To investigate the hemodynamics of intracranial circulatory arrest, the authors correlated the findings of noninvasive transcranial Doppler ultrasonography (TCD) with those of transfemoral four-vessel angiography in 65 patients following brain death and intracranial circulatory arrest due to severe intracranial hypertension. The three TCD stages of intracranial circulatory arrest, which have been described previously, corresponded with different levels of extracerebral angiographic cessation of flow. With TCD progression from the first stage (oscillating flow) to the third stage (no flow), the level where the dye stopped descended caudad from subarachnoid to cervical levels. The study shows that, in progressing intracranial hypertension, arterial circulatory standstill within the cranial cavity develops in a distal-to-proximal direction. The basal cerebral arteries remain patent in the early stages of intracranial circulatory arrest. Experimental evidence from the literature, together with the findings of the present investigation, points to the capillary bed as the initial site of the flow obstruction in progressing intracranial hypertension.

KEY WORDS
- intracranial pressure
- intracranial circulatory arrest
- ultrasonography
- cerebral angiography
- brain death

Severe intracranial hypertension is accompanied by characteristic angiographic findings such as delayed, scarce, and tapering contrast filling of the intracranial arteries. With developing intracranial circulatory arrest, the dye column may terminate at any extracerebral level. The unanimous opinion of these authors is that extradural arrest is unequivocal evidence of cerebral circulatory standstill. Disagreement has existed on whether “stasis filling” of subarachnoid arteries without subsequent venous drainage conformed with this diagnosis. The level of angiographic cessation of flow was assumed to descend caudad with time following clinical brain death.

Using transcranial Doppler ultrasound (TCD) monitoring, Hassler, et al., recognized three different stages of intracranial circulatory arrest. The characteristic flow velocity patterns, which are recorded from the basal cerebral arteries, succeed one another in the following order: oscillating flow, systolic spike flow, and zero flow (see Fig. 1). The question studied in the present investigation was whether a correlation exists between the level of the angiographic cessation of flow and the TCD findings, and whether this could eventually provide an answer to the problem of where the vascular obstruction is located in intracranial circulatory arrest.

Clinical Material and Methods
This study included 65 patients, aged 4 to 72 years, who died of intracranial hypertension due to various causes (Table 1). Brain death was determined clinically according to the criteria of the German Medical Council, including a corroborative 30-minute recording of electrocerebral silence in all cases. Angiography was carried out after the diagnosis of brain death and after TCD had suggested circulatory arrest in the anterior and posterior intracranial circulation by the presence of one of the three flow velocity pattern types mentioned above. The TCD correlates of intracranial hypertension and circulatory arrest are given in Fig. 1. The correlation of these findings with cerebral perfusion pressure was described previously in 29 patients, who were included in the present study.

The oscillating TCD pattern is defined as a biphasic flow velocity spectrum with equivalent, opposing inflow and outflow components, so that the resulting time-averaged mean velocity in the evaluated vessel is zero.
The pattern is due to systolic-diastolic alternations of the blood column in the presence of a distal outflow obstruction ("compliance flow"). The systolic spike pattern is characterized by a sharp narrow peak at the beginning of the systole, with a maximum flow velocity of up to 100 cm/sec. Flow velocity in the evaluated vessel during the rest of the cardiac cycle is zero. The systolic TCD spikes show a typical respiration-dependent fluctuation in amplitude. "Zero flow" (that is, absence of TCD signals) can be diagnosed by repeated TCD examinations when signals have disappeared from the vessel. Signals must have been present before. The evaluations must be performed with constant depth of insonation and constant tilt of the probe.

The following basal arteries were studied with TCD in all cases: middle cerebral artery (transTemporal insonation), intracranial internal carotid artery (ICA, transitional insonation depth 55 to 60 mm), and basilar artery (BA, suboccipital insonation depth 85 to 100 mm). Patients who lacked signal from one of these vessels at the time of the initial examination were excluded. The TCD spectra were documented immediately before angiography and were compared with the radiological findings.

In our previous report, the insonation depth for suboccipital recordings from the "basilar artery" was between 70 and 90 mm. However, according to recent data, the junction of the vertebral arteries to form the BA lies deeper in most subjects. Signals from the BA can be recorded reliably with insonation depths of 85 to 115 mm. Therefore, most of our initial suboccipital recordings of oscillating flow or small systolic spike flow made at a depth of 70 to 80 mm were probably obtained from the vertebral arteries rather than from the BA. This explains discrepancies between the data in Table 3 in the present paper and those in Table 2 in the previous publication.

Transfemoral four-vessel angiography was carried out in all patients. The catheters were placed in the common carotid arteries and in the proximal vertebral
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**TABLE 2**

| Angiographic and TCD findings in 130 ICA’s from 65 patients in this series* |
|------------------|------------------|------------------|------------------|------------------|
|                  | Cervical Level   | Petrous Portion  | Siphon           | Anterior Clinoid Process† |
| Oscillating flow | 26              | 1               | 3               | 1                | 11               | 10               |
| Systolic spikes  | 35              | 4               | 19              | 1                | 1                | —                |
| Zero flow        | 69              | 58              | 6               | 5                | —                | —                |

* The transcranial Doppler ultrasonography (TCD) spectra were obtained from the internal carotid artery (ICA) and the middle cerebral artery; they suggested intracranial circulatory arrest prior to angiography. Contrast medium was administered by transfemoral catheterization of the common carotid artery.
† Ophthalmic artery still visible.
‡ Delayed and tapering filling of the supraclinoid carotid or proximal middle or anterior cerebral arteries without subsequent venous enhancement after 26 seconds.

Results

Anterior Intracranial Circulation

The correlation between the TCD data and the angiographic findings in the anterior circulation is given in Table 2. The further the ultrasonographic patterns had progressed from an “early” oscillating type to a late no-flow type, the more caudal was the angiographic termination of the contrast material column. Typical examples are shown in Figs. 2 to 5. The anterior cerebral circulation had ceased in all patients.

Posterior Intracranial Circulation

The correlation between the TCD data and the angiographic findings in the vertebrobasilar circulation is given in Table 3. In most cases, when oscillating or systolic spike patterns were detectable from the BA, the level of the angiographic cessation of flow was situated intradurally. With the occurrence of basilar zero flow according to TCD, the angiographic column of contrast medium usually ended in the atlas portion of the vertebral arteries. Typical examples are shown in Figs. 2, 4, and 5. The posterior circulation (including that in the brain stem and cerebellum) had ceased in all cases.

Discussion

The “nonfilling phenomenon” of the intracranial ICA’s in patients suffering from acutely raised intracranial pressure (ICP) was first described in 1953 by Riishede and Ethelberg. The mechanism causing this “quasi-obliteration” in mostly moribund or decerebrate patients was a matter of dispute in the following years. Riishede and Ethelberg assumed that a herniation-induced brain-stem reflex caused diminution of cerebral blood flow. Their hypothesis was supported by Horwitz and Dunsmore. In 1961, Pribram recognized that ICP elevation was directly related to cerebral angiographic nonfilling in 17 patients following subarachnoid hemorrhage. He rejected his initial working hypothesis of arterial spasms as the cause, and concluded that “the ventricles should be tapped immediately.” Pribram was the first to recognize the potential reversibility of cerebral circulatory arrest. His purely mechanical explanation was supported in an experimental study conducted on monkeys and dogs by Mitchell, et al., who found that the ICP “must equal or exceed the measured systolic blood pressure to prevent filling of the intracranial vessels with contrast medium.” If the ICP was lowered, the cerebral vessels filled well. The matter was resolved in 1966 by Langfitt and Kassell. They simultaneously recorded systemic arterial...
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Pressure (SAP) and ICP during carotid and vertebral angiography in three patients to correlate cerebral perfusion pressure with intracranial nonfilling. The ICP was equal to the systolic SAP in two cases and to the mean SAP in one case. However, the question “of the level of mechanical obstruction of the cerebral circulation in the non-filling phenomenon” remained unanswered.

Intracranial compression of the ICA at its entrance into the subdural space, discussed by Heiskanen, was considered unlikely by Langfitt and Kassell because the ICP usually did not exceed the intraluminal pressure of the vessel and because progressive reduction of the luminal area would increase the wall resistance to further compression in the thick-walled ICA. The lumina of the intracranial ICA’s were patent on postmortem examination in all patients studied by Langfitt and Kassell. They assumed that the cerebral venous outflow becomes obstructed in severe intracranial hypertension. Experimental support of a venous compression mechanism was provided by some authors.

In their experiments on monkeys, Hedges and Weinstein measured the pressure in the orbital ophthalmic artery, the femoral artery, the intracranial subarachnoid space, and the superior sagittal sinus. With ICP elevation (by means of subarachnoid fluid injection) to levels below the ophthalmic artery pressure, the pressure in the sagittal sinus dropped dramatically to an average of 40% of the initial value. As soon as the ICP started to fall, the sagittal sinus pressure returned to its original level. Similar observations had been reported by Bedford. Hedges and Weinstein observed a drop in ophthalmic artery pressure only with ICP elevation to supra-arterial levels. They concluded that: “In subarterial elevations of intracranial pressure, cerebral venous stasis results from cuffing of the cerebral veins where they cross the subarachnoid space...” However, direct evidence for a compression of bridging veins was not provided. Although a “cuff constriction” of veins near the sinus had been observed by Wright at ICP levels a bit below that of the SAP, the respiration-dependent intermittent emptying of the veins continued to be effective in his experiments in dogs (observed by the cranial window technique). Nakagawa, et al., measured an abrupt pressure drop in parasagittal venous pathways in dogs and regarded this as the site of venous compression. In contrast, Auer, et al., could not confirm compression or collapse of bridging veins at or upstream of the entrance into the sagittal sinus in a study using cranial windows in rats under artificially induced intracranial hypertension.

The circulation in cortical vessels under raised ICP was experimentally observed through closed cranial windows by several investigators. Cushing applied this technique in dogs. He produced “general compression” by means of subarachnoid saline injection and noted: “The direct examination of the cortex at this period of equalization of blood pressure and..."
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FIG. 3. Correlation between transcranial Doppler ultrasonography (TCD) and cerebral angiography in intracranial circulatory arrest. A systolic-spike TCD pattern from the right internal carotid artery (ICA r, right side) corresponds to an angiographic cessation of flow in the proximal siphon. Note the respiration-dependent amplitude fluctuations in the ICA r. A zero-flow TCD pattern from the left intracranial carotid artery (ICA l, left side) corresponds to an angiographic arrest of flow in the petrous portion. Left carotid artery angiography was performed 3 minutes after right carotid angiography. The immobile deposit of contrast material in the ICA r remains visible. The drawings were made from the last angiographic images taken 26 seconds after injection of contrast medium. $V =$ velocity.

FIG. 4. Correlation between transcranial Doppler ultrasonography (TCD) and cerebral angiography in intracranial circulatory arrest. Oscillating TCD patterns from the right intracranial carotid artery (ICA r) and from the basilar artery (BA) correspond to subarachnoid stasis filling. A systolic-spike pattern from the left intracranial carotid artery (ICA l) corresponds to an angiographic arrest of flow in the proximal siphon. The drawings were made from the last angiographic images taken 26 seconds after injection of contrast material. $V =$ velocity.

intracranial tension shows, as would be expected, an abrupt blanching of the exposed convolution. Its rosy color becomes of a grayish-yellow hue, and though the pulsating arteries themselves may be seen against this pale background, and the dark-blue veins in the sulci remain filled with blood, little if any circulation presumably passes between them." Similar observations were made by Wolff and Forbes in cats following subarachnoid injection of Ringer’s solution. He wrote: “As the pressure slowly rises the movement of the blood becomes gradually slowed, until, as the intracranial manometer enters the lower levels of the diastolic blood pressure, the blood in the capillaries and venules becomes almost stationary; ... Only when the intracranial pressure is within a few millimeters of the systolic level do the small vessels collapse; ... where they [the arterioles] join the larger arterial branches a to-and-fro movement, synchronous with the pulse wave in the larger arterial branches, is seen. At this pressure level, effective circulation in the cerebral capillaries ceases. In the greater veins there is a continued slow forward movement of blood; this has been observed to continue for half a minute with the intracranial pressure 10 millimeters of mercury above the systolic blood pressure level recorded from the femoral artery.”
Hekmatpanah observed the red blood cell flow in cortical vessels during inflation of extradural balloons in cats. Flow arrest occurred first in the capillaries and venules while blood flow continued in the larger arteries and veins. The venous blood turned bright red at that point, suggesting an arteriovenous shunting mechanism. Gradual sludging of red cells occurred in the capillaries, accompanied by clinical signs of developing brain death. However, blood flow in the larger arteries and veins ceased only with further ICP elevation. The ICP values were not recorded. Hekmatpanah’s findings also suggest that the microvascular bed is the initial site of standstill in evolving intracranial circulatory arrest.

To summarize the previous literature, conclusive evidence of a vascular collapse on one particular level of the “vascular waterfall” is lacking. Instead, circulatory standstill may be due to ICP-induced reduction of the pressure drop along the still patent vascular bed. With decreasing driving force, it seems conclusive that flow arrest commences in the vessels with slowest flow (that is, the capillaries) as observed by Cushing, Wright, and Hekmatpanah.

According to our TCD and angiographic findings, the proximal cerebral arteries definitely remain patent during the early stages of intracranial circulatory arrest. This is evidenced by the oscillating net zero TCD flow from vascular segments that frequently appeared “occluded” on angiography. Nonfilling in this first stage, however, reflects upstream congestion in the presence of downstream obstruction. The typical angiographic correlate is a delayed, tapering stasis filling of the basal subarachnoid arteries (Tables 2 and 3). In the ICA, contrast flow may also terminate at the anterior clinoid process with preserved filling of the ophthalmic artery (Table 2).

In agreement with Kricheff, et al., we do not regard the delayed intracranial stasis filling of early circulatory arrest as an indication of cerebral rest circulation. Such filling of the subarachnoid arterial segments usually

![Fig. 5. Correlation between transcranial Doppler ultrasonography (TCD) and cerebral angiography in intracranial circulatory arrest. Zero-flow TCD patterns from the left intracranial carotid artery (ICA l), right intracranial carotid artery (ICA r), and basilar artery (BA) correspond to an extracranial angiographic cessation of flow in all vessels. The drawings were made from the last angiographic images taken 26 seconds after injection of contrast material.]

![Fig. 6. Intracranial hemodynamics in early (left), intermediate (center), and late (right) intracranial circulatory arrest. The typical progress of the transcranial Doppler ultrasonography patterns (upper) correlates with a descent of the angiographic cessation of flow from subarachnoid to cervical levels (lower). V = velocity.]
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occurred later than 13 seconds following cervical injection of contrast medium. The intracranial contrast medium deposits showed no anterograde drainage. In most cases they were immobile and remained visible on the subsequent angiograms of other vessels. These phenomena may be caused by diffusion of contrast material and layering in the blind vessels.

The second stage of intracranial circulatory arrest is characterized by the appearance on TCD of systolic spikes. The typical angiographic correlate of this condition is an extradural cessation of flow in the cavernous or petrous portion of the ICA (Table 2) and proximal vertebrobasilar stasis filling (Table 3). The origin of the systolic spikes is not yet completely clear. They do not represent net positive intracranial forward flow; rather, they are due to a short anterograde movement of the blood column during the systolic pressure peak which is followed by a slow dispersed backflow during the rest of the cardiac cycle. The backflow component may be temporally too undefined to become detectable with TCD.

In the third stage of intracranial circulatory arrest, motion of the blood columns within the cranial cavity ceases completely. This is evidenced by an absence of any TCD signal which corresponded to an extracranial angiographic arrest of flow in all vessels (Tables 2 and 3). For methodological reasons, TCD does not provide information about the capillary and venous circulation. For the arterial system, however, the present investigation shows that circulatory standstill in intracranial hypertension develops in a distal-to-proximal direction (Fig. 6). During this process, the arterial vascular bed is transformed into a "blind duct" with decreasing volume. Collapse of the large arteries is not a precipitating mechanical factor in developing intracranial circulatory arrest. This conforms with the aforementioned experimental data from the literature, especially those from the studies of Cushing, Wright, and Hekmatpanah (who all used cranial windows), which suggest that the capillaries represent the initial site of circulatory arrest in progressing intracranial hypertension. In the hands of experienced investigators, repeated TCD evaluations can reliably detect intracranial circulatory arrest.

References
5. Büdingen HJ, Staudacher T: [Identification of the basilar artery by transcranial Doppler sonography.] Ultraschall 8:95–101, 1987 (Ger)

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