Evaluation of Intracranial Pressure by Transcranial Doppler Ultrasonography in Dogs with Intracranial Hypertension

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ABSTRACT. Transcranial Doppler ultrasonography (TCD) has been used to confirm changes in cerebral hemodynamics. In this study, we investigated whether the parameters for the basilar artery measured by TCD were correlated with the intracranial and cerebral perfusion pressures in extreme intracranial hypertension. An intracranial hypertension model was produced in seven dogs by inflating a balloon inserted into the epidural space. The resistance index was compared with the corresponding intracranial pressure and cerebral perfusion pressure values during intracranial hypertension. A significant correlation was recognized between the resistance index and cerebral perfusion pressure. Therefore, measurement of the basilar artery by TCD in the dog with intracranial hypertension is useful in estimating the intracranial circulation in cases where the measurement of intracranial pressure is not available or not indicated.—KEY WORDS: canine, intracranial hypertension, transcranial Doppler ultrasonography.

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Hydrocephalus, brain tumor and brain contusion may all cause the intracranial pressure (ICP) to rise. The most critical effect of an increase in ICP is on cerebral perfusion. Intracranial hypertension causes nervous ischemia, hypoxia and brain functional disorder, when it lasts. It finally develops into brain death [9]. Rapid recognition of ICP or cerebral perfusion pressure (CPP) fluctuation is necessary for the treatment of patients with intracranial hypertension [10]. The methods which are used most frequently for monitoring cerebral perfusion and ICP are intraventricular, intraparenchymal compartment or similar [8], but they are invasive and can cause complications [2].

Transcranial Doppler ultrasonography (TCD) was introduced by Aaslid *et al.* [1] and is a non-invasive technique which may be used repeatedly, allowing real-time measurement of blood flow velocity in the major cerebral arteries in humans. TCD can be used to confirm changes in cerebral hemodynamics. The brain disease and cerebral blood flow decrease which occurs secondarily in dogs can be evaluated if we can estimate the degree of ICP when it rises, by using TCD. In this study, we investigated whether the parameters of the basilar artery measured by TCD were correlated with ICP and CPP in extreme intracranial hypertension.

Seven adult mongrel dogs (mean weight 10.8 ± 2.8 kg, 3, 4) were used in this study. All the dogs were considered to be normal following physical and hemodiagnostic (complete blood count) examinations and a *Dirofilaria immitis* immunodiagnostic test (SNAP; IDEXX Laboratories Co., U.S.A.).

Anesthesia was induced with intravenous thiopental sodium (25 mg/kg) and maintained following tracheal intubation with 1.5% end-tidal isoflurane in 100% oxygen.

Paralysis was achieved by iv. administration of pancuronium bromide (0.25 mg/kg). End-tidal CO₂ was maintained with a pressure of approximately 35 mmHg using a multi gas monitor (Capnomac; DATEX Instrumentarium Co., Finland). Body temperature was measured with the aid of esophagus thermisters and maintained at approximately 38°C using a servo-controlled heating pad. The electrocardiogram was monitored with the aid of needle electrodes. Cannulation of the femoral artery on the right side was performed to allow the direct measurement of mean arterial blood pressure. A muscle relaxant was administered during the experiment, as necessary, to stabilize anesthesia.

Intracranial pressure was measured using an indwelling catheter that was placed through a burr hole in the skull at the level of the lateral ventricle. The burr hole was made in the right parietal region and used to insert a balloon into the epidural space [4]. The balloon was inflated using a syringe pump to elevate ICP to each 5 mmHg interval between 10–100 mmHg. The heart rate, mean arterial blood pressure and intracranial pressure were recorded with a cardiac monitor (Life-scope 11; Nihon Koden, Japan).

Cerebral blood flow velocity in the basilar artery was measured with the a digital ultrasound system using a multihertz (5.0–12.0 MHz) sector scanning probe (HP SONOS-5500 & S12; Hewlett Packard Co., U.S.A.) through the foramen magnum acoustic window using the method which we have reported before [5]. Once identified, pulsed wave Doppler was initiated and a spectral tracing with at least three sequential similar spectral waveforms was collected. Measurements were made on a representative spectral waveform to determine systolic peak velocity (Vp), end-diastolic peak velocity (Vd), mean velocity (Vm), pulsatility index (PI) [$PI = (Vp - Vd) / Vm$] and resistance index

Fig 1. The both figure represent the mean. ICP=intracranial pressure, PI=pulsatility index, RI=resistance index, Vp=systolic peak velocity, Vm=mean velocity, Vd=end-diastolic velocity. The left figure shows progressive decrease of cerebral blood flow velocity (Vp, Vm, Vd) with change of intracranial hypertension. The right figure shows increase of PI and RI with change of intracranial hypertension.

Fig 2. Scatterplot shows the correlation between the intracranial pressure (ICP) and resistance index (RI).

Fig 3. Scatterplot shows the correlation between the cerebral perfusion pressure (CPP) and resistance index (RI).

(RI) [$RI = (Vp - Vd) / Vp$]. CPP was calculated as $CPP = SABP$ (systemic arterial blood pressure) - ICP.

All dogs were subjected to arterial blood pressure, ICP and TCD monitoring. Values were analyzed using Pearson's correlation coefficients.

The cerebral blood flow velocity (Vp, Vd, Vm), PI and RI data during the changes in ICP between 10–100 mmHg are presented in Fig. 1. As the ICP increased, so did the PI and RI, although the Vd and Vm decreased. The Vd showed minus values at ICP of greater than 80 mmHg because the arterial cerebral blood flow reversed in the diastole. The ICP correlated with the corresponding RI ($r = 0.872$, $P < 0.001$), (Fig. 2). There was also correlation between the CPP and RI as the ICP increased ($r = -0.731$, $P < 0.001$) (Fig. 3). It appears that the RI value enables us to determine the changes in ICP ($Y = -37.35 + 109.85 * X$) and CPP ($Y = 157.41 - 116.24 * X$).

In this study, progressive intracranial hypertension was caused by a reduction in the flow velocity in the diastole

more rapidly than in the systole, with resulting increases in PI and RI. This phenomenon implies a decrease or loss of intracranial compliance with increased ICP. The PI and RI will increase in parallel to the decrease in the cerebral blood flow velocity until the latter ultimately causes arrest. The ICP was more closely correlated with RI than with PI. It is thought that RI can be useful in evaluating the degree of the increase in ICP. In progressive intracranial hypertension, an increase in microcirculatory resistance can cause intracranial circulatory disturbance [7, 11]. Therefore, it appears that an increase in RI corresponds to an increase in intracranial microcirculatory resistance.

The results of this experiment show that the RI correlates with ICP and CPP during an increase in ICP, indicating that changes in ICP and CPP can be evaluated by measuring the RI. However, we believe that it is necessary to measure the RI value carefully because changes in the values for PI and RI are affected by a number of factors, including an alteration in the hematocrit, metabolic factors such as CO_2

and O₂ concentration and the type of blood vessel [3, 6]. Therefore, TCD must be repeated with care, taking into account the influencing factors for each clinical case.

In conclusion, measurement of the basilar arterial flow velocity by TCD in the dog with intracranial hypertension is useful in estimating the intracranial circulation in cases where the measurement of ICP is not available or not indicated. In particular, we recognized the possibility of evaluating ICP ($ICP = -37.35 + 109.85 \times RI$) by measurement of the RI.

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