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Lumbar cerebrospinal fluid pulse wave rising from pulsations of both the spinal cord and the brain in humans

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There are two theories regarding the origin of the lumbar cerebrospinal fluid pulse wave (L-CSFPW): that it arises from the arteries supplying the spinal cord, and that it is due to the pulsations of the brain transmitted through the subarachnoid space of the spine.

We investigated L-CSFPW of 11 myelopathic patients with a complete (five patients, CB-group) or an incomplete spinal block (six, ICB-group) on myelography to determine the origin of L-CSFPW. Since arterial pressure amplitude (APA), the energy source of L-CSFPW, is not the same between individuals or between before and after operation, not only L-CSFPW itself but also the transfer function between the arterial pressure wave and the L-CSFPW calculated by the system analysis method was analyzed to eliminate the influence of hemodynamic fluctuations. In the system analysis, the arterial pressure wave, L-CSFPW and transfer function were decomposed into five harmonic waves (HW).

In the CB group, L-CSFPW was observed to be 0.72 mmHg on average (range, 0.25–1.00) in spite of blocking pulsations of the brain, showing that there was a contribution to L-CSFPW unrelated to the brain, that is, the spinal cord. In the CB group, however, the preoperative transfer function value of HW1 (mean, 0.056; range, 0.012–0.170) was lower than that in the ICB group (mean, 0.137; range, 0.061–0.236) ($P<0.05$), indicating that the brain pulsation also contributed to L-CSFPW.

In the ICB group, there was significant reduction of HW1 ($P<0.01$) and HW2 ($P<0.05$) transfer function after posterior decompression surgery in spite of improvement in the subarachnoid space narrowing: preoperative HW1, mean, 0.137, range, 0.061–0.236; postoperative HW1, mean, 0.065, range, 0.021–0.153; preoperative HW2, mean, 0.092, range, 0.011–0.148; postoperative HW2, mean, 0.044, range, 0.030–0.066. It has been reported that the spinal cord blood flow is decreased 20% or more by laminectomy, therefore, L-CSFPW measurement may be sensitive enough to detect a 20% or higher decrease in this flow. This suggests that L-CSFPW could possibly be used clinically as a non-invasive method of monitoring the spinal cord blood flow. For broad clinical application of CSFPW, however, further studies are needed, especially on the direct relationship between CSFPW and spinal cord blood flow itself.

Keywords: lumbar cerebrospinal fluid pulse wave; spinal cord blood flow

Introduction

In 1980 Chopp and Portnoy[1] reported that the intracranial cerebrospinal fluid pulse wave (I-CSFPW) originated from vascular pulsations of the brain using a system analysis approach, and since then I-CSFPW has been clinically used as an indicator of the status of the cerebral vasculature.[2]

There are two concepts regarding the origin of the lumbar cerebrospinal fluid pulse wave (L-CSFPW): that it arises from pulsation of the spinal cord.[3,4] and that it is due to pulsation of the brain which is transmitted through the subarachnoid space of the spine.[5,6] We recently demonstrated in dogs that L-CSFPW reflects pulsation of both the spinal cord and brain by analyzing the effect on it of the occulusion of tha aorta and vena cava using system analysis; this showed the possibility that L-CSFPW could be an indicator of the spinal cord blood flow.[7] No investigation has heretofore been carried out in the human, however.

There are many kinds of lesions which block the subarachnoid space. Since the occlusion reflects the contribution of the brain to L-CSFPW, a study on the relationship of the subarachnoid space occlusion and L-CSFPW may indicate the origin of L-CSFPW in humans. Furthermore, since it is known that the spinal cord blood flow is decreased by laminectomy and/or handling of the spinal cord,[8–10] an understanding of

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the influence of laminectomy and/or spinal cord surgery on L-CSFPW may show contribution of the spinal cord blood flow.

The energy source of L-CSFPW is arterial pressure amplitude (APA). Since the energy source is not the same between individuals or between before and after operation, it is theoretically senseless to directly compare these pulse waves themselves. System analysis[2] is a method of analyzing the unknown internal structure of a given system without the influence of input signal differences. The arterial pressure wave and L-CSFPW are periodic wave signals. To apply the system analysis approach, the wave signals are transformed by Fast Fourier Transform into a power spectrum, which makes it possible to decompose the signal into component frequencies, harmonic waves, to remove contaminating noise and to calculate the transfer function of the signals. The transfer function shows how the output signals respond to the input signals on each frequency, thereby revealing the internal structure of the system without the influence of input signal fluctuations.

We investigated L-CSFPW in patients with the subarachnoid space occlusion using the system analysis method in an attempt to learn the origin of L-CSFPW.

Patients and methods

We studied 11 patients with myelopathy, seven males and four females averaging 51 (19–72) years. The etiology of myelopathy was: tumor in the spinal canal (six cases), ossification of the posterior longitudinal ligament (two) and others (three). The level of the lesions was cervical spine in six cases, thoracic spine in three, both cervical and thoracic spine in one and lumbar spine in one (Table 1). Myelography by lumbar puncture using water soluble contrast media was performed in all of the patients, and the results were complete block in five patients (CB group) and incomplete block in six (ICB group). All subjects gave informed consent for surgery and testing.

A 21 G catheter was inserted into the left radial artery to monitor arterial pressure wave, while a Camino fiber-optic catheter (Model 420, Camino Laboratory, San Diego, CA) was inserted into the lumbar spinal canal at L3/4 or L4/5 under X-ray image to record L-CSFPW. The Queckenstedt test (Q-test) was carried out in an awake condition in the lateral position with the neck in a neutral position. Lumbar cerebrospinal fluid pressure (L-CSFP) was measured before and after a 20 s manual bilateral jugular compression.

Surgery was done under general anesthesia, and after the spinal cord decompression procedure through a posterior approach the dural pulsation in the lesion was confirmed under direct vision. There was no neurological deterioration and postoperative magnetic resonance imaging revealed restoration of the subarachnoid space in all cases.

The arterial pressure wave and L-CSFPW were measured just before the operation and after completion of spinal cord decompression, recorded on a magnetic tape recorder and analyzed with a power

Table 1 Clinical and haemodynamic data

Case	*Age*	*Sex*	*Diagnosis*	*Level*	*Myelo*	*L-CSFP (mmHg) at rest*	*L-CSFP (mmHg) increase*	*SAP (mmHg) before*	*SAP (mmHg) after*	*APA (mmHg) before*	*APA (mmHg) after*
1	58	f	SCT	C	CB	16	0	96	90	21	20
2	55	f	SCT	T	CB	11	4	74	131	30	57
3	38	m	SCT	C	CB	15	5	101	142	27	57
4	69	m	SCT	C	CB	4	6	87	129	20	43
5	42	f	SCT	T	CB	9	6	104	136	29	47
							a	b	c	d	e
Mean	52.4					11.0	4.2	92.4	125.6	25.4	44.8
SD	12.5					4.8	2.5	12.1	14.9	4.6	15.2
6	19	f	SCT	L	ICB	12	16	85	110	33	41
7	66	m	OPLL	C	ICB	15	20	94	94	36	36
8	72	m	CSM	C	ICB	5	27	85	146	32	62
9	60	m	OPLL	C	ICB	7	33	116	129	40	29
10	38	m	syringo	C+T	ICB	11	37	80	147	33	79
11	49	m	PC	T	ICB	7	45	116	144	33	52
							f	g	h		
Mean	50.7					9.5	29.7	96.0	128.3	34.5	49.8
SD	19.7					3.8	10.8	16.1	22.0	3.0	18.5

$P<0.01$: a *vs* f, $P<0.05$: b *vs* c, d *vs* e, g *vs* h. m: male, f: female, SCT: spinal cord tumor, OPLL: ossification of posterior longitudinal ligament, CSM: cervical spondylotic myelopathy, syringo: syringomyelia, PC: plasmacytoma of the spine, C: cervical, T: thoracic, L: lumbar, CB: complete block, ICB: incomplete block, L-CSFP: lumbar cerebrospinal fluid pressure at rest, increase: increase of L-CSFP by jugular compression, SAP: Systolic arterial pressure, APA: Arterial pressure amplitude, before: before operation, after: after operation spectrum program using a signal processor (7T-18, NEC-Sanei, Tokyo, Japan). The L-CSFPW power spectrum was decomposed into five harmonic waves (HWs) (Table 2) and the transfer function values between the left radial artery wave and L-CSFPW were calculated. The power spectrum measurement conditions were: calibration: 20 Hz; sample length: 10 s; frequency resolution F: 0.2 Hz; hamming window: on. The data was analyzed using Mann-Whitney test or paired *t*-test with significance set at $P<0.05$.

Results

Increase of L-CSFP by jugular compression averaged 4.2 (range, 0–6) mmHg in CB group and 29.7 (range, 16–45) mmHg in ICB group and the values were significantly different between the two groups (Table 1).

The hemodynamics data, systolic arterial pressure (SAP) and arterial pressure amplitude (APA), were not the same between before and after operation. APA varied from 20–79 mg among individuals and the changes between before and after the operation were significant in the CB group (Table 1).

In the CB group, L-CSFPW was observed to be 0.72 mmHg on average (range, 0.25–1.00), but the preoperative mean values of transfer function were lower in all five HWs than those in ICB group and the values of HW1 (mean, 0.056; range, 0.012–0.170) were lower than that in the ICB group (mean, 0.137; range, 0.061–0.236) ($P<0.05$) (Figure 1).

In the ICB group, the transfer function values decreased in most HW of all cases after operation in comparison with those before operation, and the difference was significant in both HW1 ($P<0.01$) and HW2 ($P<0.05$) (Figure 2): preoperative HW1, mean, 0.137, range, 0.061–0.236; postoperative HW1, mean, 0.065, range, 0.021–0.153; preoperative HW2, mean, 0.092, range, 0.011–0.148; postoperative HW2, mean, 0.044, range, 0.030–0.066.

In the CB group, the transfer function values tended to increase after operation, but the difference was not significant except for HW5 (Figure 3).

Discussion

Patients were divided into two groups according to the results of myelography: complete and incomplete block. The difference in the degree of subarachnoid space occlusion between the two groups was confirmed by Q-test; increase of L-CSFP by jugular compression was significantly different between the two.

The hemodynamics data including APA, the energy source of L-CSFPW, was not the same between the groups or between before and after operation, as expected. Therefore, the values of L-CSFPW themselves were not directly compared but the system analysis method was used instead to eliminate the hemodynamic changes.

L-CSFPW was recorded even in the CB group where the subarachnoid space was occluded so that pulsation of the brain was blocked. This fact indicates that L-CSFPW arises at least partly from spinal vascular pulsation. The preoperative transfer function values in the CB group, on the other hand, were significantly lower than those in the ICB group, showing that blocking of the subarachnoid space influences L-CSFPW; pulsation of the brain also contributes to L-CSFPW. These results are consistent

Table 2 Lumbar cerebrospinal fluid pulse amplitude (L-CSFPWA) and lumbar cerebrospinal fluid pulse wave (L-CSFPW) power spectrum

L-CSFPWA (mmHg)			*L-CSFPW power spectrum (mV)*									
					before					*after*		
Case	*before*	*after*	*HW1*	*HW2*	*HW3*	*HW4*	*HW5*	*HW1*	*HW2*	*HW3*	*HW4*	*HW5*
1	0.82	0.32	15.05	1.98	1.10	0.33	0.62	23.09	4.18	1.06	0.28	1.01
2	0.61	1.32	3.94	1.21	0.60	0.52	0.15	0.88	1.46	0.08	0.07	0.32
3	0.93	0.43	4.58	2.00	1.46	0.67	0.38	2.68	0.40	0.81	0.38	0.20
4	1.00	0.93	2.51	1.32	0.55	0.19	0.07	22.27	4.25	0.41	0.42	0.85
5	0.25	0.14	2.85	0.83	0.51	0.14	0.42	3.10	5.30	0.72	0.17	0.60
Mean	0.72	0.63	5.79	1.47	0.84	0.37	0.33	10.40	3.12	0.62	0.26	0.59
SD	0.30	0.49	5.25	0.51	0.42	0.22	0.22	11.24	2.01	0.38	0.15	0.34
6	1.59	0.25	2.07	0.73	0.21	0.11	0.11	4.39	1.89	0.30	0.25	0.07
7	1.96	1.82	19.14	3.79	1.87	0.16	0.20	7.48	1.94	0.49	1.20	0.54
8	0.36	0.89	3.98	1.48	2.19	0.70	0.77	2.01	1.44	0.80	0.39	0.04
9	0.68	0.25	8.08	5.24	2.06	0.95	0.25	1.48	1.07	0.69	0.52	0.18
10	3.50	2.71	5.17	4.01	1.58	0.76	0.48	3.52	3.89	0.88	0.41	0.37
11	1.36	0.32	22.86	7.97	2.44	0.96	0.37	16.90	2.35	3.30	2.11	0.82
Mean	1.49	0.99	10.27	3.87	1.73	0.61	0.36	5.96	2.10	1.08	0.81	0.34
SD	1.04	0.94	8.66	2.62	0.80	0.38	0.24	5.76	0.98	1.11	0.72	0.30

HW: harmonic wave, before: before operation, after: after operation with the analysis of the effects of occlusion of the aorta and vena cava on L-CSFPW in dogs using system analysis; L-CSFPW arises from pulsations of both the spine and brain,[7] although the vascular system of the spinal cord in humans is different from that in the dog.[11]

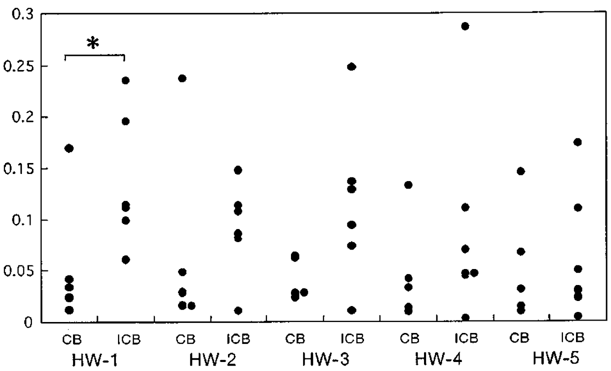

Figure 1 Transfer function before operation. CB: complete block, ICB: incomplete block, HW: harmonic wave, *:$P<0.05$

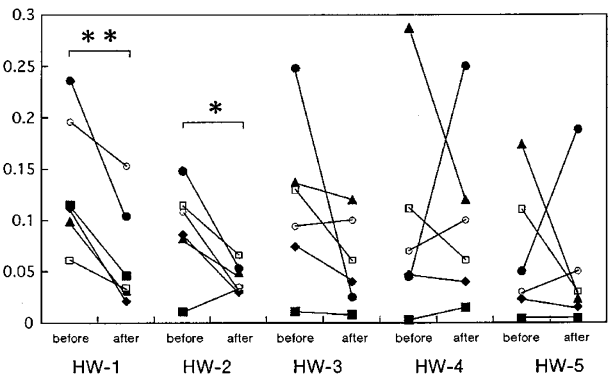

Figure 2 Changes of transfer function during operation in incomplete block group. before: before operation, after: after operation, HW: harmonic wave, **:$P<0.01$, *:$P<0.05$

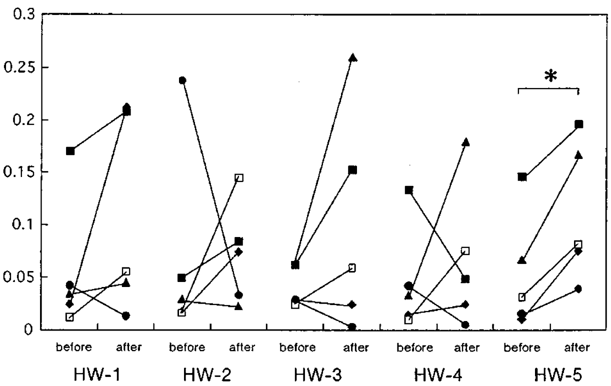

Figure 3 Changes of transfer function during operation in complete block group. before: before operation, after: after operation. HW: harmonic wave, *:$P<0.05$.

In the ICB group, transfer function decreased after posterior decompression surgery in spite of improvement in the subarachnoid space narrowing. This result is explained by the observation that the spinal cord blood flow is decreased by laminectomy and/or handling of the spinal cord; Anderson *et al.*[8] observed 22–45% decrease of spinal cord blood flow by one segment (L-2) laminectomy in cats with the reference sample method using isotope – labeled microspheres and speculated a temperature-induced vasoconstriction as the mechanism; Toribatake *et al.*[10] reported that spinal cord blood flow decreased by 22.1% on average using the hydrogen clearance method and speculated opening of the airtight condition of the epidural space as the mechanism because there was no difference in the decrease between simple laminectomy and total spondylectomy; Kuchiwaki *et al.*[9] observed that a 2 mm-lateral retraction of the spinal cord at C1-2 segments at a rate of 0.3 mm/s using a micromanipulator caused a decrease of the blood flow by 31.9–72.0% with the hydrogen clearance method. Since the spinal cord blood flow itself was not measured in this study, it is unknown how great a decrease can be detected in the blood flow measurement of L-CSFPW. However, if the animal studies described above are referred to, L-CSFPW measurement may be sensitive enough to detect about a 20% or more decrease in spinal cord blood flow. Since Toribatake *et al.*[10] reported that the average 22.1% decrease caused no conduction disturbance electrophysiologically, and there was no neurological deterioration in any of the cases of this study, measurement of L-CSFPW may reflect a mild decrease in blood flow where even spinal cord function is not disturbed.

In the CB group, the transfer function values tended to increase after operation, but the difference was not significant in spite of restoration of the subarachnoid space. This may also be explained by the effect of contribution of the brain pulsation through the restored subarachnoid space being cancelled out by decrease of the spinal cord blood flow by laminectomy.[8–10]

The methods now available to evaluate the spinal cord blood flow, like the radioactive microsphere or hydrogen clearance method, are not suitable for intraoperative monitoring because of their invasiveness.[12,13] If further findings clarify the contribution rate of spinal cord pulsation to L-CSFPW and the precise relationship between the waves and the spinal cord blood flow in human, L-CSFPW could be used clinically as a non-invasive method of monitoring the spinal cord blood flow.

In conclusion, L-CSFPW arises from pulsation of the spine as well as brain in human and may reflect a mild decrease in blood flow when even spinal cord function is not disturbed.

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