Origin of cerebrospinal fluid pulsations

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Adolph, Robert J., Hiroo Fukusumi, and Noble O. Fowler. Origin of cerebrospinal fluid pulsations. Am. J. Physiol. 212(4): 840-846. 1967.—Experiments were performed in dogs to determine whether cerebrospinal fluid pulses were venous or arterial in origin. Since all cerebrospinal fluid pulsations synchronous with the heart beat must originate either in the right atrium or the left ventricle, interventions were designed to dissociate right atrial and left ventricular events. In the majority of normal dogs, the major pulsation in the cerebrospinal fluid was arterial in origin, but smaller venous pulsations were usually also present. Under conditions in which arterial pressure is decreased and/or venous pressure is increased, the venous pulsation may predominate. Interventions in which right atrial waves could be clearly demonstrated included the surgical production of complete heart block, partial pulmonary artery occlusion, and hypothermia. Cerebrospinal fluid pulsations of arterial origin were demonstrated during pulsus alternans, complete right heart bypass, mechanical stimulation of the left ventricle, and in some dogs with complete heart block. Recognition of the origin of cerebrospinal fluid pulsations is of importance in understanding normal pressure/flow relationships.

MATERIALS AND METHODS

Mongrel male dogs weighing between 16 and 25 kg were anesthetized with intravenous sodium pentobarbital in a dose of 25 mg/kg. A total of 32 dogs were studied. The trachea was intubated and the dog was mechanically resired. An 18-gauge thin-wall needle was introduced percutaneously into the cisterna magna and connected directly to a P23DB Statham pressure transducer. Two no. 8 Cournand catheters were passed, respectively, into the right atrium from a jugular vein and into the ascending aorta or carotid artery from a femoral artery. Blood pressures were measured with Statham P23DB pressure transducers. Blood pressure gauges in all experiments were leveled with the thoracic spine. The chest was then opened in the fourth right intercostal space, and the positions of the catheter tips were verified by palpation. Aortic pressure, right atrial pressure, spinal fluid pressure, and an electrocardiogram were simultaneously recorded on a Sanborn direct-writing Polyviso.

Complete atrioventricular block was accomplished in 7 of 10 dogs without opening the heart by means of needle abrasion of the conduction system through the free wall of the right atrium. This method avoids the surgical induction of tricuspid valve incompetence. Alternatively, complete heart block was accomplished surgically in three dogs through an incision in the right atrium during inflow occlusion. The bundle of His was lacerated or damaged by the tightening of a single suture through the base of the medial leaflet of the tricuspid valve.

Premature ventricular contractions were induced by mechanical stimulation of the left ventricle with the tip of a needle. Right heart bypass was accomplished in one dog by gravity drainage of blood from the superior and inferior vena cavae into a reservoir from which blood was returned to the right pulmonary artery by a roller pump at a rate adjusted to maintain the reservoir level constant.

Normally, small pulsations of the cerebrospinal fluid which occur with each heart beat can be seen when manometric studies are done during a lumbar puncture. It is of interest that when physicians are asked whether the pulsations are arterial or venous in origin, their responses are rather evenly divided. This is perhaps not so surprising since there are very few reported studies which were designed to answer this question, and even these are lacking (1, 2, 4, 6, 9, 10).

A series of experiments in dogs was designed to study the effects of various hemodynamic alterations on the cerebrospinal fluid pressure. In particular, we examined whether cerebrospinal fluid pulses were venous or arterial in origin. Since all cerebrospinal fluid pulsations synchronous with the heart beat must originate either in the right atrium or the left ventricle, interventions were designed to dissociate right atrial and left ventricular events.

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Spinal fluid pulsations were studied during immersion hypothermia in three dogs, during partial pulmonary artery occlusion in four dogs, and following the intravenous injection of norepinephrine in a dose of 1-3 \( \mu \)g/kg in five dogs.

Pulse transmission time in the saline-filled catheter-transducer system used in these experiments was measured by comparing it with a similar transducer connected to an 18G needle. An air-filled balloon, connected by a short Y adapter to both systems, was punctured applying a square-wave input. The pressure change in both systems was recorded simultaneously at a paper speed of 100 mm/sec.

RESULTS

An interpretation of the origin of cerebrospinal fluid pulses from analysis of simultaneously recorded right atrial and central aortic pressures requires a knowledge of pulse transmission times in the saline-filled catheter-transducer systems used in these experiments. This averaged 0.02 sec for both venous and arterial cathers. Allowing for this small delay, the transmission time between the central aorta and the cisterna magna was analyzed in those tracings in which cerebrospinal fluid pulsations were clearly arterial in origin. This varied between 0.01 and 0.02 sec. That is, the recorded cerebrospinal fluid pulse occurred simultaneously with the recorded arterial pulse or preceded it by 0.01 sec. The transmission time between the right atrium and the cisterna magna, however, was somewhat longer owing to the more compliant venous vasculature. It varied between 0.02 sec and 0.06 sec. That is, a cerebrospinal fluid pulse which was clearly venous in origin occurred simultaneously with the right atrial pulse (a wave in Fig. 5B) or followed it (a wave in Fig. 2A) by 0.04 sec or less.

In Fig. 1A it can be seen that in this mechanically respired dog, phasic variations in mean pressure synchronous with respiration occur in the right atrium, central aorta, and spinal fluid. The respiratory variations in spinal fluid pressure could be arterial or venous in origin.

Figure 1, B, C and D demonstrates examples of the spectrum of results we have observed in 32 normal dogs. Recordings in the majority of dogs resemble that shown in Fig. 1B. The major positive cerebrospinal fluid pulsation begins synchronously with the central aortic pulse and often resembles a damped aortic pulsation. Smaller venous waves are often seen. In Fig. 1D no definite deflection corresponding to the right atrial a and v waves can be identified. The spinal fluid pulses shown in Fig. 1C are mainly venous in origin since they closely
FIG. 2. In most dogs with complete atrioventricular block, right atrial a waves or cannon waves are seen in the spinal fluid pressure recording. Waves of aortic origin cannot be definitely distinguished (A). In some dogs with complete heart block, only pulses of arterial origin can be seen in the cerebrospinal fluid (B).

FIG. 3. A illustrates the transmission of arterial pulsus alternans to the cerebrospinal fluid in a dog with a failing heart. Large c-v waves of right heart failure are not transmitted. The effect of mechanically induced premature contractions are shown in B. Cerebrospinal fluid pulses are entirely arterial in origin.

resemble the wave forms seen in the right atrial recording. In Fig. 1D large spinal fluid pulses are seen which correspond in time to venous a waves. The transmitted a wave is followed by a pulse of arterial origin. No definite venous pulse at the time of isometric ventricular systole, e.g., a e wave, is seen in the right atrial pressure tracing.

The significant contribution of right atrial contraction to cerebrospinal fluid pulsations in 7 of 10 dogs with complete heart block is exemplified in Fig. 2A. Pulses ("a" wave equivalents) which correspond to the P waves of the electrocardiogram are clearly seen in the spinal fluid and right atrial pressure recordings. Large cannon "a" waves which occur when the right atrium contracts while the tricuspid valve is closed are also transmitted to the CSF and tend to obscure the contribution of transmitted arterial pulsations. In another three dogs with complete heart block, however, only pulses of arterial origin could be seen in the spinal fluid pressure recording (Fig. 2B). That these pulsations do not represent transmitted cannon a waves can be seen in this figure. The idioventricular rhythm was interrupted by mechanically induced left ventricular premature contractions. The premature beat initiated an ineffective ventricular contraction, since it was not followed by either an aortic or a spinal fluid pulse. The premature contraction was followed by a venous cannon wave, however, indicating closure of the tricuspid valve. The well-developed atrial cannon wave was not seen in the spinal fluid recording. It can also be seen that the cerebrospinal fluid pulses preceded the right atrial cannon waves.

The arterial origin of the spinal fluid pulsations is clearly seen in Figs. 3, A and B. In Fig. 3A the rhythm was ventricular tachycardia. Tall peaked c-v waves of tricuspid insufficiency were seen in the right atrium but not in the spinal fluid. A pulsus alternans observed in the central aorta, however, was mirrored in the spinal fluid. In another dog, the sinus mechanism was interrupted by mechanical stimulation of the left ventricle (Fig. 3B). The spinal fluid wave forms closely follow the arterial pattern. During the long compensatory pause
CEREBROSPINAL FLUID PULSATIONS following the premature contractions, the right atrial pulsations continue but cannot be seen in the spinal fluid tracing.

Right heart bypass was also utilized to dissociate right atrial and left ventricular events in a single dog. As shown in Fig. 4, during bypass all pulsations were eliminated from the right atrium which was kept virtually devoid of blood while left heart function was preserved. Pulsations in the spinal fluid synchronous with those in the aorta were clearly seen.

The venous contribution to cerebrospinal fluid pulsations can be made to predominate under conditions of a lowered systemic arterial pressure and an elevated venous pressure. In Fig. 5A the spinal fluid pulsations would seem to be mainly arterial in origin during the control state, although the contribution of venous $a$ waves can also be seen. However, during partial occlusion of the main pulmonary artery in four dogs the aortic pressure was drastically reduced, and the venous $a$ waves were greatly increased in magnitude. Under these conditions, the spinal fluid pulsations appeared to be wholly venous in origin. There could, however, have been a small arterial contribution which was lost in the large and prolonged $a$ wave.

Similar hemodynamic changes were induced by hypothermia in three dogs. At 30°C, the major spinal fluid pulsations seemed to be arterial in origin. At 24°C, however, the arterial pressure was significantly reduced and the venous pressure was elevated. Large right atrial $a$ waves then predominated in the cerebrospinal fluid pressure recording, although arterial pulsations were still visible.

The effect of the rapid intravenous injection of norepinephrine in five dogs in a dose of 3 $\mu$g/kg is shown in Fig. 7. The cerebrospinal fluid mean pressure rose coincident with the increase in arterial pressure and before any change in right atrial pressure was detected. About 10 sec after the initial pressure rise, venous

![Fig. 4](image)

**Fig. 4.** Spinal fluid pulsations before right heart bypass appear to be arterial in origin (A). During right heart bypass, right atrial pulsations are eliminated and spinal fluid pulsations are now clearly arterial in origin (B).

![Fig. 5](image)

**Fig. 5.** Spinal fluid pulsations before main pulmonary artery occlusion appear to be both arterial and venous in origin (A). During partial occlusion of the main pulmonary artery, the mean right atrial pressure and the amplitudes of $a$ and $z$ waves are increased (B). The cerebrospinal fluid pulsations appear to be entirely venous in origin.
of the arterial pulsation to the cerebrospinal fluid, and that although the major spinal fluid pulsation is arterial in origin, smaller vibrations coincident with the right atrial a waves can sometimes be seen. The arterial nature of the pulsation was suggested earlier by Antoni (1, 2) and Goldenhson and associates (9). Excessive damping and poor frequency responses in early manometer systems made interpretation of findings difficult. Dunbar and co-workers (6) recorded the pulse waves in the CSF in the ventricles, cisterna magna, and lumbar subarachnoid space and found them almost identical to each other and to the arterial pulse wave. No attempts were made to dissociate right atrial and left ventricular events, however. They disagreed with Bering's conclusions regarding the origin of the pulsations, since their studies suggested that the arteries supplying the spinal cord, rather than the choroid plexus, were the site of origin of the transmitted pulse.

Recently, Hamit, Beall, and DeBakey (10) restudied the problem and concluded that although the arterial blood pressure is important in maintaining the static pressure of the cerebrospinal fluid, the major pulsations are venous and not arterial in origin.

We would agree with Bering that in the majority of normal dogs, the major pulsation in the cerebrospinal fluid is arterial in origin. In 20 of 32 dogs, smaller waves coincident with right atrial a or v waves could also be seen. In an occasional dog, the major pulsation seems to be venous in origin. The findings of Hamit et al. are inconclusive for several reasons. Cerebrospinal fluid pressure was measured through a 100 cm length of PE tubing which can be demonstrated to overdamp seriously and to delay pulsatile wave forms. Arterial pulsations which have a rapid rise time would be selectively damped relative to the slower venous pulsations, although all pulsations could be delayed and distorted. This is clearly demonstrated in his own base-line tracings. The critical evaluation of the origin of waves of variable delay was not feasible. The absence of calibrations of venous, arterial, and cerebrospinal fluid pressures made
the interpretation of interventions difficult. These authors do show the contribution of transmitted venous pulsations under severely abnormal hemodynamic conditions, e.g., heart block and ventricular fibrillation, but always when the arterial pressure was virtually eliminated and the heart was failing as evidenced by large c-v waves in the right atrium.

Our findings would indicate that although the major cerebrospinal fluid pulsations are arterial in most normal dogs, smaller venous pulses are usually present as well. Under experimental conditions in which arterial pressure is decreased and/or venous pressure is elevated, the venous pulsation may predominate. Arterial and venous events were dissociated in a variety of ways to evaluate the origin of cerebrospinal fluid pulses. Interventions in which cerebrospinal fluid waves of right atrial origin could be clearly demonstrated included the surgical production of complete heart block, partial pulmonary artery occlusion, and hypothermia. The mechanism of production of predominant venous pulsations in the cerebrospinal fluid in the latter two conditions is related to the decrease in systemic arterial pressure associated with a rise in central venous pressure. Pulsations clearly of arterial origin were seen during pulsus alternans, complete right heart bypass, mechanical stimulation of the left ventricle, and in some dogs with complete heart block.

The determinants of the mean cerebrospinal fluid pressure are complex and are poorly understood. Nevertheless, because cerebrospinal fluid, veins, arteries, capillaries, interstitial fluid, and brain and spinal cord themselves are all enclosed within a relatively rigid container composed of cranium, spinal column, and dura, cerebrospinal fluid pressure changes can be analyzed in terms of plethysmographic theory (5, 7). Any procedure or intervention which tends to acutely increase the volume within the cavity will increase the pressure. It has been shown (14) that the cerebrospinal fluid pressure and venous pressure within the cerebrospinal cavity must be precisely equal at every level regardless of body position or orientation, and this equality of pressure has been found in animals even during radial acceleration in a centrifuge. Hence, changes in the venous pressure are accompanied by corresponding changes in the cerebrospinal fluid pressure (12). An elevated mean cerebrospinal fluid pressure is seen during acute occlusion of the jugular veins in the Queckenstedt test (4) and in patients with elevated right atrial pressure due to severe congestive heart failure (8, 11). The mean intracranial pressure, on the other hand, is largely independent of the arterial blood pressure (3, 12, 13) but may be affected by acute changes (5, 7, 15). The mean cerebrospinal fluid pressure has been shown to fall following simultaneous occlusion of both common carotid and both vertebral arteries (10). During partial pulmonary artery occlusion (Fig. 5) and hypothermia (Fig. 6) the CSF pressure fell when the arterial pressure fell markedly and the right atrial pressure rose somewhat. The cerebrospinal fluid pressure increased during acute catecholamine infusion when the arterial pressure rose markedly and the venous pressure also rose moderately (Fig. 7). Analysis of arterial and venous pressure changes alone, however, has led to confusion in the literature regarding the determinants of pressure changes in the cerebrospinal fluid. Hamit and co-workers (10) concluded on the basis of acute arterial pressure changes that the CSF pressure was largely determined by the level of the systemic arterial pressure. Dunbar and his associates (6) recently refuted this concept and stated that the venous pressure alone determined the CSF pressure.

To conclusively resolve this problem, several critical measurements must be made simultaneously during an acute intervention: cerebrospinal arterial inflow; cerebrospinal venous outflow; cerebrospinal fluid volume; and venous, arterial, and CSF pressures at the same level within the cerebrospinal cavity. Techniques capable of measuring transient blood flow changes would be necessary. Although these measurements were beyond the scope of this and other reported studies, it seems reasonable to assume that, in the absence of acute loss or gain of cerebrospinal fluid, an acute change in cerebrospinal fluid pressure is primarily a function of the intracranial and intraspinal blood volume, which in turn is primarily determined by the net difference between arterial inflow and venous outflow. An intervention which increases the resistance to venous outflow, e.g., the Queckenstedt test, increases cerebrospinal fluid pressure. A rise in arterial pressure is usually but not necessarily an index of increased arterial inflow. Ryder et al. (15) attempted to dissociate changes in arterial blood pressure from changes in cerebral blood flow and concluded on the basis of indirect evidence that the CSF pressure was a function of blood flow. Hence, partial pulmonary artery occlusion and hypothermia probably decrease cerebral blood inflow and the CSF pressure falls despite some increase in outflow resistance. The early rise in CSF pressure following catecholamine administration could result from both increased inflow (increased arterial pressure) and decreased outflow (increased venous pressure) or either change alone.

Damped cerebrospinal fluid pulsations coincident with the heart beat may be caused by either venous or arterial pulsations without invoking flow considerations. Even so, small volume changes with each pulse must result from movement of distensible vessel walls within a semirigid cerebrospinal cavity.

REFERENCES