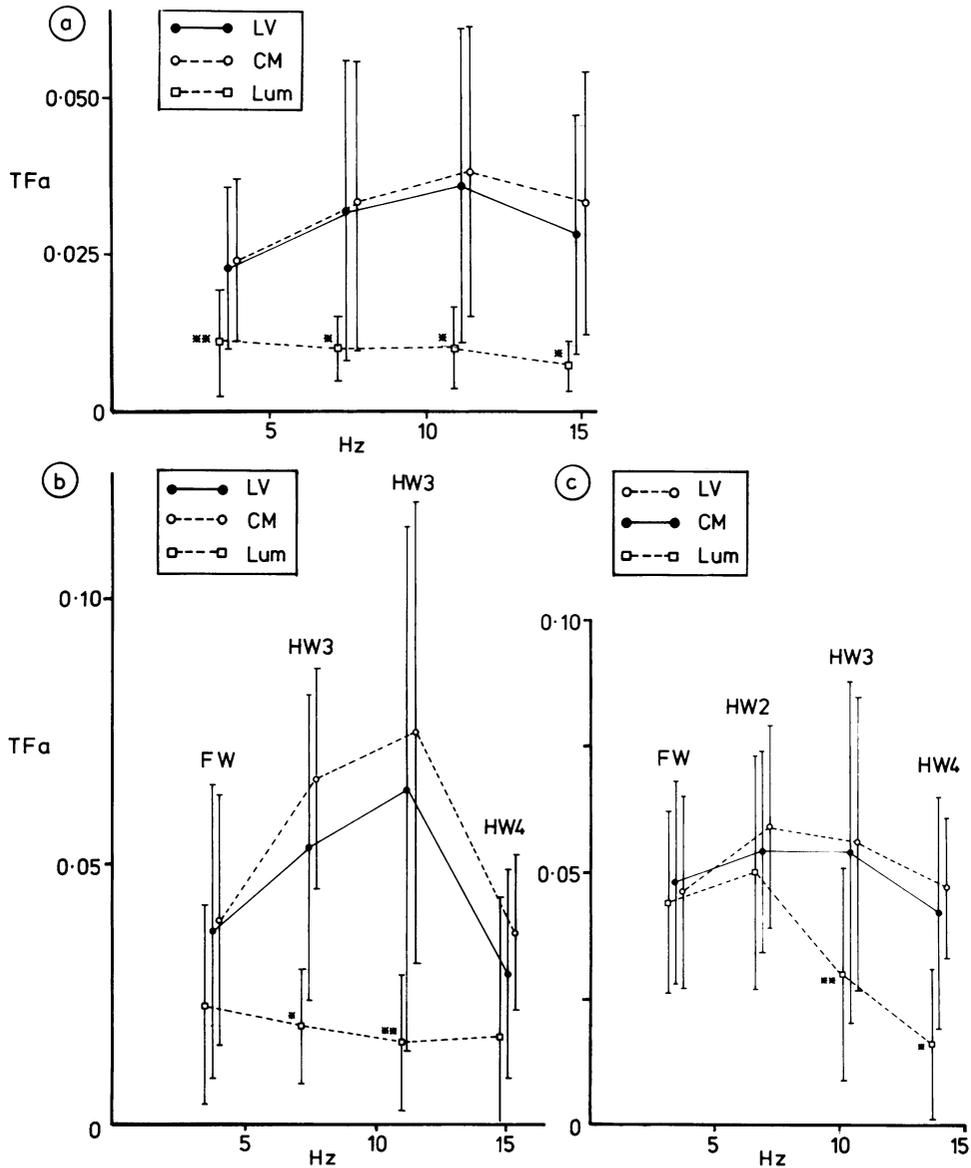
Fig 6 *Amplitude transfer function from blood pressure to CSF pulse at different locations under various conditions; (a) normal condition (significance difference from values estimated in the cisterna magna; *p < 0.02, **p < 0.05 t-test), (b) inhalation of CO_2 (*p < 0.005, **p < 0.01) and (c) saline infusion (*p < 0.01, **p < 0.05). Mean and SD are shown.*

the pulsatile vibration of the brain parenchyma derived from blood flow in the cerebral arteries.[7] Pulsation of the brain is commonly noted during intracranial surgery and would have enough energy to generate the CSF pulse. This has been also revealed by radiological study with observation of reduction in

size of the lateral and third ventricles with each pulsation.[21]

Inhalation of 5% CO_2 elevated the value of amplitude transfer function very effectively even when CSF pressure was much lower than during saline infusion. These findings suggest that cerebrovascular reactivity

is more important than the intracranial elastance as a determining factor of CSF pulse waveform, at least in the range of normal or moderately raised ICP.

(2) *Attenuation of the CSF pulse in the spinal canal*
The manner of attenuation of the CSF pulse during its conduction through the spinal canal under normal condition and CO_2 inhalation showed that the spinal canal worked as a simple attenuator against the CSF pulse; all spectral components were almost equally diminished.

When saline was infused, the spinal canal was seen to function as a low-pass filter; smaller attenuation for spectral components of lower frequency. This function of the spinal canal as a low-pass filter, was also noted in a clinical study which we have reported previously.[12]

One possible criticism of this hypothesis would be that the CSF pathway might be blocked at the cervical portion by the hyperextension of the neck. We considered it was unlikely because there was no discrepancy between the values of mean CSF pressure measured in the cisterna magna and lumbar sac during the entire course of experiments.

In another experimental study, we have found that saline infusion into the CSF space reduced the "buffering capacity" of the CSF space as measured by the pressure-volume index.[22] Under these conditions bolus injections of saline into the CSF space produced abrupt changes in pressure which were more freely conducted across the craniospinal axis after saline loading. The present results suggest that the same factor may apply in the conduction of CSF pulse waveform in the spinal canal.

Thus it can be proposed that factors related to cerebrovascular reactivity regulate the form of the intracranial CSF pulse and the transfer functions of amplitude from the arterial to the CSF pulse. On the other hand, factors related to intracranial elastance are responsible for the conduction of CSF pulse from the cranial to the lumbar CSF space.

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