LETTERS TO THE EDITOR

Letters to the Editor will be published, when suitable, and as space permits. They should not exceed 1,000 words (typed double spaced) in length, and may be subject to editing or abridgment.

Rebound Hypercoagulability

To the Editor:

The article by Link et al1 reported a dramatic increase in the risk of cerebral infarction (42% per year) during the four months following cessation of anticoagulant therapy in stroke patients, stating there "was no obvious explanation for our high stroke rate." Olsson et al2 reported two cerebral infarctions within one month of cessation of anticoagulants among 67 TIA patients (a risk extrapolated to 36% per year). A possible explanation for the high stroke rate following withdrawal of anticoagulants noted in these studies is a rebound hypercoagulable state induced by cessation of anticoagulants.

The existence of rebound hypercoagulability after cessation of coumadin is controversial.3 Tinker and Tarhan,4 for example, reported no thrombotic complications in 159 patients with prosthetic heart valves in whom coumadin was discontinued for elective surgery, suggesting no clinical hypercoagulability exists in this setting. Anecdotally, we have recently seen two patients with rheumatic heart disease and prosthetic valves who have sustained preoperative cerebral infarctions shortly after cessation of coumadin for elective surgery. Laboratory evidence for a hypercoagulable state persisting up to eight weeks following cessation of anticoagulants has recently been presented.5 Of related interest are reports of cerebral infarction in patients with intestinal malabsorption of vitamin K which occurred following parenteral administration of vitamin K in direct relation to correction of prothrombin time.6

Could the high rate of cerebral infarction following cessation of anticoagulants or correction of vitamin K deficiency be totally, or in part, iatrogenic? It would be of interest to know the specific temporal relationship between cessation of anticoagulants and onset of cerebral infarction in the 10 patients reported by Link et al.1 Such a rebound phenomena, if it exists, might have profound implications for chronic anticoagulation of patients with dubious compliance as well as for elective cessation of therapy.

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References


Comment on "Study Design of Stroke Treatments"

To the Editor:

We found the review of "Study design of stroke treatments" by Spence et al (Stroke, 13, 94-99, 1982) both interesting and controversial and are encouraged by the increasing cooperation between clinicians and methodologists evident in current publications.

The discussion of the scoring system for acute stroke patients was of particular interest, since we are at present designing just such a system. We agree that previous scoring methods are woefully inadequate, either not reflecting the bedside clinical judgement or utilizing such useless modalities as pupillary size or the excitableability of deep tendon reflexes. However, there are two major shortcomings in the scoring system suggested by Spence et al. First, certain deficits commonly or solely encountered in stroke patients are omitted, e.g., hemianopia or pure sensory deficits. Secondly, the attempt to combine acute neurological scoring with later outcome (activities of daily living) is inappropriate (table 1). The factors that determine mortality and neurological status in stroke patients are omitted, e.g., hemianopia or pure sensory deficits. Our comments are encouraged by the increasing cooperation between clinicians and methodologists evident in current publications.

To the Editor:

The term "rebound hypercoagulability" is a confusing one because it begs a question. It implies that changes in hemostatic mechanisms are causally related to further episodes of thromboembolism.

There is no clear evidence that blood changes leading to thrombosis occur in patients after anticoagulant therapy is stopped. All of the observations made in the references cited by Dr. Hart, et al., can be explained on the basis of persistent risk factors (that lead to the original thromboembolic episode) which are unmasked when the patient is no longer protected by anticoagulant therapy.

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TABLE 1 The Toronto Stroke Scale

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Alert</td>
</tr>
<tr>
<td>1</td>
<td>Drowsy</td>
</tr>
<tr>
<td>2</td>
<td>Stupor</td>
</tr>
<tr>
<td>3</td>
<td>Light coma</td>
</tr>
<tr>
<td>4</td>
<td>Deep coma</td>
</tr>
</tbody>
</table>

TABLE 2. Toronto Stroke Scale Related to Clinical Bedside Assessment

<table>
<thead>
<tr>
<th>Stroke severity (number of cases)</th>
<th>Mean score</th>
<th>Stand. dev.</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild (11)</td>
<td>24</td>
<td>18</td>
<td>1-50</td>
</tr>
<tr>
<td>Moderate (24)</td>
<td>72</td>
<td>24</td>
<td>11-102</td>
</tr>
<tr>
<td>Severe (7)</td>
<td>114</td>
<td>33</td>
<td>75-155</td>
</tr>
</tbody>
</table>
the acute phase are quite different from those relating to placement of patients, when motivation, type of rehabilitation or home situation play a major role. Also, the longer the time from treatment that outcome is tested the weaker and more suspect the association becomes and the greater the number of patients needed to make such an association.

We have devised a clinical scoring system for acute stroke patients using the systematic observations from a computerized data base. Results so far indicate that this system accurately reflects stroke severity. Using the initial scores of 42 consecutive cases with completed strokes admitted to the Unit, we were able to relate three grades of clinical severity to this stroke scale which was evaluated separately by different observers. We found a linear relationship (Spearman correlation coefficient, R value = 0.87) (table 2 and fig.).

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To the Editor:
I am somewhat puzzled by the above letter, as it seems to be completely in agreement with what was said in our paper regarding neurological scoring systems. What we said was that it was inappropriate to use the same scoring system to measure the initial severity, as to measure outcomes. For this reason, an example of a scoring system that could be used to determine initial severity was given in table 4, and a separate group of scoring systems that might be used to measure outcomes were given as examples in appendix A.

The initial severity score which we proposed was essentially based on the experience of Oxbury et al., who found that the best predictors of acute stroke outcome were level of consciousness, severity of hemiplegia, and the presence or absence of a gaze palsy or deviation. They assessed a number of other factors which did not contribute in a major way to the prediction of acute outcome, including a number of the traditional elements of a neurological examination such as sensory dysfunction.

I applaud the efforts of Drs. Norris and Hachinski to develop reliable scoring systems, and to test them. Such scoring systems as the one they have put forward in their letter will do much to improve and standardize clinical trials of stroke. I would be concerned about the inclusion of aphasia as a heavily weighted item in their scoring system, since aphasia reflects to a great extent the location rather than the degree of the brain injury. Although the scale they describe would be suitable for prognostic stratification on the basis of severity, I do not believe it would be suitable for measuring outcomes. The success of stroke treatment depends more on whether the patient is able to return home to useful function, than on the results of his neurological examination.

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Reference
Comment on "Study design of stroke treatments".
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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/13/4/527.citation