Data (Serum Values) From 20 Months of Follow-up of an 85-Year-Old Female Patient With Hypogonadotrophic Hypogonadism

<table>
<thead>
<tr>
<th>Date</th>
<th>T&lt;sub&gt;4&lt;/sub&gt;, µg/dL</th>
<th>TSH, µU/L</th>
<th>LH, mIU/mL</th>
<th>FSH, mIU/mL</th>
<th>Estradiol, pg/mL</th>
<th>PRL, µU/L</th>
<th>Peak Cortisol, µg/dL</th>
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</thead>
<tbody>
<tr>
<td>8/12/91</td>
<td>1.01</td>
<td>29.4</td>
<td>...</td>
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<tr>
<td>9/12/91*</td>
<td>3.59</td>
<td>12.3</td>
<td>5.8</td>
<td>22.1</td>
<td>&lt;24.5</td>
<td>174</td>
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</tr>
<tr>
<td>10/30/91†</td>
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<tr>
<td>1/2/92‡</td>
<td>5.07</td>
<td>9.6</td>
<td>7.6</td>
<td>22.0</td>
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<td>2/3/92</td>
<td>6.86</td>
<td>2.3</td>
<td>8.3</td>
<td>21.7</td>
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<tr>
<td>3/13/92</td>
<td>7.02</td>
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<td>9.6</td>
<td>24.8</td>
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<tr>
<td>7/11/92</td>
<td>8.03</td>
<td>3.7</td>
<td>11.4</td>
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<td>&lt;24.5</td>
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<td>3.37</td>
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<td>238.31</td>
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<td>2.99</td>
<td>8.82</td>
<td>26.26</td>
<td>19.07</td>
<td>&lt;400</td>
<td>...</td>
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</tbody>
</table>

Reference range: 3.9-11.7, 0.4-3.8, 10.8-61.4, 35-151, 0.4-3.8

The stroke occurred in March 1991.
T<sub>4</sub> indicates thyroxine; TSH, thyroid-stimulating hormone; LH, luteinizing hormone; FSH, Follicle stimulating hormone; and PRL, prolactin.

*Patient was put on T<sub>4</sub> replacement therapy.
†Short synacthen test was performed.
‡Thyroid antibodies were absent.
§LH-releasing hormone test was performed.

Response

Dr Jolobe’s report of an 85-year-old woman with a history of stroke, primary hypothyroidism, and low gonadotropin levels appears to conform to the findings we described in a study of postmenopausal women with stroke.1 The presence of primary hypothyroidism in his patient is in contrast to findings in our group, in which thyroid function was normal. The fact that hypogonadotropic hypogonadism persisted after adequate replacement with thyroid hormone suggests that the occurrence of the two endocrine perturbations together, in this case, was coincidental.

The result of dynamic testing with gonadotropin-releasing hormone demonstrated the functional status of the patient’s pituitary gonadotrophs, suggesting that her lesion was at the hypothalamic level or higher, possibly an outcome of ischemic injury. We found that infarcts involving the basal ganglia, particularly the caudate nucleus, were associated with hypogonadotropic hypogonadism in postmenopausal women.1 As corroboration, it would have been interesting to learn whether Dr Jolobe’s patient had an injury in this area.

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Reference


Microembolism and Hemodynamic Changes in the Brain During Carotid Endarterectomy

To the Editor:

We were interested to read the article by Jansen and colleagues1 about the association of postoperative brain infarcts (assessed by magnetic resonance imaging [MRI]) with intraoperative episodes of embolization detected by transcranial Doppler (TCD) monitoring during carotid endarterectomy (CEA). We recently completed a similar intraoperative TCD monitoring study of 100 consecutive patients undergoing CEA. All patients had preoperative and postoperative computed tomography brain scans, neurological examination, and psychometric testing with a battery of Wechsler cognitive function tests.2 In addition, the last 50 consecutive patients also had MRI brain scans.

One of our main findings was that patients experiencing more than 10 particulate emboli during carotid dissection had a significant decrease in postoperative cognitive function. This would appear to support the conclusions of Jansen et al1 that emboli occurring during this phase are important and may have clinically significant consequences. However, we were unable to detect any new infarcts associated with these emboli on CT or MRI.3

Unlike Jansen et al,1 we believe there is evidence that it is possible to differentiate between the main categories of emboli (air and particulate) using TCD signal criteria under certain operative conditions.3 Differentiation is not totally specific with...
fast Fourier spectral analysis, but greater reliability has been achieved experimentally using the Wigner method of spectral analysis. Nevertheless, fast Fourier analysis does allow a differentiation that is practically applicable, and appreciation of this enabled us to diagnose three cases of early postoperative carotid thrombosis based on the detection of persistent particulate embolization in the immediate postoperative period. TCD detection of these emboli made possible an early diagnosis of this condition before the development of neurological signs and enabled early operative intervention. This minimized the neurological deficit in two patients and averted deficit in the third, and we believe that this represents an important clinical application of TCD monitoring during CEA. In addition, we were able to determine that the majority of emboli occurring during other stages of the operation were predominantly characteristic of air emboli and not associated with postoperative neurological deficits.

In conclusion, we agree with Jansen et al that intraoperative TCD monitoring during CEA is valuable in detecting conditions of inadequate collateral cerebral blood flow and significant episodes of intraoperative embolization. However, we contend that appreciation of the character of the emboli and continuing TCD monitoring into the early postoperative period may be of significant benefit in reducing perioperative mortality and morbidity associated with the operation.

References

Role of Transcranial Doppler Sonography in the Differentiation of Multi-infarct and Alzheimer-Type Dementia
To the Editor:
In their interesting article, Ries et al concluded that the effective pulsatility range is a noninvasive additional criterion in the differential diagnosis of dementia. However, I have doubts that the methodology used in the article was adequate for this differentiation; there are physiological variables affecting transcranial Doppler (TCD) velocities that were not taken into consideration in the statistical analysis.

According to Hagen-Poiseuille's law, blood viscosity has an important influence on cerebral vasomotion and on hemodynamic changes in the proximal part of the intracranial supplying vessels. If all other factors remain constant, blood flow is inversely proportional to viscosity, and blood viscosity of which hematocrit is the most important factor, is a major determinant of blood velocity. Brass et al have reported an inverse correlation between hematocrit and TCD velocity.

The principal metabolic factors that affect cerebral blood flow and that may be reflected in TCD measurements are PaO2 and PaCO2. Cerebral blood flow does not begin to rise because of hypoxia until
Microembolism and hemodynamic changes in the brain during carotid endarterectomy.
M E Gaunt, J L Smith, P R Bell, P J Martin and A R Naylor

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