A LUMPED PARAMETER MODEL FOR THE SIMULATION OF CRANIAL VOLUME-PRESSURE TEST

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INTRODUCTION

The cranial volume-pressure test consists of recording the cranial pressure (P_{CSF}) while the volume of the cerebrospinal fluid (CSF) is varied. The relation between the added volume of CSF (ΔV_{CSF}) and P_{CSF} is referred to as a cranial volume-pressure curve. The curve has two flat regions, one around the normal value of P_{CSF} of approximately 1.3 kPa (10 mmHg), and the other around the point where P_{CSF} reaches the arterial blood pressure. Other zones of the curve are steep, with the steepest part for P_{CSF} exceeding the arterial pressure [1].

An elevated P_{CSF} has a profound effect on the cerebral circulation; P_{CSF} above 5.3 kPa (40 mmHg) results in reduced cerebral blood flow, and P_{CSF} above the arterial pressure cuts the flow to almost zero [2]. The coupling between P_{CSF} and the cranial vascular compartment is often attributed to the fact that the cranial volume must always remain constant. It is argued that the additional volume of CSF is accommodated by expelling blood from the intracranial veins, and that the volume-pressure curve reflects the compliance of the venous bed at different levels of P_{CSF} [3]. The experimental studies show that the intracranial veins are collapsed by introducing an additional volume of CSF; for P_{CSF} below 6.7 kPa (50 mmHg) there is always a point in the venous system where the pressure exceeds P_{CSF}, and at least a portion of the venous bed is open; for P_{CSF} above the arterial pressure the whole venous bed appears to be emptied [4-6].

Although the venous collapse during volume-pressure test is well documented, the connection between the cranial vascular compliance, vascular resistance and P_{CSF} is not completely clear. We propose a simple mathematical model that establishes that relation.

METHODS

The cranial cavity is occupied by CSF, blood, and brain all of which are essentially incompressible. It is assumed in the model that the skull is sufficiently stiff and that the changes in its volume can be neglected. It is further assumed that the changes in volume due to CSF production-absorption, and brain swelling can be neglected for the time scales of interest. Under the above assumptions, the sum of the CSF volume and the blood volume is constant.

The vascular bed is represented as a resistive circuit formed of three lumped compartments in series: arterial, proximal venous, and distal venous. The sinuses are represented as a constant resistor. The blood flow is driven by difference between the mean arterial pressure and the central venous pressure which are model constant inputs. The resistances, and volumes of the vascular compartments under the normal conditions ($\Delta V_{CSF}=0$), are taken from the literature. Under conditions other than normal they are determined from the equations derived from a one-dimensional theory of flow in collapsible tubes [7]. The equations provide the relation between the transmural pressure and compartmental volume, as well as the relation between the volume and the resistance. The volume-pressure law for the venous compartment is highly non-linear, with a flat region around zero transmural pressure, and two steep regions, one for high negative, and the other for high positive transmural pressure. The behaviour of the cranial system is described by eleven non-linear algebraic equations in terms of P_{CSF}, cerebral blood flow, the volumes and resistances of the vascular compartments, and the blood pressures at the ends of the compartments. The independent variable is ΔV_{CSF} which was varied from –0.25 to 0.95 of the total cerebral venous volume at normal conditions.

RESULTS

The results for the volume-pressure curve and cerebral blood flow are consistent with the experimental results reported in the literature (see Figures 1 and 2). The blood flow remains constant until $\Delta V_{CSF}>25$ cm^3 and $P_{CSF}>5.3$ kPa (40 mmHg). The volume pressure curve is flat for $0 < \Delta V_{CSF} < 20$ cm^3 and 20 $cm^3 < \Delta V_{CSF} < 35$ cm^3, and steep for 20 $cm^3 < \Delta V_{CSF} < 35$ cm^3 and $\Delta V_{CSF} > 40$ cm^3. The approximate borders of the characteristic zones are marked by points 1-4 in Figure 1. The shift from one zone to the other is marked by a significant change in the compliance or/and resistance of one of the venous compartments which occurs when the venous volume is altered. Between points 1 and 2 the blood is expelled from the distal venous compartment, without a significant increase in the venous resistance or a drop in the venous compliance. Between points 2 and 3 the collapse of the distal

bed has progressed to the point that its resistance increases dramatically, and compliance drops. Between points 3 and 4 the distal venous bed is severely collapsed, and extremely stiff. The blood is expelled from the proximal bed, which is highly compliant. At point 4 the collapse of the proximal venous compartment is severe and its compliance becomes low. The volume of the arterial compartment remains essentially unaltered for the range of ΔV_{CSF} used in the study. For negative ΔV_{CSF}, both venous compartments are inflated.

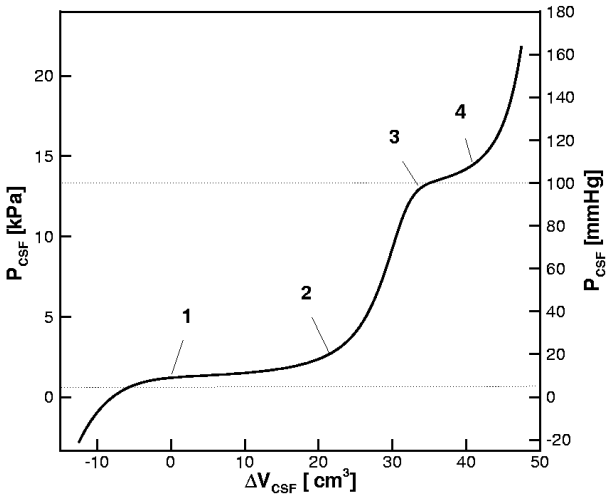

Figure 1. Cranial pressure (P_{CSF}) as a function of the added volume of cerebrospinal fluid (ΔV_{CSF}). Dotted lines give central blood pressures.

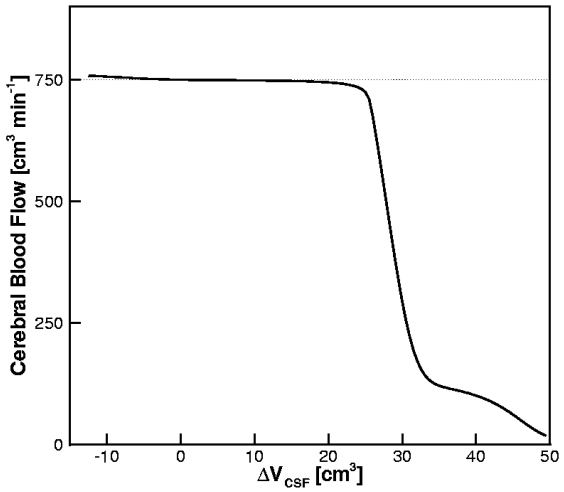

Figure 2. Cerebral blood flow as a function of the added volume of cerebrospinal fluid (ΔV_{CSF}). The dotted line gives the normal value of flow.

DISCUSSION

The changes in the venous pressure, resistance and compliance brought on by altering the volume of the cranial CSF compartment are complex and involve both the proximal and the distal venous bed. However, in the region of the volume-pressure curve bounded by points 1 and 3, which of the greatest clinical importance, the increase in the CSF is mainly achieved by compressing the distal venous bed i.e. medium size and large cerebral veins. The results suggest that the drop in cerebral blood flow is caused by venous collapse and elevated venous resistance. This is often interpreted in the literature as a drop in cerebral perfusion pressure, defined as the difference between arterial pressure and P_{CSF}, which approximately equals the venous pressure in the non-collapsed portion of the venous bed [8]. However, this interpretation becomes invalid when all of the veins are collapsed and the venous walls can support a significant negative transmural pressure.

CONCLUSIONS

The study suggests that the shape of the cranial volume-pressure curve is primarily determined by coupling of the CSF and venous cranial compartments. This results from the condition that the cranial volume is constant and that any change in the CSF volume must be accommodated by a complementary change in the cerebral venous volume. This profoundly affects the venous hemodynamics and is reflected in the cranial pressure. The arterial bed does not appear to play a significant role in determining the shape of the volume-pressure curve.

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