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Correlation of Intracranial Pressure and Transcranial Doppler Resistive Index after Head Trauma

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PURPOSE: To investigate the usefulness of transcranial Doppler ultrasonography in the evaluation of intracranial pressure changes after head injury. **METHODS:** Transcranial Doppler examinations and intracranial pressure measurements using intraparenchymal monitors were performed in 12 cases of closed head injury. Twenty-four sets of data, including the Glasgow Coma Scale, intracranial pressure, transcranial Doppler, and carbon dioxide pressure were compared. The side-to-side difference in the resistivity index was also assessed. **RESULTS:** There was a significant correlation between increased pressure values and resistivity index when comparing measurements from the same side. There was no correlation between carbon dioxide pressure and any transcranial Doppler parameter or intracranial pressure measurement. No significant correlation was found between the resistivity index and the Glasgow Coma Scale. End diastolic velocity was a stronger determinant of resistivity index than peak systolic velocity. **CONCLUSION:** The relationship of ipsilateral measurements of intracranial pressure to resistivity index is valid. The resistivity index must be analyzed within the context of the particular disease studied, especially with respect to the hemodynamic alterations. Initial findings suggest that intracranial pressure monitoring cannot be replaced by serial transcranial Doppler measurements in the treatment of the patient with acute head injury.

Index terms: Head, injuries; Head, ultrasound; Ultrasound, Doppler; Brain, pressure

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Intracranial pressure affects cerebral pathophysiology and, if elevated in the patient with head injury, is associated with ongoing clinical deterioration and poor outcome, placing patients at risk for sudden cerebral herniation or delayed ischemic deficits (1–3). Rapid and accurate recognition of intracranial pressure fluctuation is critical for treatment of patients with head trauma. The most common method by which intracranial pressure is followed is invasive monitoring with direct and continuous pressure measurements taken from the epidural, subdural, or intraparenchymal compartment (4). Decision for placement of intracranial pressure monitors is based on clinical examination as reflected in Glasgow Coma Scale scores. Proper treatment is implemented depending on intracranial pressure measurements and other values, including arterial blood gases, serum osmolarity, hematocrit, and arterial blood pressure.

Transcranial Doppler ultrasonography is a noninvasive tool for investigating cerebrovascular hemodynamics. Indexes of pulsatility, derived from blood flow velocity measurements within major cerebral arteries, obtained by transcranial Doppler have been reported to correlate positively with cerebrovascular resistance (5, 6). A correlation between resistivity index and intracranial pressure was noted from both experimental study and work on hydrocephalus (7–9). There has been little investigation of the value of resistivity index in patients with severe head injury. However, transcranial Doppler is portable, safe, and noninvasive, which makes it suitable for monitoring critically ill patients with

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head injuries who require frequent intracranial pressure follow-up within an intensive care setting. This motivated us to evaluate the relationship between intracranial pressure values obtained by direct pressure measurements and the resistivity index.

Materials and Methods

Our research plan entailed studying a group of patients with head injuries with intracranial pressure monitors and comparing intracranial pressure measurements with carbon dioxide pressure and transcranial Doppler parameters, in particular the resistivity index. Data from 12 patients (9 male, 3 female; 4 to 52 years of age) with closed head injuries were studied. All subjects had admission Glasgow Coma Scale score of 9 or less, for which intracranial pressure monitoring was indicated (10). Hyperventilation, diuretics, and head-of-bed elevation were used in an effort to normalize intracranial pressure. Routine laboratory tests, including hematocrit and arterial blood gases, were obtained on a daily basis. Blood pressure was recorded hourly. Intraparenchymal intracranial pressure monitors (Camino, San Diego, Calif) were used, rather than subdural or epidural systems, because of their reliability and ease of placement and maintenance. Monitors were placed in the right frontal area in 11 patients. One patient had an intracranial pressure monitor placed on the left because of scalp injuries on the right side. Intracranial pressure was continuously monitored and recorded every hour, or more frequently when values were markedly elevated.

Transcranial Doppler examinations were performed with a Transpect system (Medasonics, Fremont, Calif), using a 2-MHz pulsed transducer, according to technique described by Aaslid in 1982 (11). Both middle cerebral arteries were insonated through the temporal squamosa. Although other segments of the basal arterial system were examined, the results were not used for the study, because the blunt angle of vessel insonation in these cases produces significant error in computing absolute blood flow velocities from Doppler shift. This problem is diminished when insonating middle cerebral arteries transtemporally, because the angle is closer to zero and produces less error (12, 13). In addition, because the middle cerebral artery is a large terminal artery, it is less subject to anatomic variants, and its signal is easier to find even when vascular configuration is altered by traumatic lesions. Peak systolic velocities, end diastolic velocities, and resistivity indexes were determined for both middle cerebral arteries. The resistivity index was calculated according to the formula: peak systolic velocity − end diastolic velocity/peak systolic velocity (14). Values were compared with age-related norms established for the adult and pediatric populations (15, 16).

Transcranial Doppler results were correlated with intracranial pressure values obtained within 20 minutes of each other. Transcranial Doppler data from the same side as the intracranial pressure monitor were analyzed. Side-to-side differences in resistivity indexes were calculated. Average resistivity index measurements for both sides were calculated. In an attempt to follow the relationship between intracranial pressure variability and related resistivity index changes, 11 of 12 patients had second transcranial Doppler studies with respective intracranial pressure data compared 2 to 3 days after the first studies. All patients had carbon dioxide pressure, hematocrit, and blood pressure values recorded at the time of intracranial pressure and transcranial Doppler comparisons, because they are known to affect blood flow velocities in cerebral arteries (17).

Correlational statistics were performed to assess any statistically significant relationship between intracranial pressure values measured by monitor and resistivity index, peak systolic velocity, end diastolic velocity, and carbon dioxide pressure. The difference between two subsequent sets of results obtained on 11 of 12 patients was also compared. The Pearson correlation coefficient was calculated and evaluated.

Results

Intracranial pressure, carbon dioxide pressure, and transcranial Doppler parameters included in statistical analysis are summarized in Table 1. Twenty-four intracranial pressure measurements were obtained in 12 patients. The values ranged from 3 to 42 mm Hg. Eight patients had initial and subsequent values less than 20 mm Hg, which was considered the upper limit of normal (18). In two cases, both measurements exceeded 20 mm Hg. Two patients had one of two measurements abnormally elevated. Carbon dioxide pressure readings were, with one exception, less than 35 mm Hg. The carbon dioxide pressure was lowered by hyperventilation therapy. Blood pressure was maintained within normal limits. Hematocrit was in the low range of normal in all patients.

Transcranial Doppler parameters from 23 of 24 studies were obtained. One study was eliminated because of suboptimal quality (uncooperative, agitated patient). The resistivity index values were between 0.41 and 1. The majority of results oscillated along the upper limits of normal (resistivity index = 0.7) as per Aaslid (16). One patient had markedly increased resistivity indexes in both studies. This was patient 3, in whom persistent elevation of intracranial pressure was also noted. The side-to-side differences in resistivity indexes from 23 transcranial Doppler examinations are shown in Table 2. In 9 cases, the difference was equal to or greater than 10%.

TABLE 1: Analyzed data from 12 head-injured patients

Patient	Glasgow Coma Scale Score, Intubated	Intracranial Pressure, mm Hg	Resistivity Index	Peak Systolic Velocity, cm/s	End Diastolic Velocity, cm/s	Carbon Dioxide Pressure
1	6	13				
		3	0.56	101	45	34
2	6	6	0.72	77	21	25
		15	0.87	93	12	29
3	6	35	1.00	50	0	26
		42	0.89	32	4	36
4	7	14	0.73	71	20	27
		15	0.63	107	47	25
5	6	6	0.67	123	41	29
		7	0.47	86	45	27
6	4	15	0.56	158	70	34
		15	0.73	96	27	32
7	4	30	0.72	121	34	20
		9	0.53	135	61	30
8	4	15	0.55	112	50	26
		10	0.41	134	78	32
9	3	17	0.72	140	39	25
		8	0.53	94	45	30
10	7	7	0.72	66	19	23
		12	0.82	68	11	25
11	7	30	0.77	133	31	26
		27	0.74	116	31	27
12	7	10	0.61	112	57	27
		25	0.47	148	69	30

Note.—Parameters are right-sided except for patients 3 and 11.

TABLE 2: Side-to-side difference in resistivity index

Patient	Resistivity Index, Right	Resistivity Index, Left	Difference, Right/Left, %
1	0.56	0.45	11
2	0.72	0.60	12
	0.87	0.65	22
3	1.00	1.00	0
	0.53	0.89	36
4	0.73	0.63	10
	0.63	0.65	2
5	0.67	0.77	10
	0.47	0.56	9
6	0.56	0.56	0
	0.73	0.72	1
7	0.72	0.76	4
	0.53	0.61	8
8	0.55	0.56	1
	0.41	0.63	22
9	0.72	0.77	5
	0.53	0.51	2
10	0.72	0.73	1
	0.82	0.68	14
11	0.72	0.77	5
	0.75	0.74	1
12	0.61	0.61	0
	0.47	0.62	15

Significant correlation was found between ipsilateral resistivity index (RI) and intracranial pressure (ICP) values ($P < .05$) (Fig 1) with coefficients calculated for pairs ICP1 – RI1 and ICP2 – R12, where 1 and 2 describe, respectively, the first and second tests on one patient ($r1 = .6148$; $r2 = .6062$). Using $P < .001$, the relationship was approaching significance because of small numbers in each category (12 and 11). There was no significant relationship between intracranial pressure and resistivity index when resistivity index was calculated as a mean value for indices of both sides together. There was very strong inverse correlation between RI1 and end diastolic velocity (EDV)1 ($r = -.8756$; $P < .001$) and RI2 and EDV2 ($r = -.9333$; $P < .001$). The relationship between peak systolic velocity (PSV) and resistivity index was also significant but weaker: RI1/PSV1, $r = -.6215$; $P < .001$; and RI2/PSV2, $r = -.6911$; $P < .001$. No statistically significant relationship was found between carbon dioxide pressure and any of included variables. The resistivity index and Glasgow Coma Scale did not correlate with statistical significance in this small series.

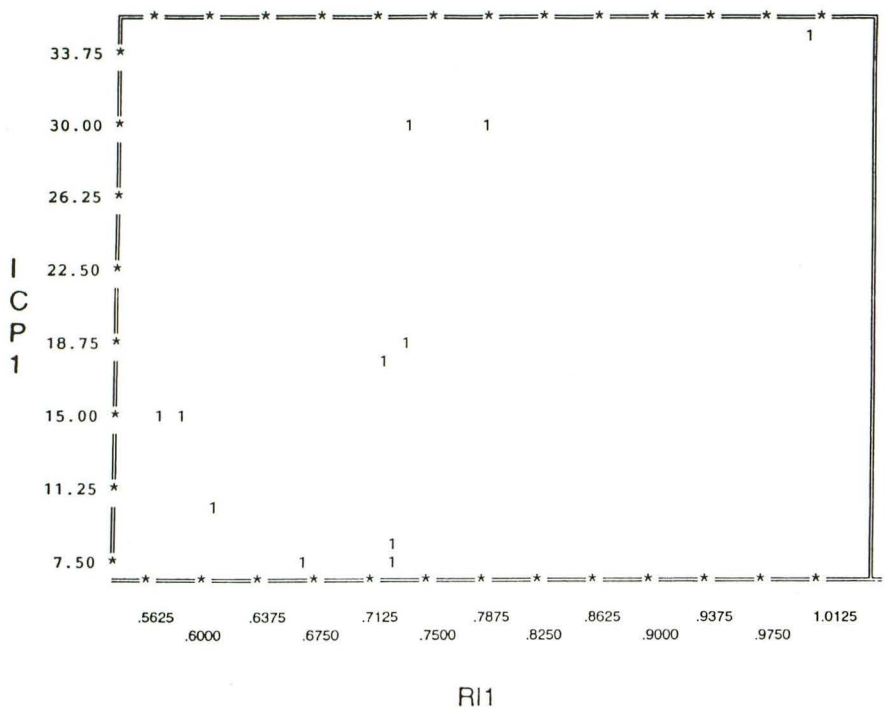

Fig 1. Significant correlation between ICP1 and RI1 in 11 patients with head injuries ($r1 = .6148$; $P < .05$).

Discussion

The Pourcelot resistivity index was shown to increase with a rise in intracranial pressure in neonatal hydrocephalus in an early study by Bada et al (8). The relationship between the two parameters in patients after severe head injury is subject to complex multifactorial influences, which makes it difficult to assess reciprocal interactions. In our study, the resistivity index correlated well with intracranial pressure values when both tests were obtained from the same hemisphere. However, despite the overall trend, there was a considerable spread of the data (Figs 1 and 2). The range of normal resistivity index is broad, between 0.3 and 0.7, so that comparison to a narrow normal range of intracranial pressure was not successful (12, 16). Similar results were obtained by Klingelhofer et al, who evaluated the RI – ICP correlation in 13 comatose patients with varied cerebral diseases (19). In both our series and that of Klingelhofer, the patients had low Glasgow Coma Scale scores. Sanker et al noticed the rise of pulsatility index, another transcranial Doppler parameter (PSV – EDV/mean velocity), in two children with posttraumatic brain edemas (20).

The side-to-side differences in the resistivity index seemed to be an important aspect in investigating the correlation with intracranial hypertension in our group of patients. This originates from the specificity of posttraumatic brain damage, which is frequently unilateral, predominantly unilateral, or with asymmetric involvement of the two cerebral hemispheres. In 9 of 23 studies in our group, differences between right and left resistivity indexes were equal to or greater than 10%. There was no correlation between intracranial pressure and resistivity index calculated as an average value from both middle cerebral artery measurements.

Our results confirm that the resistivity index is mainly modified by end diastolic velocity variability (19). Peak systolic velocity is less of a resistivity index determinant, but its influence cannot be ignored, because it was also significantly inversely correlated with the resistivity index (Fig 3).

The carbon dioxide pressure did not demonstrate any significant relationship with any of the analyzed transcranial Doppler parameters or with intracranial pressure. This differs from the results reported from the studies on healthy volunteers, in which strict relationships were determined. In the study by Vriens et al, blood velocity decreased with a reduction of carbon dioxide pressure. In the same study, however, the authors observed that this relationship concerns individual carbon dioxide pressure reactivity and that patients with high or low blood flow velocities at one carbon dioxide pressure

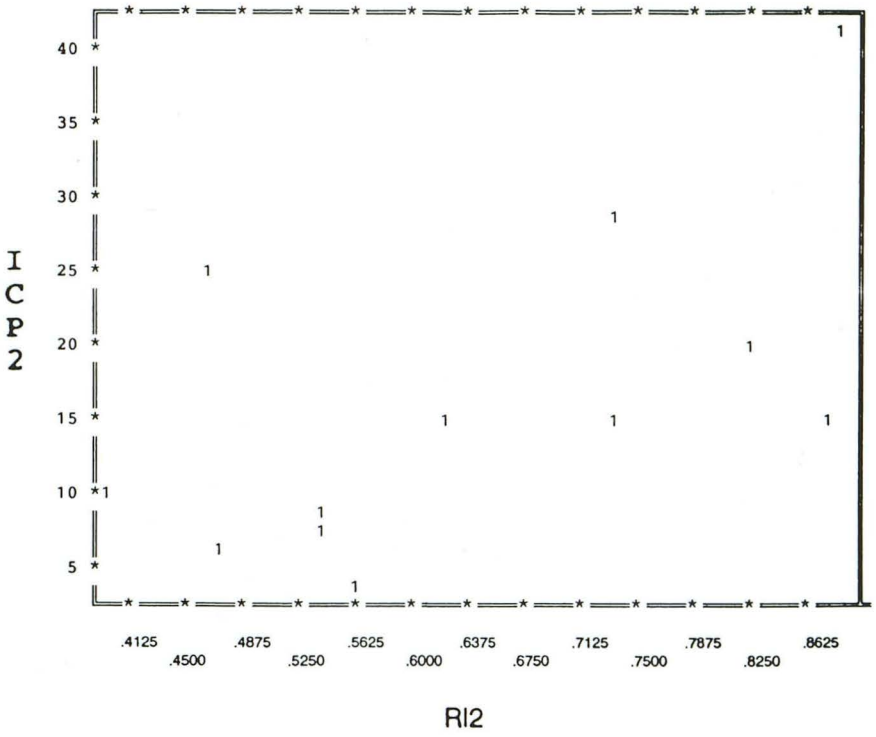

Fig 2. Significant correlation between ICP2 and RI2 in 12 patients with head injuries ($r2 = .6062$; $P < .05$).

level tend to maintain relatively high or low blood flow velocities at another pressure level (21). Markwalder et al, investigating individual reactivity to hypocapnia, found an exponential relationship, without significant difference between varied age groups (22).

These findings can be helpful in assessing individual reactivity to changes in carbon dioxide pressure and can be extrapolated for patients with presumed intact autoregulation. This does not apply to our group, because all patients had low Glasgow Coma Scale scores (less than 9), with presumably impaired autoregulation (23, 24). The assumption that blood flow velocities within basal cerebral arteries reflect carbon dioxide pressure–induced changes in arteriolar blood flow cannot be applied in this situation. Investigators noted that, in healthy

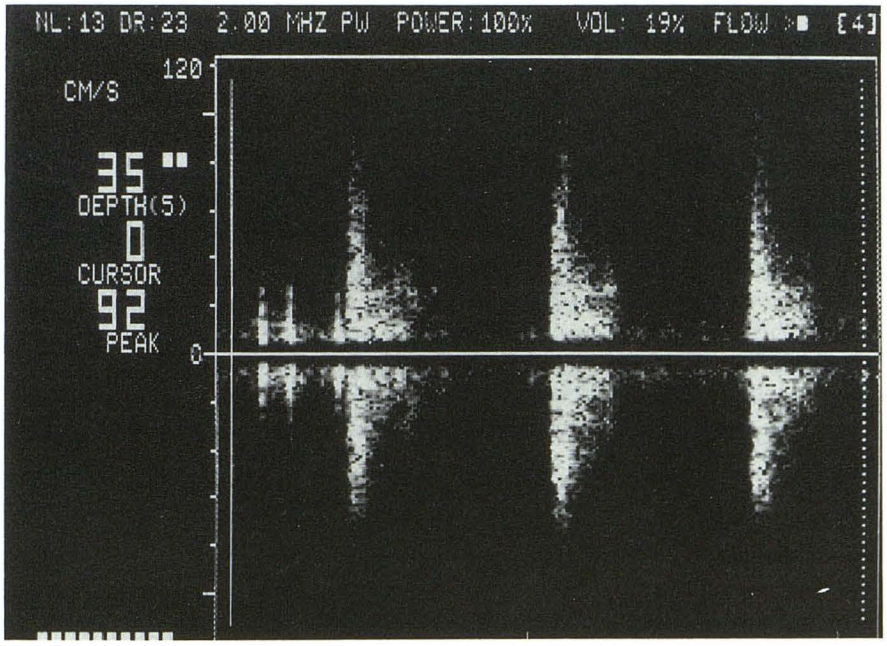

Fig 3. Resistivity index calculated using the formula PSV − EDV/PSV is normal (0.56) in a patient with a head injury with intracranial pressure of 15 (normal intracranial pressure, less than 20).

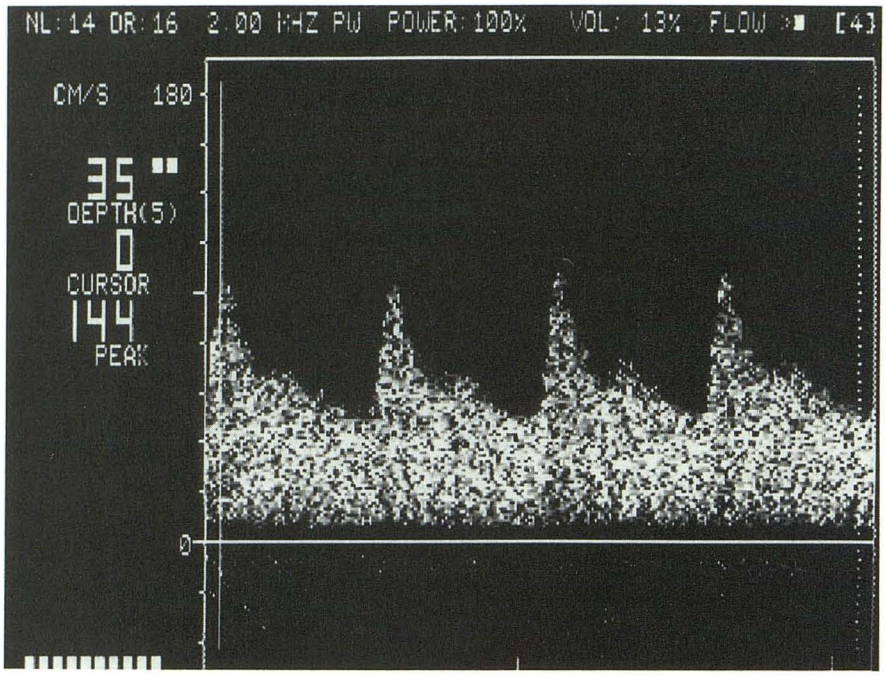

Fig 4. Cessation of anterograde flow in diastole with resistivity index approaching 1 in a patient with a head injury with elevated intracranial pressure (pressure of 35).

subjects, arteries within the circle of Willis did not change their calibers in response to changes in carbon dioxide pressure (25). The decreases in the calibers of peripheral arterioles are reflected by changes in blood flow velocities in main cerebral arteries (22).

In addition to impaired autoregulation, other pathologic mechanisms can alter blood flow velocities in main cerebral arteries. The best-recognized are posttraumatic arterial vasospasm and hyperemia, both causing increased flow velocities (26, 27). Assuming the inverse correlation between resistivity index and blood flow velocity, the resistivity index could decrease in the presence of any of these processes, without a corresponding increase in intracranial pressure (Fig 4). In vasospasm, found to be a frequent posttraumatic sequela, the basal arteries narrow and no longer directly reflect arteriolar reactivity (28). The group studied here had specific alterations in cerebrovascular hemodynamics, as noted. Therefore, our results cannot be applied to the general population with preserved autoregulation or to patients with cerebrovascular pathophysiology without considering the specific underlying mechanism.

The studied group presented very small interindividual differences in several aspects (eg, blood pressure, hematocrit, serum osmolarity, and carbon dioxide pressure) because these were kept within a narrow range. This factor also diminished interindividual variability of physiologic reactions.

According to Mamarou, there are two vascular mechanisms leading to increases in intracranial pressure, with cerebrospinal fluid factors playing a lesser role (29). Increased intracranial vascular pressure results from vascular engorgement caused by impaired autoregulation and hyperemia, or is from brain edema. Cerebrospinal fluid pressures rise as a result of the raised vascular pressure, although the exact site of vascular pressure rise has not been identified. This supports the use of transcranial Doppler measurements and indexes to evaluate intracranial pressure. Furthermore, Miles et al, comparing six transcranial Doppler parameters in an arterial model in vitro, found resistivity index correlated best with flow (30).

Our recommendation is that transcranial Doppler evaluation in head injury be implemented according to the following schedule:

1. Initial study, day of injury (or within 24 hours);
2. Day 2 after injury (48 hours);
3. Day 3 to evaluate at time of maximal brain swelling (72 hours);
4. Day 7 to follow up peak systolic velocity and evaluate for resolution of spasm versus hyperemia;
5. As needed if patient deteriorates; and
6. Days 10 to 14 for baseline, or before hospital discharge.

In an actual clinical situation, the complexity of these relationships should be kept in mind.

The resistivity index is an indirect assessment of intracranial pressure but cannot be analyzed independently from other pathophysiologic factors induced by the original disease entity and its treatment.

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