would be of clinical interest to know something about the condition of these patients e.g. did any of the patients with VT have a suspect associated myocardial infarction or did complete heart block occur in patients (on digitalis?) with severe neurologic deficit including an impaired sensorium?

The only patient in our series of 100 consecutive stroke patients with 24 h Holter ECG recordings, soon after admission who showed repetitive VT also had an acute myocardial infarction. Fifty-five percent of our patients in sinushythm showed VPB's and serious forms were usually seen in association with a concomitant acute myocardial infarction and/or congestive heart failure.

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References


Editor's Note: The previous letter was submitted to the authors for their comments.

To the Editor:

The findings of Helmers and his colleagues are generally consistent with our own data. In both series of 100 stroke patients, there were relatively few life threatening cardiac arrhythmias. In particular, ventricular tachycardia and complete heart block were quite rare. The proportion of patients with ventricular arrhythmias was similar in the two studies and there were 22 patients with atrial fibrillation in our group and 23 in the earlier series.

Dr. Helmers speculates if our results would have been different if we had compared stroke patients with heart disease but no hypertension with the remaining patients with respect to arrhythmia occurrence. Further data analysis has shown that 12 of the 25 stroke patients with heart disease and no hypertension had serious cardiac arrhythmias (as defined in our paper) compared with 23 of the 75 individuals without heart disease (difference not significant). Similarly, serious cardiac arrhythmias occurred in 64 hours of monitoring in the 25 patients with heart disease compared with 161 hours in the remaining 75 patients (difference not significant). The most serious arrhythmias (ventricular tachycardia and complete heart block) were observed during 4 hours in 4 stroke patients with heart disease compared with 15 hours in 2 stroke patients without heart disease. None of these patients with serious arrhythmias had associated ECG findings consistent with acute myocardial infarction. Two of the 4 stroke patients were on digoxin at the time of admission but the dose being administered was appropriate and serum levels were not recorded.

These comparative findings do not support an increased frequency of cardiac arrhythmias in stroke patients with heart disease. However, neither study can draw any firm conclusions regarding the possible importance of coexisting heart disease and the occurrence of cardiac arrhythmias following stroke since the stroke patients in both groups did not undergo specific tests to diagnose heart disease (e.g. coronary arteriography or treadmill exercise). The combined data in the 200 stroke patients do suggest that potentially life threatening arrhythmias following acute stroke are relatively rare whereas virtually all categories of cardiac arrhythmias appear to occur more frequently raising the possibility of a causal association between the cerebral event and cardiac abnormalities.

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References


Cerebral Infarction in Young Adults

To the Editor:

I enjoyed reading the paper written by Robert G. Hart M.D. and Vincent T. Miller M.D. entitled: “Cerebral Infarction in Young Adults: A Practical Approach,” which appeared in STROKE 14, No. 1, pages 110–114. For the sake of completeness I want to point out that in table 1 of this paper “Differential Diagnosis of Cerebral Infarction in Young Adults,” another two categories should be added. One of them is “Parasitic” (Cysticercosis and Trichina Vasculitis) which should occupy No. 6 in the subgroup A of group III. In subgroup B of the same group III, there should be also a place for “Transient Embolic Aorto-Arteritis.”

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References


Stroke in Young Adults: Parasites and Occult Cardiac Disease

To the Editor:

We thank Dr. Rangel-Guerra for his comments. In our table 1, we classified transient embolic aorto-arteritis in group IIC (transient embolicogenic aortitis) because of its typical clinical presentation as embolic stroke. To our knowledge, Dr. Rangel-Guerra’s patient is the only one reported from the Western Hemisphere. Arteritis related to parasitic infestation should not be overlooked. Meningobasal cisticercosis can invade arterial walls with resultant occlusion and stroke, and should be considered in endemic areas. The neuropathology of CNS trichinosis would appear to be that of encephalitis and, although focal features and angiographic attenuation of small arteries have been reported, primary trichinella vasculitis with a stroke-like presentation is a very uncommon feature of an uncommon illness.

Other colleagues have raised the question of the need for cardiac catheterization in young adults with unexplained stroke who have a normal cardiac examination, chest x-ray, ECG, and echocardiography. Cardiologists are often reluctant to undertake this invasive procedure in these circumstances, suspecting it to be of too low yield.

However, several reports suggest that cardiac catheterization may be important in this subset. Aupy et al. reported 12 young stroke patients with suspected embolic stroke based on cerebral angiography but in whom clinical examination, ECG, Holter monitoring and M-mode echocardiography was nondiagnostic. In ten of these 12 patients, angiography provided a possible source of embolism: mitral valve prolapse in eight, left atrial myxoma in one, left atrial thrombus in one. Rice et al. described a 34 year-old man with multiple cerebral emboli and a normal cardiac examination, including ECG, chest x-ray and echocardiography. Cardiac catheterization demonstrated apical hypokinesis; gingival and rectal biopsies established a diagnosis of amyloidosis.

Paradoxical embolism via a patent foramen ovale causing stroke has been emphasized by several workers and requires right-heart catheterization for diagnosis. Transient right to left shunting during Valsalva maneuver has been documented by cardiac catheterization in patients with occult embolic sources who have a patent foramen ovale. 5, 8
To the Editor:

Woodcock, Ropper and Kennedy in their recent article (Stroke 13: 785–787, 1982) on “High Dose Barbiturates in Non–Traumatic Brain Swelling: ICP Reduction and Effect on Outcome” have added to the literature five patients treated for focal infarction with deep barbiturate coma. The desire to intervene with unproven therapeutics in a patient whose condition seems desperate can be appreciated and need not be criticized; however the risk of such treatment must be appreciated. The effectiveness of barbiturates for global cerebral ischemia remains unproved, but the ameliorating effects of barbiturates in focal cerebral ischemia in the experimental setting are well documented. The effectiveness of barbiturates in focal ischemia however is very much time dependent. In fact, it is likely that a therapeutic window exists and that barbiturate coma started too late can in fact be detrimental.

Since all five of the patients presented by the author had or were developing high ICP, from a stroke, barbiturates were obviously instituted in the hopes of controlling the ICP rather than protecting from ischemia. Previous clinical experience of barbiturate therapy for acute infarction reported by Rockoff and Associates revealed similar results. These authors described four patients with strokes who were treated promptly with five days of barbiturate therapy. All four patients died of high intracranial pressure following barbiturate withdrawal. It is our suspicion that in this group of patients, barbiturates may in fact be detrimental. There is no doubt that barbiturates will temporarily lower ICP in the laboratory and clinical setting, but there is no evidence that it makes any appreciable permanent difference in “malignant” ICP resulting from edema secondary to focal infarction. Furthermore recirculation appears to be a critical component of barbiturate protection in our experimental model. In the MCA occlusion model without recirculation, the administration of barbiturates were deleterious as evidenced by an increased incidence of malignant ICP compared to controls.

In light of the risks of aggravating ICP with either delayed barbiturate administration, or administration in a non–revascularized brain, we believe that clinical use of barbiturate coma for focal cerebral ischemia should await further delineation in the laboratory of the relationship of such variables as dosage, timing and recirculation to potential therapeutic and deleterious results.

We feel that the clinical use of barbiturates for focal cerebral ischemia is most appropriate in the operating room for temporary vessel occlusion — where its administration is prompt and where recirculation can be established.

We agree with the cautious approach recommended by Dr. Yatsu in his eloquent editorial.

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References

To the Editor:

It is a form of poetic yet savage irony that the brain — the highest expression of evolutionary development — is so frail and exquisitely vulnerable. Rising expectations for scientific breakthroughs in brain resuscitation, provoked as a natural consequence of modern technological advances, behave as a menacing crowd, seeking its due but kept at bay. The sobering cautions, however, of Spetzler et al., on the uncertainties of pharmacologic protection place the human problem in proper perspective. The staggering complex reactions triggered by strokes simply cannot be reduced to single factors such as raised intracranial pressure since vagaries exist, akin to Heisenberg’s “uncertainty principle” in physics. Like buttressing a collapsing house, preservation of
Stroke in young adults: parasites and occult cardiac disease.
R G Hart and J D Easton

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The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/14/5/829.2.citation

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