ANGIOGRAPHIC STUDIES OF EXPERIMENTAL INTRACRANIAL HYPERTENSION*

WILLIAM E. HUNT, M.D., J. N. MEAGHER, M.D.,
A. FRIEMANIS, M.D., AND C. W. ROSEL, M.D.

Department of Surgery, Division of Neurological Surgery, and Department of Radiology,
Ohio State University Health Center, Columbus, Ohio

(Received for publication May 31, 1962)

The relationship between intracranial hypertension, cerebrovascular dynamics and medullary failure still is not completely understood. Astley Cooper,\(^3\) in 1824, showed bradycardia and a diminished state of consciousness by pressure of the finger on the surface of the cerebrum. In 1895, Bayliss et al.\(^1\) described the phenomenon that bears the name of Bayliss. They observed that pial vessels constricted when blood pressure was raised and dilated when blood pressure fell. They expressed doubt about the existence of any central or peripheral vasomotor control over cerebral blood vessels. They stated further that in all physiological conditions the volume of the brain was practically "invariable" and that "a rise of arterial pressure accelerates the flow of blood through the brain, and a fall slackens it". However, they concluded that "the cerebral circulation passively follows the changes in the general arterial and venous pressures," and that there was "no compensatory mechanism by which the intra-cranial pressure is kept constant." They added that "brain matter does not suffer from pressure alone, but from pressure producing anaemia."

In 1901, Cushing\(^1\) reported experimental observations concerning increased intracranial pressure, stating that it was not the degree of compression but the rise in intracranial pressure that produced symptoms of hypertension and bradycardia attributed to "capillary anemia of the medulla". He stated that intracranial pressure had to reach arterial pressure before symptoms were produced. In his diagrams he implied, but never stated, that it was the diastolic pressure that was critical, although he has been misquoted as having said "systolic blood pressure". He stated further that apnea did not occur in his experiments but, with a compromise of the bulb, the one sign that was called forth regularly and was to be taken as a serious alteration in the local circulation was a persistent rise in the blood pressure. This "may or may not be associated with a pronounced vagus pulse, with rhythmic alterations in blood-pressure and with a retardation or periodicities of the respirations approaching a Cheyne-Stokes type."

Forbes et al.,\(^13,14\) in 1937, stated that the immediate cause of arterial dilatation in the pia appeared to be a sudden slowing of the blood flow. They later concluded, after stimulating the geniculate ganglion, that higher mammals possessed a true vasodilator innervation of the arteries supplying the parietal cerebral cortex.

In the late 1930s, Fog\(^16-12\) observed the Bayliss effect and thought that the dilatation and constriction were caused by changes in "endovascular pressure". He stated that the mechanisms by which the tonus of the vessels was regulated were not yet established.

By 1942, the term "vasospasm" had gained prominence. Echlin\(^7\) produced vaso-spasm in pial arteries of all sizes by stretching them. He observed no propagation of the spasm, and therefore concluded that it was a stretch reflex and not a neurovascular mechanism.


This work was supported by a grant from the Central Ohio Heart Association.
The last decade has seen major changes in our concepts of dynamic cerebrovascular pathology and physiology. Denny-Brown\(^6\) commented in 1951 that the previous views regarding the signs of increased intracranial pressure were being modified by theories of herniation at the tentorial opening and other distortions. Evans \textit{et al.},\(^9\) in the same year, were able to produce experimentally in man higher levels of intracranial hypertension than had been seen clinically, without producing any significant changes in vital signs or level of responsiveness. Rodbard and Saiki,\(^\text{18}\) in 1952, using inhalation of nitrogen and tracheal occlusion, questioned the theory of anemia as the stimulating factor in the medulla.

Meyer and Denny-Brown,\(^\text{15}\) in 1957, introduced a polarographic technique to study cerebral anoxia. Recording the availability of cortical oxygen, they showed that cerebral anoxia results first in the slowing of the electroencephalogram. They concluded that the Bayliss effect appeared to cause the rapid compensatory changes. Thereafter, slower circulatory adjustments occurred that appeared to be mediated by metabolic factors.

Pool,\(^\text{17}\) in 1958, summarized the current concepts on the behavior of cerebral vessels. By his study on the large vessels of the base of the brain, he was able to show prompt vasoconstriction with mechanical, thermal and chemical stimuli. He stated further that this vasoconstriction may lead to permanent neurological deficits and even death. This concept is supported by the observations of Birse and Tom\(^2\) in autopsy material from patients who died of subarachnoid hemorrhage. Dott\(^6\) has postulated that vasoconstriction resulting from acute stretching may be a cause of the syndrome of cerebral concussion. He stated that the concept of “cerebral vasospasm” was valid as it could be shown clinically, angiographically, experimentally and surgically. He felt that vasoconstriction was mediated principally by the vascular musculature but that intrinsic activity of the neurogenic reflex or extrinsic innervation of the cerebral arteries also may play a part.

Thompson and Malina,\(^\text{21}\) in 1959, postulated that since the changes observed by Cushing often are seen clinically before the intracranial pressure reaches systolic blood pressure, another mechanism must be operative. They concluded that when cardio-respiratory symptoms developed in the presence of increased intracranial pressure, an acute dynamic axial distortion of the brain stem was taking place.

The present study explores by means of cerebral angiography the intracranial circulation during intracranial hypertension, systemic hypertension, and systemic hypotension.

**MATERIALS AND METHODS**

Adult male and female mongrel dogs weighing 10 to 15 kg. were anesthetized with intravenous pentobarbital sodium (30 mg./kg.). A #12 Foley catheter with a 30 cc. bag was inserted into the cranial epidural space through a small opening. Blood pressure was recorded from the right femoral artery. The left femoral artery was cannulated with a #10 NIH catheter which was advanced to the region of the aortic valve under fluoroscopic control. A blood-pressure cuff was wrapped about the animal's chest in order to record respirations. A polyethylene catheter (.090" internal diameter) was passed into the cisterna magna through a 16-gauge needle to record pressure and to inject fluid. Electrocardiographic leads were placed, and the animal was positioned on the roentgen-ray unit (Schönander film changer, type AOT).

An "Electronics for Medicine" (Model DR8) research recorder was used to monitor and record the experimental data. The electrocardiogram and electroencephalogram were connected to the recorder directly. The blood pressure, respirations, and cisternal pressure were recorded through Statham transducers.\(^*\) The base lines were established by opening the transducers to atmospheric pressure, and all pressures were recorded at the same gain. The remaining channels in the recorder were used to register injection signals, film signals, and pressure lines.

The #10 NIH catheter was connected to a high-pressure injection syringe (original Gidlund Cat. Nr. DTF 685), set to deliver 20 ml. of 75 per cent sodium diatrizoate (Hypaque) at 37°C, under a pressure of 10 kg./cm.\(^2\). The injector unit was set to trigger the angiographic unit, which exposed 16 films at a rate of 4 per sec.

A control angiographic series was done on each animal. Angiography was repeated under various conditions of intracranial pressure and systemic blood pressure.

Postmortem injection of contrast agents

\(^*\) Statham Instruments, Inc., Hato Rey, Puerto Rico.
ANGIOGRAPHY OF INTRACRANIAL HYPERTENSION

(barium-sulfate suspension or Lipiodol) into the common carotid artery or directly into the cerebral arteries was done on 8 animals. Roentgenograms were made of the intact head, of the head without the brain, and of the brain itself. A photograph of the brain was taken for comparison to the roentgenograms.

RESULTS

Anatomy. The anatomy of the dog’s cerebral circulation as demonstrated by the control angiograms was correlated with the postmortem roentgenograms of the barium-injected specimens described above. Fig. 1 is a photograph of such a specimen. Note the extensive contribution of the occipital artery via the anterior spinal and vertebral arteries, the internal carotid arteries and the internal ophthalmic arteries. We shall refer to these vessels as the extracranial portion of the cerebrospinal circulation. As will be shown, the cerebrospinal circulation, both intra- and extracranially, may differ in its physiological responses from the somatic or extracranial portion of the cranial arterial tree. It may be that the cerebrospinal arterial tree takes on its special character at its origin in the neck rather than at its point of entrance into the subarachnoid space.

It seems, therefore, that 3 pairs of arteries feed the intracranial circulation: internal carotid, vertebral, and ophthalmic. These vessels are of comparable size. Well defined Anastomoses with the middle meningeal and occipital arteries also are present.

Control Observations. Control observations were made at the beginning of each experiment before intracranial hypertension was produced by any means. In several animals, control observations were repeated after the animal had been stressed and allowed to recover, in order to determine if the dog’s circulation and/or medullary centers had been damaged. In all, 26 control observations were made in 21 animals. The injection time of the 20 ml. of Hypaque lasted from 0.54 to 0.62 sec. and was followed by a brief rise in blood pressure, especially in the diastolic component (Fig. 2). The succeeding 4 to 6 beats of the pulse were slowed slightly. Following this bradycardia, a mild hypotension was observed which lasted from 10 to 20 sec. Only 1 animal showed a reaction to the control injection which was severe enough to require termination of the experiment.

Intracranial Hypertension Produced by Injection of Saline Solution into Cisterna Magna. The elevation of intracranial pressure by the injection of 0.9 per cent solution of sodium chloride into the cisterna magna provides the

Fig. 1. Autopsy specimen of barium-injected circulation through left common carotid artery (brain left in situ). Note overlap of intracavernous-intrasosseous portion of internal carotid artery and posterior communicating artery. The anterior spinal artery filled via the occipital artery.
the simplest experimental situation for studying the effects of increased intracranial pressure, since factors such as shift of the brain stem and traction on intracranial vessels do not have to be considered.

Twenty-one observations were made in 11 dogs, whose blood pressure initially ranged from 140/100 to 230/140. The volume required to produce symptoms depended upon the rate of injection, since cerebrospinal-fluid pressures returned rapidly to normal, presumably by absorption of the excess fluid (Fig. 3). When fluid was administered fast enough to exceed the rate of absorption, cerebrospinal-fluid pressure rose and approached diastolic blood pressure before any significant effect was noted. If the rate of injection rapidly reached and passed diastolic blood pressure, there was little rise in blood pressure prior to the abrupt appearance of marked bradycardia, hypotension, and apnea. An angiogram made at this juncture showed no filling of the intracranial circulation and often contrast material did not even enter the internal carotid, internal ophthalmic or vertebral arteries.

If the cerebrospinal-fluid pressure approached diastolic arterial pressure more slowly, the blood pressure would begin to rise when cerebrospinal-fluid pressure came within 10 to 15 mm./Hg of diastolic arterial pressure. By slowly adding fluid so that cerebrospinal-fluid pressure continued to rise gradually, a marked hypertension with minimal or no bradycardia could be evoked.

Systolic blood pressure and the pulse pressure often were increased by over one-half of their initial values. When cerebrospinal-fluid pressure was forced above the diastolic pressure, there was an immediate drop in blood pressure accompanied by apnea and failure of the cerebrospinal arteries to fill (Fig. 4). The pulsations of cerebrospinal-fluid pressure at this point were increased and during the period of falling blood pressure, their diastolic phases tended to coincide closely with diastolic arterial pressure, although systolic pressures were quite different.

If the arterial diastolic and the cerebrospinal-fluid diastolic pressures remained the same, apnea persisted, and blood pressure continued to fall, often with bradycardia. However, if the cerebrospinal-fluid pressure fell more rapidly than arterial diastolic pressure, the cerebral circulation would fill again, arterial pressure would rise, and respirations would resume spontaneously (Fig. 5).

**Effects of Inflation of Intracranial Balloon.** In 6 dogs, 13 observations were made of the effect of a "mass" lesion created by the inflation of a balloon over one hemisphere. This produced a complex situation in which shift and distortion of nervous and vascular tissues occurred, in addition to generalized pressure.

Up to a point, cerebrospinal-fluid pressure returned to normal fairly rapidly after the rise caused by injection of small increments of saline solution into the balloon. Presumably, this resulted from the absorption of cerebrospinal fluid. After about 3 ml. have

---

**Fig. 2.** Recording during control injection of 20 ml. of Hypaque. The vertical lines are at intervals of 0.2 sec. Film S. indicates angiographic exposures. The fourth line records respirations. Dye S./160 shows the time and duration of injection of contrast medium in the line used to indicate 100 mm. of Hg pressure. CSF indicates cisternal pressure.

**Fig. 3.** Recordings taken just after cessation of injection of physiological saline solution into the cisterna magna. The cerebrospinal-fluid pressure falls rapidly. Time is indicated in min. and sec. Injection was stopped at 1 min., 50 sec.
been added, however, complete compensation was not seen within the relatively short periods of observation in these experiments. As the size of the balloon was increased, difficulty often was encountered in keeping the cisternal catheter open, as if the basal cistern were being obliterated or the brain stem were being shifted.

Changes in vital signs were less predictable than in experiments in which pressure was produced by intracisternal saline solution. Hypertension was less noticeable, and apnea would occur without marked bradycardia. On the other hand, marked bradycardia with gross exaggeration of the normal sinus arrhythmia was seen in some instances.

As in the experiments with saline alone, however, final decompensation occurred when diastolic arterial and diastolic cerebrospinal-fluid pressures were equal and cerebral circulation failed to fill. Falling blood pressure with apnea marked this event (Fig. 6).

This shows a shallow gasp in the time of delivery of the bolus of dye in an animal previously apneic. This gasp was followed by a few beats of high arterial pressure during which there was very weak, delayed filling of the anterior spinal, basilar and ophthalmic arteries. This animal then received artificial respiration, but the balloon was not deflated (Fig. 7). A marked hypertension developed and spontaneous respirations were resumed.

Fig. 4. (Left) Angiographic filling of cerebral circulation under control conditions. (Right) Exposure taken when cisternal pressure was forced above diastolic blood pressure. Note the intracranial circulation does not fill. Bradycardia was not present.

Fig. 5. Recordings following cessation of cisternal injection of saline solution. Note that as the diastolic arterial pressure and cisternal pressure separate, respirations resume.
Changes in Calibre of Cranial Vessels and Circulation Time. Changes in calibre of arteries were studied by a double blind method in which vessels were measured by calipers and a micrometer scale. Errors in measurements were of the order of magnitude of 10 to 20 per cent depending on the size of the vessels. Significant changes were noted in 14 of the 21 animals under various circumstances. In the common carotid artery and its extracranial branches, changes were much less marked than in the cerebrospinal arterial circulation. The cerebrospinal arteries showed rather marked variations in calibre. The diameter of the contrast column in these vessels might be reduced as much as one-third or increased as much as three-fourths of its initial size (Fig. 8). In general, changes in calibre were related to blood pressure, regardless of how the changes in blood pressure had been induced. The cerebrospinal arterial tree dilated when blood pressure fell and constricted when it rose.
Certain observations were difficult to explain and require further study. For example, in 2 instances cerebral vessels dilated in the presence of systemic hypertension. In both instances, bradycardia was present. With marked hypotension or hypertension, circulation always was slower than in the control observations. The time required for the dye to pass from the origin of the common carotid artery to the intracavernous portion of the internal carotid artery might be prolonged up to 0.75 sec. over the control series. The times of internal and external carotid circulation did not change to the same degree during the same experiment.

DISCUSSION

It would seem that when the medullary circulation is threatened by a rising intracranial pressure a pressor response is induced with or without producing some bradycardia. When this systemic hypertension no longer is able to maintain cerebral flow an abrupt vasodepressor effect occurs. The blood pressure falls, presumably because of a massive sympathetic inhibition. Coincidentally, there may be produced a marked bradycardia caused by vagal stimulation. The appearance of these events is evidence of virtually complete cerebral ischemia, and would indicate that the diastolic component of the cerebrospinal-fluid pressure equals or exceeds that of the diastolic blood pressure.

It is known that the outflow of a given vascular bed occurs during diastole. It would seem that when the diastolic blood pressure and the cerebrospinal-fluid pressure are identical, this relationship would result in an obstruction to the tract of outflow primarily and to the tract of inflow secondarily. As pointed out by Evans, spinal-fluid pressure cannot be maintained without cerebral blood flow. The total cerebral ischemia demonstrated here may explain the advanced necrosis in the central nervous system found at autopsy in many patients who have been maintained in a respirator for a number of days following respiratory arrest secondary to lesions producing increased intracranial pressure. The “respirator brain” is not seen in those instances in which respiratory arrest was caused by a focal lesion or lesions without increased intracranial pressure.

Our findings agree with those of others regarding the effect of a “mass” lesion in producing malfunction of the brain stem without necessarily raising intracranial pressure per se to the critical levels described above.

The Bayliss effect has been shown to act in such a manner as to provide an important homeostatic control for cerebral circulation. Obviously, the hypertension and increased cardiac output that accompany somatic activity may not be needed by the central nervous system, whose demands for oxygen may be the same under conditions of physical inactivity as during exercise. The comment of Bayliss that the velocity of the cerebral circulation changed with the blood pressure may well be accurate, but velocity of flow cannot be equated with minute-volume flow, especially in the presence of major changes in the calibre of vessels and, therefore, in cerebral vascular resistance.

Contrary to the opinions of Roy and Sherrington and Bayliss, the cerebral circulation does not passively follow the systemic circulation but remains remarkably constant over a wide range of systemic blood pressures. Under physiological conditions, variations in cerebral blood flow of significant degree probably are brought about chiefly, if not exclusively, by changes within the central nervous system itself. The work of Meyer et al. lends support to this hypothesis.

We have noted 2 instances in which the Bayliss effect was reversed. In both instances, vascular dilatation occurred in the presence of systemic hypertension; however, the blood pressure was falling and bradycardia was present. One might infer from this that a reflex mechanism exists capable of overriding the intrinsic mechanism of the Bayliss effect in the cerebrospinal arterial tree. Further work is under way to study this response.

SUMMARY

1. An angiographic technique has been
used to study the cerebral circulation in dogs under conditions of intracranial hypertension produced by two different methods, and by modifying the systemic blood pressure by drugs and by aortic occlusion.

2. The effect on vital signs of simple increase in intracranial pressure is not identical with the effect of "mass" lesion.

3. Apnea, bradycardia and hypotension reflect total cerebral ischemia when cerebrospinal-fluid pressures reach diastolic arterial levels.

4. The Bayliss effect is pronounced in the cerebrospinal arterial circulation, and negligible in the somatic circulation in the intact animal.

5. There is some evidence that a reflex mechanism may override the Bayliss effect in the vessels of the central nervous system.

6. The significance of these findings in clarifying some of the homeostatic factors controlling the cerebrospinal circulation is discussed.

REFERENCES


DISCUSSION

Dr. Guy L. Odom: The authors of both papers are to be congratulated on an excellent series of physiological experiments which are related directly to important clinical problems. Bradford's work demonstrates the pathophysiologic cause of the complication of persistent increased intracranial pressure that occasionally occurs following extensive subarachnoid hemorrhage of a ruptured aneurysm or other causes. It is unfortunate that they did not pursue the study further and attempt to determine where the "block" in the absorption occurred. That is, whether the obstruction occurs in the arachnoid villi or the spaces along the cranial or spinal nerves or those of the blood vessels. It is thought that, in all probability, the high-pressure injections, 135–400 mm. of mercury (1800–5440 mm. of water), played a role in the fact that additional anesthesia was not necessary in the prolonged experiments. I do not think that the arteriosubarachnoid shunt can be compared to the rupture of a subarachnoid artery and that the results in this experiment are exactly what one would have anticipated if blood were pumped in continuously by hand, since there could be no mechanism brought into play to attempt to stop the bleeding as might possibly occur from a ruptured aneurysm or intracranial vessel.

Dr. Hunt and associates have shown in an excellent
manner the physiological changes that occur in the intracranial vascular system with increased intracranial pressure. Their work confirms that of Bradford and others in that it is difficult to maintain increased intracranial pressure by injecting fluid into the subarachnoid space in animals because of the rapid rate of absorption and that, in order to maintain increased pressure, the rate of injection must be rapid or the injection must be done with a high pressure. They have demonstrated in animals what we see occasionally in man when arteriography fails to fill the intracranial vessels in cases of marked increased intracranial pressure.

It is unfortunate that Eben Alexander, or one of his associates, is not discussing this paper because they presented a very similar study at the recent meeting of the Society of Neurological Surgeons. They reported several cases of nonfilling of the intracranial vessels on carotid arteriography when there was marked increased intracranial pressure. They carried out similar experiments to those of Dr. Hunt in dogs and monkeys and found that failure to fill the intracranial vessels by arteriography occurred in dogs and monkeys when the intracranial pressure approached the systolic blood pressure.

They warned against the possibility of interpreting this nonfilling as thrombosis of the carotid artery and have shown that in cases in which nonfilling occurred, if the intracranial pressure was lowered, the arterial tree was visualized clearly.

I would like to ask Dr. Hunt whether they took the opportunity to study the retinal vessels during the period of increased pressure when the cerebral circulation failed to fill. Wolff and Davies in similar experiments of pressure injections of 100–200 mm. of mercury noted that the retinal vessels disappeared, the fundus became pale and the optic discs became white. Respiration stopped and cardiac action became weak. If the pressure was dropped, the retinal arteries reappeared and the color of the fundus became normal. I think that there is one point that should be clarified so that there will be no misunderstanding. Dr. Hunt stated that slowly rising cerebrospinal-fluid pressure produced hypertension. I feel certain that this slowly rising pressure that he refers to in these experiments occurred in a matter of minutes and is the same as the acute rise in intracranial pressure that we refer to from a clinical standpoint.

I would again like to congratulate both groups of authors on their excellent work.

DR. JOSEPH P. EVANS: It is an interesting fact that even after 50 years of active and increasingly widespread surgical attack upon the central nervous system, there is so little real understanding of the cerebrospinal fluid: its formation and its mode of absorption. The efforts of Drs. Bradford and Sharkey are directed toward a better understanding of the physiology of the subarachnoid space, as studied in the dog. In these efforts they have utilized investigative techniques not available to Harvey Cushing and his contemporaries.

Their experiments with saline, blood and India ink essentially are confirmatory of earlier work, and their results with newer agents, such as colloidal gold, indicate that these substances behave in much the same fashion.

The arteriosubarachnoid shunts have demonstrated clearly the obstructive effect of blood as opposed to saline, though even saline when introduced above a critical rate can be lethal in its effects of pressure.

Their suggestion that the potentials of absorption of the subarachnoid space in man might be tested in advance is an interesting one, as yet, as they point out, unproven.

Perhaps it would be well to reiterate here, lest anyone in the room be unaware of its dangers, that methylene blue must never be used to color the cerebrospinal fluid. Its deleterious effects, which at times can be so devastating, have now been widely documented.

The paper of Dr. Hunt and his group represents a very sophisticated approach to a very complicated problem—one already studied extensively by Ryder and his associates—with the addition of arteriography as a means of studying intracranial vascular effects. As they indicate, additional work will be necessary to elucidate the finer aspects of the problem, but a good and impressive start has been made.

Perhaps the most important practical lesson to be learned is the variability in physiological response under different physiological circumstances. Particularly, a warning should be reiterated concerning the Cushing doctrine, for though rising blood pressure and slowing pulse may be exceedingly helpful, as in the case of depressed fracture of the skull in the temporal region we have just seen, they must not be relied upon as an invariable accompaniment of changes of pressure, as Browder and Meyers so clearly indicated. The neurosurgeon always must recognize the variables in the equation of pressure dealt with in the paper by Dr. Hunt and his associates. We shall all look forward with interest to the further elaboration of this work.

This approach to vascular changes is an important one. Dr. Hunt is fully aware that there are pitfalls in angiographic interpretations and unquestionably will guard against them.

DR. EBEN ALEXANDER, JR.: It is not often that one has the skills greased so well. I am sorry I do not happen to have the slides in my pocket; I sent them back home.

I enjoyed both of these papers. I think we can be of some help in pointing out a number of papers that Dr. Ernesto de la Torre published with Dr. Netsky and others on angiographic studies of dog's cerebral vessels. He pointed out it is necessary to inject the dye at a rate of about 1 cc. in 4 sec. through the common carotid artery. If one injects more rapidly than that, the dye will go into the external carotid system, and one does not have a situation comparable to the human at all. One has to get very close, detailed films to point these out.

Our studies, though very much the same physiologically as Dr. Hunt's, would indicate there is no spasm whatever of vessels. After one reduces the intracranial pressure below the systolic pressure, one gets as good filling as before, providing the dog is still alive. If the pressure is not kept over 3 min., the dog will survive. We did not find any effect of torsion with the balloon. We found, actually, the reflection of the pressure from the balloon to the cistern was very poor. This may be attributed to the fact that dogs have a bony tentorium, and it may not be as clearly reflected as in the human.

DR. F. KEITH BRADFORD: I want to answer one of Olom's remarks. This shunt, simulating a ruptured
aneurysm, is quite comparable except the rupture is an all-out rupture and would give Dr. Odom 6 min. to get there to pronounce the patient dead.

I want to say one thing about Dr. Hunt's paper. I wondered if injections into the cisterna magna may have some direct effects on the dog that would be at variance with the effect of intracranial pressure alone. Earlier we used a catheter at T1 because the laminectomy is much easier to perform at that level than in the low lumbar region. In some of these experiments, the effect of blood pressure was unobtainable because the spinal cord was damaged at that level. The effect of increasing intracranial pressure was far more lethal without any protective rise of blood pressure during the intermediate pressures that could be compensated for.

Dr. William E. Hunt: With regard to the study of retinal vessels that Dr. Odom asked, the dogs were upside down on the film and we had to rely on the visualization of the ophthalmic artery at its origin from the internal maxillary artery. It does fail to fill under these conditions.

Slowly rising pressure, of course, is a purely relative term. The relationship of the pressure to the diastolic arterial pressure seems to be the significant factor in provoking the rise. Obviously, what we call "slowly" is very acute by ordinary standards. We are aware that this is a crude technique, Dr. Evans, and that there are many pitfalls. We have other plans as to how we might explore the phenomenon.

With regard to Dr. de la Torre's work on carotid injection, it is for this reason we did our angiograms from the aortic valve because it is pretty well established that, if one puts a needle in the common carotid artery, one cannot fill the intracranial circulation very well unless one delivers a bolus under high pressure, probably because of the rich collateral circulation.

We did not imply—I think this is quite important—there was any factor of spasm. When the pressure of the spinal fluid falls away from the diastolic pressure, the circulation does fill again exactly as it did before, and the calibre of intracranial vessels is a function of the intravascular pressure in almost all instances. This is the Bayliss effect in its simplest form, and is not a function of intracranial pressure. It is a function of the blood pressure.