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The Relationship between central conduction time and intracranial pressure in head injured patients[x]

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Summary: The relationship between intracranial pressure (ICP) and central somatosensory conduction time (CCT) was investigated in patients with head injury. The patients were studied in two groups. In 67 patients, CCT was recorded simultaneously with ICP and MBP at different levels of spontaneous changes of ICP. In 20 patients the same measurements were carried out before and after mannitol administration. There was a low but significant correlation between CCT and ICP, and also between CCT and CPP. However, it seems that there is no any CCT latencies that corrospond with given mean ICP values. It might be concluded that CCT does not provide an index that may be used in the diagnosis and treatment of increased ICP in head injured patients.

Key words: Central conduction time, head injury, intracranial pressure, cerebral perfusion pressure

Potential effects of ICP on evoked potentials have been hypothesized by many authors in head injury (8,10,14,19-21,27). Experimental models of concussion and coma have demonstrated that somatosensory evoked potentials are altered at the brain stem and mesencephalic levels as well as at the level of cerebral cortex, without evidence of prolonged changes in ICP (20,21,27). However , the direct cortical response has been shown to be quite sensitive to elevation of ICP when CBF was reduced (11). Recent studies demonstrated that measurement of evoked potentials might be a helpful adjunct in the diagnosis of intracranial pressure changes (1,18,23,29,30). We attempted to elucidate the possible correlations between ICP and CCT in head injured patients.

Material and Methods

Normal subjects

Recording of CCT from 23 healthy volunteers (5 male, 18 female) served as controls. Age ranged from 18 to 60 years (mean 30 years). None had any pre-existing neurological deficit.

Head injured patients

Sixty seven patients (59 male,8 female) with severe head injury , aged between 5 and 74 years (mean 31 years) were

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Recording Techniques

Central Somatosensory Conduction Time (CCT)

Silver/silver chloride electrodes were applied to the scalp with collodion. The recorded signal was amplified (Tracor Northern, TN 3007 physiological pre-amplifier), averaged (Tracor Northern, TN 3000) and plotted on an XY recorder (Hewlett packard 7010 B).

Electrodes placed over the parietal region (7 cms. lateral to the vertex) and over the spinous process of second cervical vertebra (C_2), simultaneously recorded the somatosensory evoked potential in response to the stimulation of contralateral median nerve with a 0.2 milisecond constant current square wave pulse delivered at 4.7 H_z (parietal and C_2 electrode-active, forehead-reference). The depolarizing pulse intensity was determined by observing the onset of thumb twitch and raising the stimulus intensity 2mA above threshold. The sweep length was 50 milliseconds; 1000 sweeps were averaged; the pre-amplifier bandwidth ranged from 10 H_z to 3 KH_z

The peak latencies of major negative wave in the neck (N14) and the initial negative wave from the contralateral scalp electrode (N20) were measured with cursers and subtracted to provide the somatosensory central conduction time. The study was repeated twice on each side, and a mean value of CCT obtained for each hemisphere. The shorter latency in either hemisphere was recorded as the "best" conduction time and only conduction times of the best hemispheres were analyzed.

For analysis, CCT was categorised into three gropus: normal, abnormal (more than 2SD above the mean latencies of the normal subjects) and absent.

Intracranial Pressure (ICP)

Intracranial pressure was monitored continuosly by an intraventricular catheter connected to a pressure transducer and Gould pen recorder. The mean blood pressure (MBP) was also recorded simultaneously.

Procedures

Two kinds of recording were carried out. First, CCT, ICP, and MBP were recorded simultaneously at the different levels of ICP for each patient without attempting to reduce the ICP. Second, in twenty patients the same recordings were made before and after intravenous administration of a bolus of 20% mannitol (0.25-0.5 /kg).

Rusits

Normal subjects

Table I shows the range of values, mean latencies and standard deviations obtained in the 23 normal subjects for the CCT.

Table I. Median, mean and standard deviation of the CCT in each hemisphere in 23 normal subjects.

CCT	median (msec)	mean (msec)	SD (msec)
right	5.6	5.67 ± 0.09 (5.0 - 6.6)	0.42
left	5.5	5.66 ± 0.09 (5.1 - 6.5)	0.41

Patients

At the time of examination 49 of the 67 patients had a coma score of 8 or less, and of these 13 had a coma score of 5 or less.There was a significant correlation between the CCT and coma score (r= -0.43, P<.001), but a strongest correlation was found between the CCT and motor response (r= -0.64, P<.001). However, there was no relationship between coma score and ICP; patients who were in coma at the time of study showed a correlation of r= -26, P>..05. The same correlation was r= -0.009 for noncomatose patients.

Mean ICP and CCT

Figure 1 and 2 illustrates the latency values for the CCT plotted against ICP and CPP. A low but significant correlations are seen between CCT and ICP (r= 0.34, P<.001), and also between CCT and CCP (r= -0.35, P<.001).

The CCT was normal in 63 records. Of these, the mean ICP value was under 15 mmHg in 25, between 15-30 mmHg in 25, between 31-40 mmHg in 8, and over 40 mmHg in 5 records. In 52 abnormal CCT records, the mean ICP value was under 15 mmHg in 25, between 15-30 mmHg in 8, between 31-40 mmHg in 6, and over 40 mmHg in 13 records (Table II).

Table II. Relationship between mean ICP and CCT

ICP mmHg	CCT Normal	Abnormal		
< 15	25	25		
≥ 15	38	27	X^2= 0.817	P> 0.05
< 30	50	33		
≥ 30	13	19	X^2= 2.839	P> 0.05
< 40	58	39		
≥ 40	5	13	X^2= 5.057	P<0.05

Pressure Waves and CCT

There were no pressure waves in 87 records, of these , the CCT was normal in 52 and abnormal in 35 records. In II records with A waves the CCT was normal in 5, abnormal in 6 records. In 17 records with B waves, the CCT was normal in 14 and abnormal in 3 records (Table III).

Table III. CCT-ICP waves relationships

CCT	ICP no wave	A wave	B wave
Normal	52	5	14
Abnormal	35	6	3

Mannitol and CCT

In mannitol administered group, the correlation between CCT and ICP before mannitol statistically was not significant (r= 0.37, P>.05). After the mannitol administration, however, this relationship became significant (r= 0.50, P<.05). The

correlation between CCT and CPP was found to be; r= -0.59, P<.01 before mannitol, and r= 0.43, P>.05 after mannitol. On the other hand, mannitol had no significant effect on CCT, ICP, and CPP (Table IV).

Table IV. Relationships between CCT, ICP, CPP in mannitol administered patients.

	before mannitol	after mannitol	
CCT (msec)	(1) 6.83 ± 0.30 SD.1.34	(4) 6.79 ± 0.30 SD.1.36	P>.05
ICP (mmHg)	(2) 19.65 ± 2.69 SD.12.02	(5) 19.50 ± 3.52 SD.15.76	P>.05
CPP (mmHg)	(3) 73.8 ± 3.48 SD. 15.54	(6) 78.65 ± 3.9 SD.17.46	P>.05

1x2. r= 0.37, P> .05
1x3. r= -0.59, P<. 01
4x5. r= 0.50, P <.05
4x6. r= -0.43, P>.05

Discussion

Researsch into the correlations between ICP and spontaneous cerebral activity represented by EEG failed to yield the expected results, in the beginning (15, 28). During traumatic coma some relationship seems to exist between ICP variations and EEG changes (6,17). The direct cortical response has been shown to be quite sensitive to elevation of ICP when CBF was reduced (11). Evoked responses (SEP and EP), however, were considerabley more resistant to changes in CPP (7, 11,24) although VEP may be somewhat less resistant (23, 29, 30). A close relationship have been suggested between elevation of ICP and increased latency of the N_2 wave of the VEP in hydrocephalic patients (29). Potential effects of ICP on evoked potentials have been hypothesized by many authors (8,10,14,19-21,24,27,29,30). The mechanism whereby increase in ICP following head trauma might produce the prolonged CCT latencies is uncertain. The CCT represents conduction in the somatosensory pathway between the dorsal column nuclei and the somatosensory cortex , and it reflects the pathway's integrity (12,14). Because of its length in the CNS, running from the dorsal column nuclei in the spinomedullary junction, through the thalamus, and into the sensory cortex, CCT can be affected by ischemia at anypoint along the way. It has been suggested that correlation in head-injured patients between ICP and evoked potentials is on the basis of decreased CBF and oxidative metabolism produced by increase in ICP above critical

levels (10). In our patients, significant increase in CCT latencies occured at ICP levels well below those that should produce reductions in CPP sufficient to alter CBF or cerebral metabolism, assuming normal autoregulation was present (4). However, autoregulation is frequently disturbed either locally or globally in severe head injured patients (4,5). Therefore, the prolonged latency noted in some our patients might be related in part to reductions in regional CBF secondary to reductions in CPP produced by elevations in ICP.

The concept of ischemic thresholds has been well established experimentally in resent years (2,3,12,13,16) and the CCT has been shown to be a sensitive indicator of impending ischemia in patients with subarachnoid hemorrhage (22,26). Nevertheless, results of these studies are difficult to apply to patients with head injury. Experimental models of concussion and coma have demonstrated that somatosonsory evoked potentials are altered at various levels along the somatosensory way, without evidence of prolonged changes in ICP (20,21,27).

The rise in CBF is independent of the effect of mannitol on ICP (5). If brain cell activity measured by CCT is reduced because of inadequate CBF, improvement in CBF brought about by mannitol might improve CCT. Following the mannitol administration, although ICP and SBP changed slightly in a few patients in response to mannitol, there was no significant difference between the change in CCT before and after mannitol. This finding was not surprising since there is no correlation between ICP or CBF and neurological status or $CMRO_2$ except at very high levels of ICP (5). ICP does not correlate well with the severity of brain injury measured by clinical criteria.

The disturbances of cortical electrical conduction might be influenced by elevations of ICP in the presence of cerebral edema (4,9,19,25). Cerebral compression, as might occur in a diffuse manner with increased ICP, may produce reduction in conduction velocity as a result of axonal alterations such as changes in hydrostatic pressure or bulk flow of axoplasm which in turn could result in increased evoked potential latencies (19). Our findings could not be explained on the basis of this suggestion since patients had variable degrees of cerebral swelling at the time latency shifts were noted in the CCT in association with elevation of ICP. Therefore, there is no reason to expect that the intracranial hypertension would produce changes in CCT secondary to compromised hydrostatic pressure of axoplasm.

Whatever the mechanism for prolongation of CCT, the present study suggests that CCT does not provide an index that may be used in the diagnosis and treatment of increased ICP in patients with cerebral edema secondary to head injury.

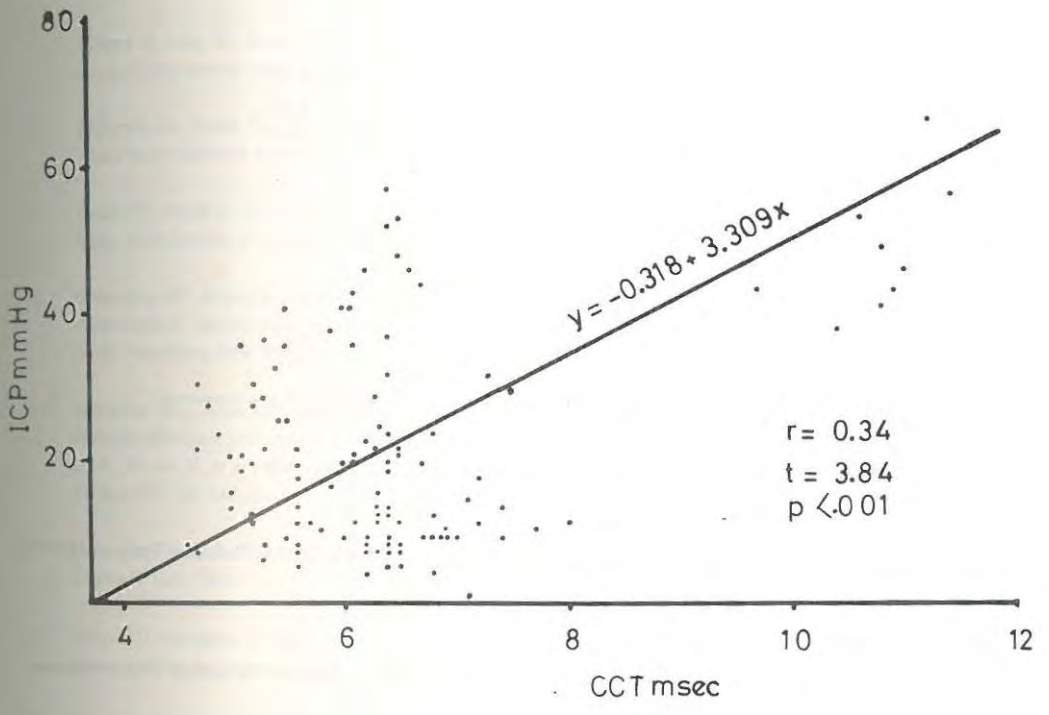

Figure 1. Latency values for Central Conduction Time (CCT) plotted against Intracranial Pressure (ICP)

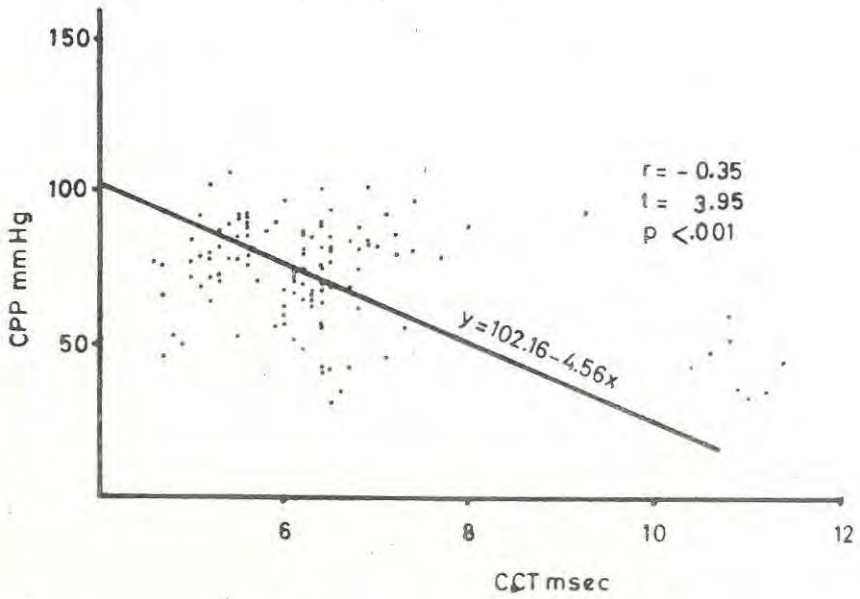

Figure 2. Latency values for Central Conduction Time (CCT) plotted against Central Perfusion Pressure (CPP)

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