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CRANIAL AND SPINAL COMPONENTS OF THE CEREBROSPINAL FLUID PRESSURE-VOLUME CURVE

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ABSTRACT

A quantitative analysis of the contributions of the cranial and spinal compartments to the cerebrospinal fluid pressure-volume curve was made using dogs. The curve was determined by rapid continuous injection of fluid into the cisterna magna with simultaneous measurement of the pressure. Spinal block at the C 1 level was produced by inflation of an epidural rubber balloon allowing the recording of the pressure-volume curve for the isolated cranial system. By subtraction of the two curves obtained, the spinal pressure-volume curve could be calculated. 70 % of the variation in volume within the system was related to the spinal section and 30 % to the cranial section. The intracranial curve represents the effects on the fluid pressure of forced alterations in the volume of the intracranial vascular bed. The spinal compartment has a quantitatively defined and probably mechanically important function as an expansion vessel for the intracranial system.

The volume of the intracranial system is constant but variations may occur in its different components. While spatial compensation is possible with time, by means of a reciprocal change in the cerebrospinal fluid (CSF) volume, immediate adjustments are conceivable from a mechanical point of view, in two principal ways. One mechanism is a reciprocal change in the intracranial blood volume. Another possible mechanism is displacement of CSF to and from the spinal compartment, presumably involving in that case mainly changes in the total volume of the system.

The fundamental question as to whether a rapid change in the volume of an intracranial component takes place at the expense of a reciprocal change in the vascular volume, or by means of a change in the total volume, is controversial. The series of papers concerning "cerebrospinal elasticity" by *Weed* and co-workers (*Weed* 1929, *Weed et al.* 1932, *Flexner et al.* 1932) seems to have ended in a concept recognizing both kinds of volume adjustment. *Ryder et al.* (1953 a-b)

contended, however, that changes in CSF volume "lead to an equal and opposite change in the volume of the craniospinal venous bed rather than to an appreciable change in the total volume of the craniospinal contents". On the other hand it seems definitely established, by observations during Pantopaque myelography, that the spinal dural sac may be subjected to considerable dimensional variations, e. g. when changes are induced in the intracranial blood volume (*Martins et al.* 1972). These authors denied explicitly any significance of the craniospinal venous vascular bed in the elasticity of the system. The pressure-volume curve of the isolated spinal dural sac has been determined by *Gilland* (1965) in clinical cases of spinal block, and an exponential relationship demonstrated.

The stated problem may thus be less a matter of deciding in favour of one or the other of these hypotheses than of assessing the quantitative importance of each mechanism. The present experimental study was undertaken with this aim. The method used is based on the recording of the pressure-volume curve of the CSF space, before and after exclusion of the spinal division of the system by producing a block at the C 1 level. The pressure-volume of the intracranial system is thus obtained, and by subtracting this curve from the curve of the total craniospinal system, the spinal pressure-volume curve can be estimated. It was found technically unfeasible to obtain acceptable pressure-volume curves directly from the isolated spinal compartment, as the narrow fluid spaces precluded the free aspiration and injection of fluid.

MATERIAL AND METHODS

The experiments were performed on six dogs, ranging in weight between 18 and 25 kg. Brain weights, as determined after the experiment, were between 73.6 and 95.0 g. The dogs were anaesthetized with sodium pentobarbital (30 mg/kg i.v.) and given additional small doses as required. Artificial respiration was maintained by means of a respirator pump, using room air at intermittent positive pressure through an endotracheal tube. Muscle relaxation was produced by suxamethonium chloride. The dogs were placed horizontally in the right lateral position.

The CSF pressure-volume curve was determined with a technique described in another report (*Löfgren et al.* 1973). This report should also be consulted regarding the errors in the method. The cisterna magna was punctured percutaneously with a double-lumen cannula. One lumen was used for measuring the CSF pressure. The other lumen of the cannula was used for changing the volume of the fluid space. CSF was first aspirated to set the pressure to a slightly negative value, and thereafter a mixture of CSF and Ringer's solution at 38° C was forced into the fluid space at a rate of 0.25 ml/s, by means of a constant rate infusion pump. When the pressure approached 60–70 mmHg the injection was interrupted and fluid aspirated to reset the pressure to its initial value. The duration of the procedure was about 1 min.

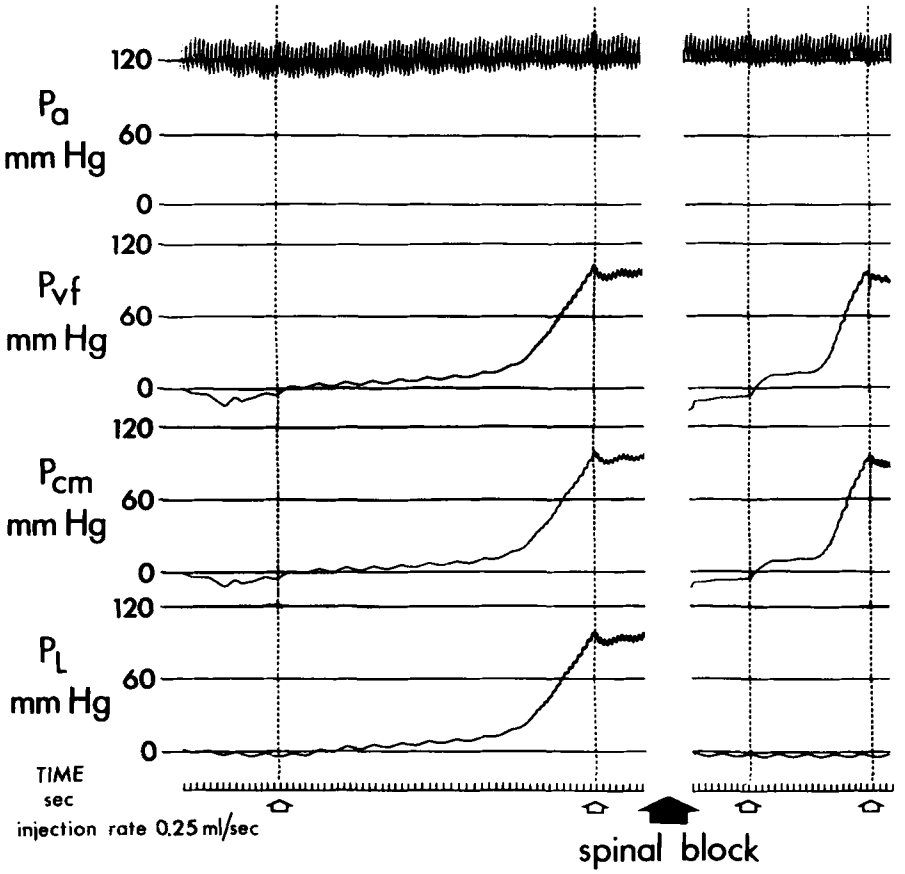

Figure 1. The CSF pressure-volume curve determined by injection of fluid into the cisterna magna before and after blocking the spinal canal at the C 1 level. The systemic arterial pressure (P_a), the ventricular fluid pressure (P_{vf}), the cisterna magna pressure (P_{cm}) and the lumbar pressure (P_l) are recorded. Note the absence of a pressure change in the lumbar space in the situation of spinal block, when the pressure-volume curve of the isolated intracranial system is recorded.

Pressures were also measured in one lateral ventricle (with a polyethylene catheter inserted through a parietal burr hole), in the lumbar subarachnoid space (with a catheter tied watertight into the dural sac after laminectomy at L 7), in the abdominal aorta (with a catheter via a femoral artery) and in the inferior vena cava (with a catheter via a femoral vein). The pressure were recorded by Statham P23AC transducers and displayed on a Grass Polygraph pen writer. Zero reference was the level of the spinal axis.

Reversible block of the spinal canal was achieved by inflation of a fluid-filled epidural rubber balloon. A midline exposure was made of the lamina of C 1, avoiding the region of the membrana atlanto-occipitalis. A burr hole with a diameter of 15 mm was made in the lamina. A metallic cylinder with an attached rubber

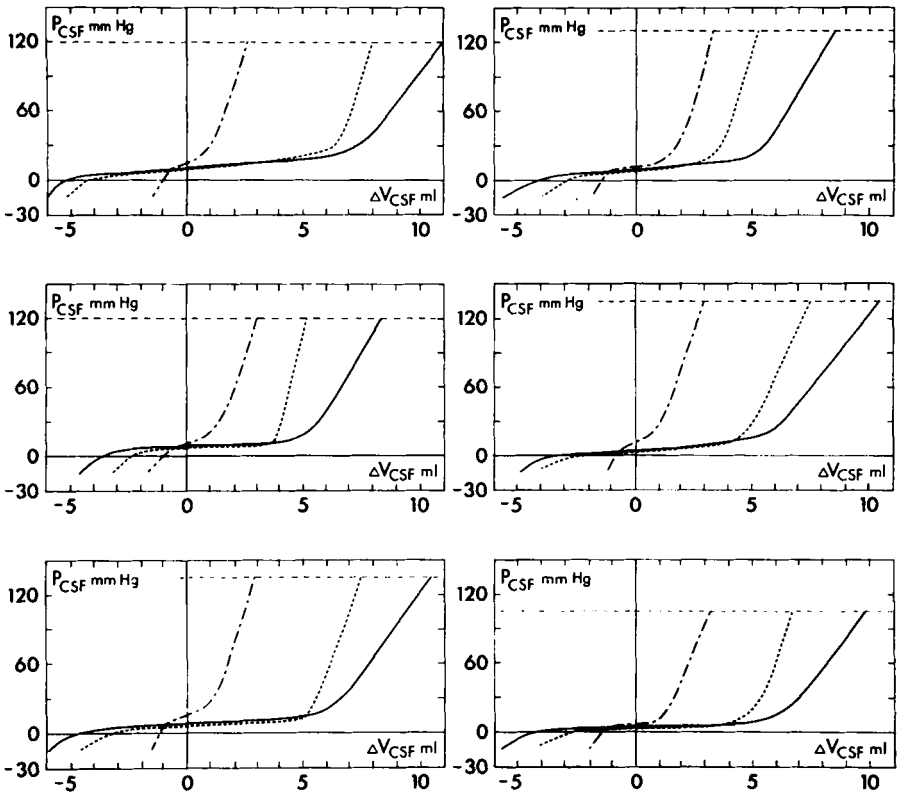

Figure 2. CSF pressure-volume graphs in 6 experiments. From right to left in each graph: the curve of the total system, the spinal curve and the cranial curve. The horizontal dotted lines denote the mean arterial blood pressure.

balloon at its lower end was screwed into the hole. The balloon was collapsed initially and could be inflated by a syringe.

Neck compression for the purpose of raising the cephalic venous pressure was accomplished by a sphygmomanometer cuff placed around the neck below the balloon device and inflated to various pressures.

RESULTS

After recording the pressure-volume curve of the unobstructed CSF system, spinal block was produced at the C 1 level by inflation of the epidural balloon. Too much inflation caused a severe increase in the blood pressure and bradycardia. This reaction, which could possibly interfere with the mechanical analysis of the system, was largely avoided by using the minimum volume necessary to effect a complete obstruction, which was accordingly tested in each animal. This volume

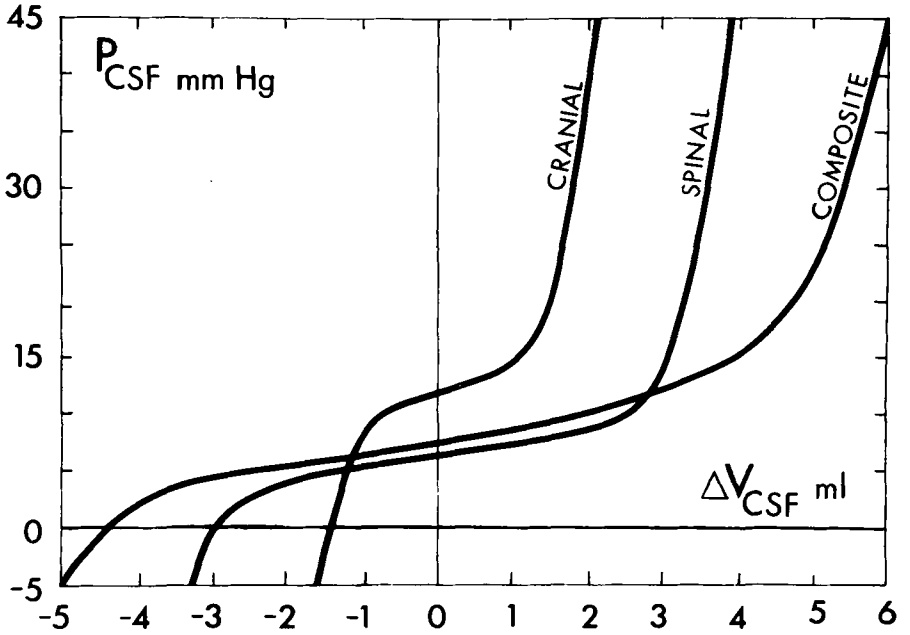

Figure 3. Interrelations between the cranial and spinal components and the composite pressure-volume curve in the region of the inflection point in a particular experiment.

was 1.4–2.0 ml. Even with this volume there was a slight increase in the blood pressure of about 10 mmHg. The blood pressure changes were immediately reversible on emptying the balloon. The procedure was repeatable with practically identical results.

The completeness of the block was ascertained by the absence of a change in the lumbar CSF pressure, during the rapid infusion of fluid into the cisterna magna, when recording the pressure-volume curve. A further verification was obtained by applying a steady intracranial fluid pressure of 60 mmHg for some minutes by means of an external reservoir, with no alteration in the lumbar pressure.

Figure 1 shows the pressure-volume curve before and after spinal block. The pressures were measured in the lateral ventricle, the cisterna magna and the lumbar space. After the induction of the spinal block there is a definite change in the shape of the curve. The range of volume change is diminished and the slope of the curve (the elastance dP/dV) is increased.

The pressure-volume curve, obtained after the induction of a block, is obviously that of the isolated intracranial fluid space. Figure 2 shows a set of diagrams from the six experiments in which the two curves

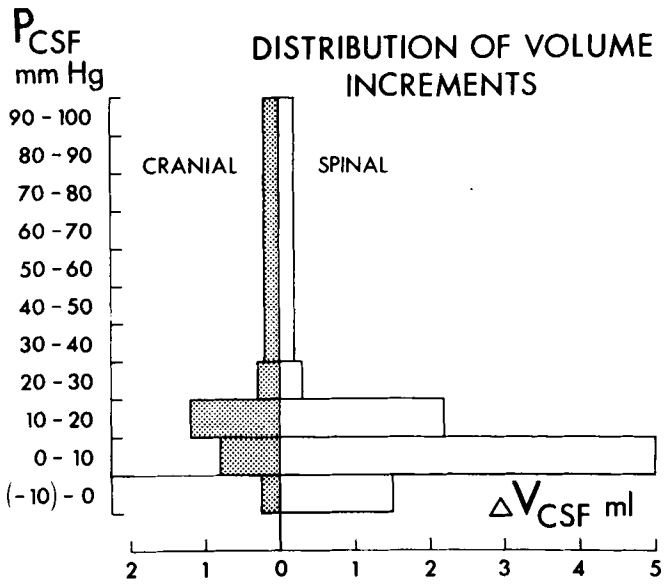

Figure 4. Diagram of the partitioning of a volume increment between the cranial and spinal compartments at different intervals of CSF pressures.

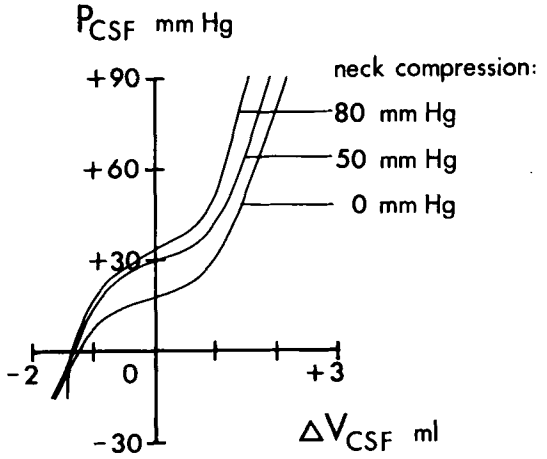

Figure 5. The pressure-volume curve of the intracranial CSF space recorded with neck compression at different pressures. The curve is elevated with increasing pressure.

are related to each other in the same system of ordinates, using the point of inflection as a convenient reference point. This is the point at which the curve crosses the tangent. By subtracting the intracranial curve from the control curve, the pressure-volume curve of the spinal

section of the system has been calculated. The resulting curve has a smooth and logical shape which speaks in favour of the appropriateness of the procedure. Figure 3 is a close-up view of the interrelations of the curves in the region of their inflection points.

When the curves are compared it is seen that the range of variation in volume is the smallest in the case of the cranial curve. The volume range measured from the breakpoint in the negative part of the curve to the intersection with the arterial blood pressure, to which the curve is extrapolated, has a mean value of 4.2 ml (range 3.9–4.5) for the cranial curve, 9.7 ml (range 7.5–11.8) for the spinal curve and 14 ml (range 11.5–17) for the control curve. 70 % of the total variation in volume takes place in the spinal compartment and 30 % in the cranial compartment. Figure 4 shows schematically the distribution of an increment in volume at different intervals of pressure. At lower pressures, in the normal or subnormal pressure range, most of the variation takes place in the spinal section whereas at higher pressures, above 20 mmHg, there is an approximately equal partitioning of the added fluid volume between the two subdivisions of the system.

The point of inflection of the cranial curve is systematically located at a higher pressure than that of the spinal curve. The elastance of the flat part of the curve (the low pressure elastance) is also larger in the cranial curve. The high pressure elastance of the steep part of the curve is, however, of the same order of magnitude in both curves, the elastance of the total system being obviously less. The mean value of the high pressure elastance of the cranial curve is 61 mmHg/ml (range 48–69) and of the spinal curve 59 mmHg/ml (range 45–75). The control curve of the total system had a mean elastance of 29 mmHg/ml (range 25–33).

The hypothesis that the pressure-volume curve of the intracranial fluid space is related to distension and compression, respectively, of the intracranial vascular system, was tested by recording the curve at various levels of increased venous pressure in the head, in the presence of a spinal block. According to this hypothesis an increase in the venous pressure would displace the pressure-volume curve upwards on the pressure axis. If, on the other hand, the pressure-volume curve was related to meningeal distension, changes in the venous pressure would hardly affect the curve. An increase in the venous pressure was produced by inflating a sphygmomanometer cuff around the neck, to different pressures. The result i shown in Figure 5 with a compression of 50 and 80 mmHg. The intracranial pressure-volume curve is displaced upwards with increasing pressure exerted on the tissues of the neck.

DISCUSSION

An immediate change in the volume of an element within the rigid skull cavity, produces a change in the CSF pressure which is dependent upon the distensibility of the CSF space, as expressed by the elastance, dP/dV (mmHg/ml), of the system (*Löfgren et al.* 1973). The mechanical basis of this function could be either a reciprocal change in the volume of the intradural vascular compartment, a change in the total volume of the system or a combination of these factors. This problem of total or reciprocal changes in volume within the system is a classical one in intracranial physiology, as discussed in the Monro-Kellie theory (*Weed* 1929). The present study is an attempt to clarify this problem by determining the CSF pressure-volume relationship of the intracranial and intraspinal compartments, separately.

The characteristic pressure-volume curve of the CSF system has been described previously (*Löfgren et al.* 1973). The principal result of the present study is the demonstration that the pressure-volume curve can be resolved into a cranial and a spinal component, i. e. these subdivisions of the system each have an individual pressure-volume curve. The shape of the spinal curve is in general agreement with that described by *Gilland* (1965), in patients with spinal block. The different shapes of the cranial and spinal curves suggest that the factors responsible for the elastic behaviour of these subdivisions are rather different. The considerably larger capacity for volume change of the spinal compartment in spite of its smaller total volume, supports the assumption that the pressure-volume relationships in this case are related primarily to changes in the total volume brought about by varying the degree of filling of the spinal dural sac. At the same time these volume variations of the dural sac should depend on reciprocal changes in the volume of the extradural venous system, since the total volume of the craniovertebral system, i. e. within the bony confines is presumably constant.

There are some features in the shape of the spinal pressure-volume curve which are of mechanical significance, and which indicate that the spinal dural sac does not have distensibility characteristics similar to rubber balloons or tubes. The low elastance over a vide range of volumes at and below the normal CSF pressure, suggests that the dural sac in this situation is flaccid, i.e. in a state of partial collapse with a trans-dural pressure of zero (equal intradural and extradural pressures). The spinal dural sac can vary freely in volume in this condition with almost no change in pressure. When the dural sac has reached a distended state, however, comparatively little fluid is accepted by further dis-

tension under pressure, an effect presumably related to a tightening of the collagen fibres, which are characterized by a low degree of extensibility (*Bull* 1957). In most of the spinal curves shown in Figure 2 there is a rather sharp break at the transition from a collapsed to a distended state. However, even a small radial distension may, for geometrical reasons, cause a significant change in the total volume of a tubular structure of a given length.

At the point of inflection, i. e. at the mid-point of the low elastance part of the curve, the elastic stresses are at a minimum. The CSF pressure at this point is closely related to the central venous pressure (*Ryder et al.* 1953 b, *Löfgren et al.* 1973). Decreasing the volume from this point causes, eventually, a rapidly increasing negative pressure, presumably due to distension of the extradural and intradural venous systems and a complete collapse of the dural sac, "folding" around the intradural contents.

The mechanical basis of the pressure-volume curve of the intracranial fluid space is more difficult to establish. There is a remote possibility that the curve actually depends upon changes in the total volume, by distension of meningeal structures not included in the present blocking procedure. However, the experiments involving elevated cephalic venous pressure, which caused a displacement to higher pressure levels of the intracranial pressure-volume curve, indicate that the curve is related chiefly to changes in the vascular volume.

The volume range of the cranial curve was ⅓ that of the spinal curve. The low elastance part of the curve was narrow, implying a rapid change from distension to compression. It is obvious that the low elastance part of the total system is completely dominated by the corresponding part of the spinal curve. The shape of the cranial curve does not support the concept, sometimes proposed, of a large high-compliant venous reservoir within the skull cavity, which could accomodate volume changes with insignificant effects on the pressure. On the contrary, the vascular bed seems explicitly resistant to volume changes, actually not permitting any free change in volume at all. When, however, the CSF pressure is varied extensively from its normal level to the level of the arterial blood pressure, 2–3 ml of blood is evidently entering or leaving the vascular bed, an amount which probably represents a considerable fraction of the total intracranial blood volume.

The point of inflection in the cranial pressure-volume curve is the point at which mechanical stresses are minimal in the cranial system. A volume decrement causes a distension and a volume increment a compression of the vascular bed, essentially of the venous section in

both cases presumably. The changes in CSF pressure may consequently be considered to be determined by the mechanical resistance of the vascular bed to forced reciprocal changes in its volume.

From a mechanical point of view the CSF system may be considered to consist of two parts arranged in parallel. Figure 3 illustrates the dynamic equilibrium existing between the two components. The pressure at the inflection point was lower in the spinal than in the cranial compartment. It seems, therefore, that at the normal level of the CSF pressure of about 10 mmHg, the conditions are such that the cranial system is in an essentially relaxed state and the spinal system in a slightly distended state.

Reconsidering the mechanism of the immediate spatial adjustment to an intracranial volume change, there are consequently two simultaneous and parallel events, i. e. a reciprocal change in the volume of the intracranial vascular bed and a displacement of CSF down into or up from the spinal dural sac. The partitioning of volume changes between the cranial and spinal compartments depends on the elastances of the two systems and will vary with the fluid pressure as shown in Figure 4. At higher pressures the distribution is about equal and at lower pressures most volume variations take place in the spinal compartment. The spinal compartment may be characterized as an expansion vessel coupled to the rigid skull cavity, moderating the intracranial pressure and volume changes.

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