Doppler frequency spectrum displays a qualitative measure of the blood flow velocity, only. In contrast to blood flow volume, blood flow velocity does not depend on vessel wall diameter. Although blood flow velocities — i.e. systolic peak frequencies — are not influenced by contralateral obstructions, (collateral) blood flow volume may increase due to dilatation of vessel diameter.

References

Non-Invasive Diagnosis of Intracranial Lesions in the Vertebobasilar System. A Comparison of Doppler Sonographic and Angiographic Findings

E. BERND RINGELSTEIN, M.D., HERMAN ZEUMER, M.D., AND KLAUS POECK, M.D.

SUMMARY The accuracy and the reliability of extracranial vertebral Doppler flow (continuous wave Doppler sonography) for the detection of intracranial vertebrobasilar lesions was studied prospectively in twenty-one patients with brainstem disturbances and/or coma. The Doppler findings were compared with cerebral angiography. Doppler sonography was highly reliable for both detection and exclusion of high-grade intracranial vertebrobasilar lesions. The pathognomonic Doppler finding was an abrupt deceleration of the blood column during systole and stoppage or even reflux during diastole. The same blood flow alterations were found in two additional illustrative cases having therapeutically or brain-death induced blockages of the vertebral arteries. The pathophysiologic mechanisms of flow disturbances and the limitations of Doppler sonography in this area are discussed.

THE INTRADURAL PART OF THE HINDBRAIN ARTERIES can be viewed as a functional entity. Thompson et al (1979) coined the term “intradural vertebrobasilar artery.” This arterial segment is the most critical part of the hindbrain vascular system when considering the severity of neurological deficits following occlusive lesions. The closer the vascular lesion is located to the brain the more crucial it is for an impaired cerebral blood supply.\(^2\)\(^3\)

Several non-invasive diagnostic techniques have been applied to the vertebrobasilar system with limited success.\(^4\)\(^5\) However, recent experiences with Doppler sonography in the atraumatic diagnosis and followup of patients with thrombosis of the distal vertebrobasilar (i.e. intracranial vertebrobasilar system) have been encouraging.\(^10\)\(^11\)

The following report describes the clinical, Doppler sonographic and angiographic findings in 21 patients. It was presumed on admission that these subjects suffered from intracranial thrombosis of the vertebrobasilar artery. Clinical and laboratory findings will be compared to define the diagnostic value of CW-Doppler sonography for intracranial occlusive vertebrobasilar disease.

Methods

Doppler Sonography

Using a continuous wave Doppler device with a probe emitting 4 MHz ultrasound (Delalande D 800)
and a zero-crossing frequency meter to provide a voltage proportional to the mean of the instantaneous velocity spectrum, all subjects were examined in the following way (in parts already described by other authors).

1. Insonation of the mastoidal slope of the vertebral artery in both directions is performed (position and direction of the probe are shown in figure 1a).

2. After passing the probe to the other hand it has to be drawn through the supraclavicular fossa in a lateromedial direction (fig. 1b). Simultaneously, the submastoidal region has to be compressed rhythmically and quickly in order to modulate the vertebral signal. With the help of this rhythmical modulation of the velocity profile, the vertebral artery can be identified in the lateral neck region before entering the bony canal of the transverse processes and can be differentiated from other vessels. In order to avoid confusion with the common carotid artery (which may be modulated by compression of the external occipital artery during manipulations at the mastoid) a doublecheck has to be made. When compressing the superficial temporal artery no modulation of the "vertebral signal" should appear (fig. 1c). Otherwise, the signal definitely stems from the common carotid artery and not from the proximal vertebral artery.

3. Using the modulation of the vertebral flow signal by rhythmical compression of the mastoidal slope as a guide, the probe should gradually be moved downward until the subclavian-vertebral junction is reached, indicated by an abrupt change in the sound signal. This is necessary in order to detect lesions of the vertebral arteries at their origins.

4. It is always mandatory to screen the whole vertebrobasilar system. Consequently, the subclavian arteries should be insonated in both directions (fig. 1e and f) and the right common carotid artery must be insonated in an upstream direction in order to detect lesions of the innominate artery (fig. 1d).

Normally, the vertebral flow signal is characterized

**Figure 1.** Mode of extracranial Doppler ultrasound examination of the hindbrain circulation. Insonation of the mastoidal slope (A) and the origin (B, C) of the vertebral artery. Modulation of the flow signal by rhythmical compression at the mastoidal region (arrow in B) helps to identify the vertebral artery in the supraclavicular fossa at its origin. By contrast, modulation of the vertebral flow signal should not occur if the superficial temporal artery is compressed (arrow in C). (D–F) The parent vessels (anomalous artery, subclavian artery) are also examined.
by a gradual decrease of the flow velocity during late systole as well as a small but still measurable flow during diastole (see fig. 3i and k).

The acoustics closely reflect the visual flow diagram. With increased experience, however, the examiner should favor his acoustical perception over the use of mechanical documentation. A blockage present in the intradural vertebral artery or the basilar artery alters the flow profile substantially. Following the systolic acceleration, the blood column is immediately decelerated and completely arrested, so that there is a stagnation of blood during diastole. Most often, the recoil of the blood column from the distal thrombus can also be registered acoustically as an extratone. If intense enough, this recoil induces a reflux phenomenon visible on the recording. To summarize these pathological findings, we coined the term "high resistance flow profile." An in-vivo documentation of various degrees of the "high resistance flow profile" is given in figure 2. In this normal subject, the vertebral blood flow was gradually blocked during neck rotation at the occipitocervical junction illustrating various degrees of the "high resistance flow profile" within the proximal segment of the vertebral artery (see legend of fig. 2).

**Angiography**

For selective arteriography of the vertebral arteries the transfemoral route was used in all cases. The angiographic findings served as a control for the Doppler sonographic diagnoses.

**Patient Selection**

During a period of 16 months, 21 patients were examined with progressive brainstem symptoms or with coma of unknown origin. The severity of the neurological disabilities ranged from "minor brainstem stroke" to a rapid deterioration into coma and respiratory arrest.

After all subjects had been screened neurologically and their histories taken, they immediately underwent Doppler sonography prior to CT and angiography. The ultrasound findings led to a preliminary angiologic diagnosis. The clinical signs and symptoms, the angiologic findings, the therapeutic consequences, the final outcomes and other sample data are listed in table 1. The Doppler findings of two additional patients with specific alterations of the vertebral blood flow following cerebral death (one case) and occlusive neuroradiological intervention (second case) are also described.

**Results**

According to the angiographic data the subjects may be divided into four subgroups.

**Occlusion or Subtotal Stenosis of the Intracranial Vertebral Basilar System (subgroup I)**

In seven patients, occlusion or bilateral subtotal stenosis* of the vertebrobasilar system was predicted correctly on the basis of the Doppler sonographic findings. Thus, no false positive results appeared in this group. Case No. 1 and 2 have already been published elsewhere. 10, 15, 16 The angiographic and sonographic findings of case No. 3 are documented in figure 3.

In four of the patients, thrombolysis was achieved with the use of streptokinase introduced into the vertebral artery. This procedure gave favorable results in three of them (see table 1). Close relationships were found between changes in sonographic and angiographic findings during the course of local intraarterial fibrinolysis10, 15, 16 in all patients who underwent this therapy.

All patients with bilateral occlusion of the vertebrobasilar system revealed the same striking alterations of the vertebral flow pattern as illustrated in figures 2 and 3. The typical sonographic findings were either an absence of a vertebral flow signal on one side associated with a so-called "high resistance flow profile" on the opposite side or a "high resistance flow profile" on both sides.

**Unilateral Occlusion of the Intracranial Vertebral Artery (subgroup II)**

Unilateral vertebral occlusion of the angiograms was predicted by ultrasound in all of the seven patients in this group. In most of the cases, the proximal vertebral artery in the neck was free from thrombus and the lesion was located completely intracranially. In two cases, the occlusion also involved the mastoidal slope of the vertebral artery (cases 8 and 12). In all patients in the subgroup the unilateral occlusion of the distal vertebral artery generally was manifested clinically as a dorsolateral medullary syndrome (Wallenberg syndrome) (See table 1).

In conclusion, this type of unilateral vertebrobasilar thrombosis was characterized by a striking discrepancy between the vertebral flow profiles of each side. On the side of occlusion a "high resistance flow profile" was found whereas normal or even compensatorily elevated flow velocity was seen on the other side.

**No Stenotic Lesions (subgroup III)**

Angiography did not reveal circumscribed stenosing lesions of the vertebrobasilar system in six patients. The diagnosis of vertebrobasilar thrombosis had already been rejected by normal Doppler sonographic findings on admission. These subjects suffered from one of the following illnesses: Drug intoxication (2 cases), extreme elongation and dilatation of the basilar artery due to severe hypertension18 (2 cases), spontaneous brainstem hemorrhage (1 case) and lacunar stroke of the brainstem (1 case). (See Nos. 15-20 in table 1).

**Nonhemodynamic Stenosis of the Basilar Artery (subgroup IV)**

In the one patient in this group (case No. 21), a transient sensorymotor hemiparesis and contralateral third cranial nerve palsy was thought to be a brainstem T.I.A. Doppler sonography was normal. Angiography of the posterior circulation, however, revealed an ap-
FIGURE 2. In-vivo demonstration of the so-called "high resistance flow profile." During sagittal position of the head (position a), a normal flow signal can be achieved at the proximal segment of the left vertebral artery (dots at the top of the tracing indicate rhythmical compression of the perimastoid region for identification of the vessel). Head rotation to the right leads to a flow blockage within the vertebral artery at the occipito-cervical junction (position b) and induces the occurrence of an incomplete "high resistance flow profile" (incomplete means without reflux (arrow) but with lack of diastolic blood flow). Maximal rotation (position c) induces the occurrence of a complete "high resistance flow profile" with reflux during diastole (crossed arrow, reflux is indicated by arrow head). Variations of the flow profile (double arrow) are due to slight changes in the position of the probe. When the head is returned to a sagittal position, a normal flow profile reappears (position d).

FIGURE 3A-K. Angiographic and sono-graphic findings in patient No. 3 on admission and following successful transluminal intravertebral fibrinolysis. (A) Vertebral arteriogram revealed complete occlusion of the basilar trunc (arrow). The trickle of contrast medium running straight up the midline indicates the posterior meningeal artery. (B, C) Flow signals of the left vertebral artery are very faint and short. (D, E) Over the right vertebral artery, perpendicular flow with reflux phenomena (arrow heads) and sharp and short signals has become obvious. Vertical bar indicates a mean flow velocity of 20 cm/s (chart velocity 5 mms). (F) Following fibrinolytic therapy, the vertebrobasilar system now appears quite normal (arrow indicates site of former basilar occlusion). (G-K) Striking improvement of the ultrasound flow signals at the mastoidal slope (G, I) and at the vertebral origin (H, K) paralleled the lysis of the thrombus. After recanalization of the basilar trunc, the signals showed a gradual increase of the flow velocity during systole and considerable diastolic flow occurred (K). Dots at the top of the tracing indicate period of compression at the mastoidal region.
TABLE 1  
Clinical and Laboratory Findings in the Patients Examined

<table>
<thead>
<tr>
<th>Case no./sex/age</th>
<th>History and clinical state on admission</th>
<th>Doppler findings</th>
<th>Angiographic findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 F 27</td>
<td>Clouding of consciousness, hemihypaesthesia, hemiataxia</td>
<td>RVA: no signal LVA: minimal flow</td>
<td>RVA occluded, top-of-the-basilar-embolus</td>
</tr>
<tr>
<td>2 F 49</td>
<td>Nuchal headache, vomiting, transient cortical blindness</td>
<td>RVA: subtotal stenosis at mastoidal slope. LVA: no signal</td>
<td>Bilateral subtotal stenoses of VA’s at mastoidal slopes</td>
</tr>
<tr>
<td>3 F 34</td>
<td>Dysarthria, gaze palsy, nystagmus, hemiataxia, hemihypaesthesia, tetraparesis</td>
<td>RVA: HRP LVA: HRP with PF</td>
<td>RVA: subtotal stenosis and proximal occlusion of the basilar trunk</td>
</tr>
<tr>
<td>4 M 39</td>
<td>Severe nuchal headache 1 week previously, acute midbrain upper pons-stage</td>
<td>Faint signal with PF on both sides</td>
<td>Subtotal stenoses of intradural vertebral arteries on both sides, severe basilar trunc atherosclerosis</td>
</tr>
<tr>
<td>5 M 43</td>
<td>Acute midbrain-stage</td>
<td>RVA: no signal LVA: HRP with PF</td>
<td>Complete basilar trunc thrombosis</td>
</tr>
<tr>
<td>6 M 59</td>
<td>Severe brainstem dysfunction, coma</td>
<td>RVA: HRP with PF LVA: HRP</td>
<td>Complete basilar trunc thrombosis</td>
</tr>
<tr>
<td>7 M 57</td>
<td>Transient diplopia 3 weeks prior to onset, locked-in syndrome</td>
<td>RVA: no signal LVA: HRP with PF</td>
<td>Complete basilar trunc thrombosis</td>
</tr>
<tr>
<td>8 M 64</td>
<td>Top of the basilar-syndrome</td>
<td>LVA: normal RVA: HRP, mastoidal collaterals</td>
<td>RVA: occlusion beyond mastoidal slope</td>
</tr>
<tr>
<td>9 F 52</td>
<td>Dorsolateral medullary syndrome</td>
<td>RVA: normal LVA: HRP</td>
<td>LVA: occlusion of intracranial segment</td>
</tr>
<tr>
<td>10 F 52</td>
<td>Foville syndrome</td>
<td>RVA: normal LVA: HRP with PF, tight ICA stenosis</td>
<td>Confirmative LVA: occlusion of intracranial segment</td>
</tr>
<tr>
<td>11 M 69</td>
<td>Dorsolateral medullary syndrome</td>
<td>RVA: HRP with PF LVA: normal</td>
<td>RVA: occlusion of intracranial segment</td>
</tr>
<tr>
<td>12 F</td>
<td>Transient perioral tingling, dorsolateral medullary syndrome</td>
<td>RVA: normal LVA: HRP, mastoidal collaterals</td>
<td>RVA: occlusion beyond mastoidal slope</td>
</tr>
<tr>
<td>13 M 67</td>
<td>Dorsolateral medullary syndrome</td>
<td>RVA: normal LVA: HRP with PF</td>
<td>RVA: occlusion of intracranial segment</td>
</tr>
<tr>
<td>14 M 64</td>
<td>Dorsolateral medullary syndrome</td>
<td>RVA: normal LVA: HRP with PF</td>
<td>RVA: occlusion of intracranial segment</td>
</tr>
<tr>
<td>15-20 (3×M, 3×F)</td>
<td>Severe brainstem dysfunctions, coma</td>
<td>Bilaterally normal</td>
<td>Confirmative, but 2 patients with severe ectasia and elongation of basilar artery</td>
</tr>
<tr>
<td>21 M 42</td>
<td>Two attacks of transient hemihypaesthesia</td>
<td>Bilaterally normal</td>
<td>Mid-basilar trunc stenosis</td>
</tr>
</tbody>
</table>

"Illustrative" cases, see case-descriptions in the text.

LIF = local intravertebral fibrinolysis, RVA = right vertebral artery, LVA = left vertebral artery, PICA = posterior inferior cerebellar artery, PF = perpendicular flow (to-and-fro movement of blood column), HRP = high resistance profile of mean blood flow velocity, ICA = internal carotid artery, inf. = infarction.

proximately 70% smooth hour-glass-shaped stenosis of the mid-basilar artery. Examination of cerebro-spinal fluid and serum gave a strong positive reaction for syphilis, indicating the probability of syphilitic angiitis of the central nervous system.

Two Additional Cases

The above-mentioned findings were substantiated by two illustrative cases, in whom the circulatory disturbances were not due to atherothrombosis of the intracranial vertebrobasilar artery but to brain death with stoppage of the intracranial circulation or a therapeutically produced extracranial blockage of the vertebral artery. In the latter case, a posttraumatic aneurysm and an arteriovenous fistula of the right vertebral artery were treated by embolization with glue, leaving a blind proximal stump of the vertebral artery. Following this procedure, ultrasound revealed the same high resistance flow profile as in the other patients of subgroup I and II.

Discussion

Continuous wave Doppler sonography has proven to be useful and reliable in the detection of vascular lesions of the extracranial carotid arteries. However, with a few exceptions, diagnostic value of sonography at the vertebral arteries has been underestimated.
As far as we know, intracranial lesions of the vertebrobasilar system have not been taken into account by other authors during non-invasive studies of the hindbrain circulation. Meanwhile, the advent of new therapeutic approaches in patients suffering from thrombosis of the intracranial vertebrobasilar system has become a stimulus for the establishment of non-invasive, and reliable diagnostic procedures. A strikingly close relationship between Doppler sonographic and angiologic findings could be demonstrated when the angiologic states of the patients changed during intravertebral fibrinolytic therapy. In the paper presented here, the results of Doppler sonography for non-invasive detection of hemodynamically relevant intracranial lesions of the vertebrobasilar system could be substantiated for both the primary diagnosis of such lesions as well as during their followup. In the patients suffering from bilateral or unilateral vertebrobasilar occlusion as well as those without stenosing lesions, none of the diagnoses made by Doppler ultrasound was false-negative or false-positive. However, some limitations of non-invasive screening of the vertebrobasilar system became apparent during this study: 1. Although upstream from the lesion a high resistance profile with decreased diastolic flow and reduced mean velocity can be recorded even if the lesion itself is inaccessible to the probe, such effects are not found unless the underlying vascular lesion is hemodynamically significant. A measurable increase of prestenotic flow resistance cannot occur until the stenosis has reached a degree of at least eighty percent or more. Case No. 21 is a good example for demonstrating this limitation. In

<table>
<thead>
<tr>
<th>Management</th>
<th>Outcome</th>
<th>CT on admission</th>
<th>Repeat CT</th>
</tr>
</thead>
<tbody>
<tr>
<td>LIF</td>
<td>Minimal deficit</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>LIF</td>
<td>Partial defect of visual field</td>
<td>Occipital lobe infarction</td>
<td>Identical</td>
</tr>
<tr>
<td>LIF</td>
<td>Moderate deficits</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>LIF</td>
<td>Succumbed next day from brain death</td>
<td>Normal</td>
<td>Necrosis of pons, multiple bleedings</td>
</tr>
<tr>
<td>Conservative</td>
<td>Died 2 days later</td>
<td>Lacune of thalamus</td>
<td>Necrosis of pons and cerebellum</td>
</tr>
<tr>
<td>Conservative</td>
<td>Locked-in, died 2 months later</td>
<td>Necrosis of pons and cerebellum</td>
<td>Identical</td>
</tr>
<tr>
<td>Conservative</td>
<td>Bulbar stage, died 4 weeks later</td>
<td>Necrosis and swelling of pons</td>
<td>Identical</td>
</tr>
<tr>
<td>Conservative</td>
<td>Cortical blindness, mental deficiency</td>
<td>Lacunes and bi-occipital infarctions</td>
<td>Identical</td>
</tr>
<tr>
<td>Anticoagulative drugs</td>
<td>Moderate defects, able to walk</td>
<td>Partial inf. of PICA territory</td>
<td>Identical</td>
</tr>
<tr>
<td>Conservative</td>
<td>Hypaesthesia, ataxia</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Conservative</td>
<td>Nearly full recovery</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Conservative</td>
<td>Nearly full recovery</td>
<td>Normal</td>
<td>No control</td>
</tr>
<tr>
<td>Conservative</td>
<td>Partial recovery</td>
<td>Small ICA-territory infarction</td>
<td>No control</td>
</tr>
<tr>
<td>Anticoagulative drugs</td>
<td>Deterioration, death from pneumonia</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Conservative</td>
<td>Slight deficits or full recovery; one death</td>
<td>Lacune of thalamus (case no. 17), brainstem bleeding (case no. 16), otherwise normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Penicillin, corticoids, anticoagulative drugs</td>
<td>No further stroke</td>
<td>Normal</td>
<td>Normal</td>
</tr>
</tbody>
</table>
this subject with a 70% stenosis of the midbasilar trunk, no upstream alteration of blood flow occurred, thereby preventing the detection of the lesion by ultrasound.

2) From carotid ultrasound studies,13 we know that even acute embolic occlusions of the middle cerebral artery can rarely be detected by changes of the extracranial carotid flow profile. The collateralizing capacities of the circle of Willis compensate for the increase in peripheral flow resistance. Comparable mechanisms also occur within the vertebrobasilar system. In case No. 2,10 a circumscribed "top of the basilar" occlusion did not severely increase the peripheral flow resistance of the vertebrobasilar vessels as the blood was bilaterally drained by way of the cerebellar arteries. Nevertheless, the vertebral velocity profiles were abnormal in that the profile could not be detected on one side and revealed a complete lack of diastolic blood flow on the other side. Since a reflux phenomenon was not present, the diagnosis of basilar thrombosis in this patient could only be presumed.

3) Local hemodynamic changes occurring with severe occlusive disease of the extracranial vertebral or subclavian arteries may mask the high resistance flow profile of an intracranial vertebrobasilar lesion.11

The two illustrative cases described above convincingly demonstrate that alterations of blood flow within the extracranial segment of the vertebral artery are characteristic and pronounced if the distal portion of the artery is completely occluded. Even if the site of the blockage is located intracranially, as was the case with patients having brain death, typical extracranial sonographic findings can regularly be achieved and allow for definite diagnosis. Particular changes of the flow patterns during brain-death have been described by other authors with reference to the carotid system21-22 but not to the vertebral system. Other causes of intravertebral to-and-fro movement of the blood column, i.e. latent subclavian steal mechanism due to moderate stenoses of the proximal subclavian artery or with subtotal stenosis of the extracranial vertebral artery at its orifice and severe valvular heart disease, must be excluded.11, 26

The examiner’s acoustical training is essential for correct non-invasive diagnosis of intracranial lesions of the vertebrobasilar system.23 In some obese patients, or if the vertebral artery is hypoplastic, the intensity of the flow signal is not strong enough to be clearly visible on the scope and cannot be distinctly recorded. The examiner, in such cases, would then have to rely on his acoustic experience alone.

With respect of the promising results of local intraarterial fibrinolysis, an early diagnosis of occlusion of the vertebrobasilar system is of great importance. The latter might help prevent propagation of thrombus that would worsen the patient’s prognosis.1, 10, 11, 17, 28-33

Acknowledgment

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Reduction of Neurologic Deficit by 1,3-Butanediol Induced Ketosis in Levine Rats

EDWARD F. LUNDY, M.D., JAN DYKSTRA, BENOIT LUYCKX, GERALD B. ZELENOCK, M.D., AND LOUIS G. D’ALECY, D.M.D., PH.D.

SUMMARY The objective of this study was to determine if 1,3-butanediol would reduce a neurologic deficit in rats exposed to ischemic-hypoxia (Levine rats). Age and weight matched male Sprague-Dawley rats were anesthetized with 2% halothane. The right common carotid and external jugular vein were ligated and cannulated and EEG screws were implanted following a 2 hour recovery period. Thirty minutes prior to exposure the rats received either 1,3-butanediol (47 mmole/kg i.v.; n = 11) or an equal volume of saline (n = 10). The rats were then exposed to 4.5% O₂ until mean arterial blood pressure fell to 70 mm Hg. The oxygen level was then increased to 8% for 30 minutes, after which the rats were returned to room air. Posture, hemiparesis, circling, shuffling, activity, and ability to hang on to a vertical screen were scored 1 (normal) to 5 (severe deficit) at 2 and 20 hours after insult. The time to 70 mm Hg was extended from 7.9 ± 0.9 min for saline treated rats to 19.0 ± 2.3 min for the 1,3-butanediol treated rats (p < 0.001). All eleven 1,3-butanediol treated rats survived the hypoxic insult; 90% (9/10) saline treated rats died. In an attempt to reduce the insult, six additional saline treated rats were switched to 8% O₂ at 75 mm Hg and still 4/6 died. The mean score at 20 hours for three surviving saline treated rats was 3.4. A significantly better (p < 0.002) mean 20 hour score for the surviving 8/11 1,3-butanediol treated rats was 1.2. 1,3-butanediol increases survival and decreases the neurologic deficits associated with this ischemic-hypoxic insult.

INTerventions in cerebral ischemia produce clinical benefit by reducing mortality and/or by lessening neurologic sequelae of a cerebral ischemic event. The mortality associated with spontaneous cerebrovascular events is well established.1-3 Equally well established, but less widely appreciated, is the magnitude of the residual disability that accompanies cerebrovascular events.4,5 This differs from ischemic events in other vascular beds in that the morbidity that accompanies myocardial infarction or peripheral vascular occlusive disease, while potentially disabling, pales in comparison to the morbidity of a cerebral infarction. Blindness, paralysis, aphasia, and loss of higher cortical function make cerebral infarctions more visibly disabling than comparable tissue insults in other vascular beds.

Previous studies from this laboratory have demonstrated enhanced survival following 1,3-butanediol administration in two animal models: mice exposed to a
Non-invasive diagnosis of intracranial lesions in the vertebrobasilar system. A comparison of Doppler sonographic and angiographic findings.
E B Ringelstein, H Zeumer and K Poeck

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