must admit that we are proud to have "originally championed the autoradiographic approach to Xenon/CT LCBF measurements" at a time when most CT scanners were slow (≥ 20 seconds scan time) and interscan delays were much longer (≥ 30 sec) than those currently available on many machines. There is no doubt that this method is now outdated. Since 1979–80 we, along with other groups, have been developing and using more sophisticated multivariable methods of analysis such as the weighted mono- or bi-compartmental least square fits. We believe that the true flow through a tissue volume should be used as a reference flow in error analyses and therefore, we do agree with Rottenberg, et al that what they define as \( E_2 \) is the error due to heterogeneity if the mono-compartmental model is used. Rottenberg’s data show that \( E_2 \) is significantly smaller than \( E_1 \) in tissue with a significant mixture (percent gray < 75%). Therefore, the statement we made in our recent review in STROKE is fully supported by their own data. The rest of the story has already been published and rebattled in the Journal of Cerebral Blood Flow and Metabolism and we see no reason to repeat it here.

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Atriopathic Arrhythmias — Sick Sinus Syndrome

To the Editor:

I would like to comment on the paper of Abdon et al in Stroke 13 (6), 832–837, 1982, by the following letter-to-the-Editor:

The study of Abdon et al demonstrates that the incidence of finding the heart as the source of a focal cerebral infarction is related to the intensity of investigating the heart as such. In their study on long-term electrocardiographic recording they found a significant higher prevalence of "atriopathic arrhythmias" in a population of stroke patients than in a reference group. Sick sinus syndrome was one of these arrhythmias and it was present in 19 of 88 stroke patients and in only 9 of 103 patients of the control group. I wonder what strategy the authors apply when sick sinus syndrome is a fortuitous finding, and when it appears to be present in a stroke patient. Is anticoagulant therapy indicated? The authors use the temporal pattern at stroke onset as the only criterion to distinguish embolic from thrombotic infarction and found approximately the same percentage of atriopathic arrhythmias in both groups. Does this imply that these arrhythmias occur in patients with thrombotic stroke while the cardiac arrhythmia is not causally related to the occurrence of the stroke, or was the prior clinical diagnosis incorrect and suffered the 15 patients in the supposed atriopathic group in fact a cerebral embolus? This point could be relevant regarding anticoagulant treatment.

Sincerely Yours,

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References


The authors reply:

We are pleased to answer the questions raised by Dr. Lodder. In our report the sick sinus syndrome (SSS) was present in 21 of 86 stroke patients. It has been shown that persons with stroke and SSS, even where the etiological connection is uncertain, have a future risk for cerebral emboli of 7% per individual and year.1 We therefore find preventive measures advisable where possible and subscribe to the following general policies:

1) If other aggravating drugs are present, i.e. beta blockers or digitalis in the case of sinus bradyarrhythmia, they should be withdrawn and arrhythmia re-evaluated.

2) Where possible, anticoagulants should be given.

Due to the high frequency of concomitant disease in this patient group anticoagulants are often inadvisable and individual judgement must be made.

We have followed tradition in classifying stroke which occurs during sleep or with unclear onset as "thrombotic". Some of these strokes may well have been embolic in origin. Only one patient with a "thrombotic" stroke and atriopathic arrhythmia demonstrated progressive neurological deficit after admission. We are more inclined to regard the classification of stroke as embolic or thrombotic using only anamnestic data as insufficient than to consider the presence of atriopathic arrhythmia in the "thrombotic" group as purely coincidental.

Yours sincerely,

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Joyce Carlson, M.D.
Ingemar Turesson, M.D.
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Hypertensive Encephalopathy

To the Editor:

Dinsdale,1 in his recent review of hypertensive encephalopathy (HTE) in this journal; mentions sodium nitroprusside (SNP) as often being the initial drug of choice in the treatment of HTE. While this choice is supported by a large segment of the available neurology and internal medicine literature, we feel that a significant amount of theory and objective information exists that make this drug appear less than optimal. The danger in use of SNP is the same mechanisms as for other direct acting vasodilators: cerebral arterial dilation allows delivery of more pressure to the microvascular bed, hence increased edema, and increase in cerebral blood volume (CBV), both of which increase intracranial pressure (ICP) when intracranial compliance is low.2, 3

There would not be a problem in using SNP in HTE if there was not a problem with either intracranial compliance or ICP. However, this is not the case. In a study reported in 1954, elevated lumbar pressures were found in 39% of patients with HTE.4 More recently, Griswold5 measured ICP with a subdural bolt in three children with stage III coma from HTE. ICP was found to be elevated in two of these patients with a range of 32–70 mm Hg.

Studies2, 6, 7 have already demonstrated a significant increase in ICP and decrease in CPP while using SNP. One study7 looked at ten patients with intracranial masses and demonstrated marked increases in ICP with a diminished CPP while using SNP, leading them to conclude that SNP should not be used in patients with increased ICP unless measures to improve intracranial compliance are instituted prior to its use. In a comparison of 45 patients in which deliberate hypotension was used for neurosurgery, in normocapnic patients SNP caused a significant rise in

References


2. Dinsdale J: Hypertensive Encephalopathy

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ICP compared to trimethaphan. No difference in ICP was found with either drug when the patients were hypocapnic. Because of the potentially deleterious effect on intracranial dynamics, it may be unwise to use either SNP, nitroglycerine or diazoxide unless ICP is being monitored, since all of these drugs have been shown to cause increases in ICP.2, 3, 6, 8

It is unclear to us whether the patients who do poorly with HTE do so because of their disease or treatment. Because of its lack of direct effect on cerebral vessels,7 trimethaphan may be safer to use initially in HTE, as it would not be expected to increase CBV and brain edema and hence ICP.

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References


The author replies:

I agree with Drs. Egol, Snyder, and Grenvik that increased intracranial pressure (ICP) is a serious factor in hypertensive encephalopathy (HTE). As indicated in my article, the reversal of the process producing edema requires the reduction of systemic arterial pressure, an urgent measure in the treatment of HTE.

Reported studies of the cerebrovascular effects of sodium nitroprusside (SNP) provide conflicting results. Stullken and Sokoll1 found no increase in ICP when SNP was administered to normotensive cats, in contrast to trimethaphan which did produce an increase in ICP. However, they found an increase in ICP when vasopressin was given to animals following systemic hypotension induced by either drug. As noted by Marsh2 studies such as those by Stullken and Sokoll persuaded some anesthetists to change from trimethaphan to SNP for blood pressure control during induction. Other studies3-4 have demonstrated elevation in ICP with SNP. Differences in studies undoubtedly result from factors such as species differences, variations in the nature and extent of brain swelling, level of systemic blood pressure, the method of injecting the test drug, extent of blood pressure drop with hypotensive therapy and paco2.

Care and caution must be exercised when administering any of the major hypotensive drugs. They must be used in a setting which enables constant monitoring of vital signs, but I do not agree that attempts should be made to monitor ICP in all patients with HTE.

Although it is obvious that controlled clinical data are unavailable, it is my opinion that patients with HTE did more poorly when there were no drugs such as SNP at hand to lower blood pressure effectively. Further studies in both animals and man, may lead to a better understanding and more general agreement about the modes of action of SNP.

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Mitral Valve Prolapse and Risk of Stroke

To the Editor:

Considerable difficulty exists in reconciling the rather frequent occurrence of mitral valve prolapse (MVP) in stroke patients (particularly young stroke patients), and the rarity of stroke in MVP patients in clinical series.1 This paradox is clearly presented in a recent issue of the Journal.2 One explanation, expressed in an editorial opinion by Hart and Easton, is that the frequency of stroke due to other causes is low in the younger patients with MVP thereby allowing the relation of stroke to MVP to be clearly seen. Among older persons, they suggest, the stroke associated with MVP is overshadowed by the more frequent occurrence of stroke of all types.3 We suggest that selective bias is the explanation for the phenomenon, operating in case "selection" in neurological patient series.

A classic study of how a collection of clinical cases reported from a teaching hospital differs from findings derived from a population is that of Crawford and Morris.4 Noting wide variation in the reported frequency of age, sex and anticoagulant use in cases of ruptured ventricle a comparison was made of the teaching hospital cases and coroners' mortuaries in London during 1957 and 1958. The Table summarizes their findings. While overall, ventricular rupture occurred equally in men and women in teaching hospitals, there appeared to be a predominance of men in coroners' mortuaries.

Table 1 Spontaneous Rupture of Cardiac Ventricle London 1957-1958

<table>
<thead>
<tr>
<th>Age</th>
<th>No. of cases</th>
<th>Average annual incidence per 1000 population</th>
</tr>
</thead>
<tbody>
<tr>
<td>45-54</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td>55-64</td>
<td>10</td>
<td>3</td>
</tr>
<tr>
<td>65-69</td>
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<td>70-74</td>
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<td>1</td>
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</tr>
<tr>
<td>80-84</td>
<td>3</td>
<td>18</td>
</tr>
<tr>
<td>85+</td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>

Hypertensive encephalopathy.
A B Egol, J V Snyder and A Grenvik

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