### 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>T4, µg/dL</th>
<th>TSH, µIU/L</th>
<th>LH, mIU/mL</th>
<th>FSH, mIU/mL</th>
<th>Estradiol, pg/mL</th>
<th>PRL, µIU/L</th>
<th>Peak Cortisol, µg/dL</th>
</tr>
</thead>
<tbody>
<tr>
<td>8/12/91</td>
<td>1.01</td>
<td>29.4</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<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>
<td>36.8</td>
</tr>
<tr>
<td>10/30/91†</td>
<td>...</td>
<td>...</td>
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<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<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>...</td>
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<tr>
<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>
<td>3.2</td>
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<td>24.8</td>
<td>...</td>
<td>...</td>
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</tr>
<tr>
<td>7/11/92</td>
<td>8.03</td>
<td>3.7</td>
<td>11.4</td>
<td>29.93</td>
<td>&lt;24.5</td>
<td>229</td>
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</tr>
<tr>
<td>7/28/92§</td>
<td>...</td>
<td>...</td>
<td>10.56</td>
<td>24.68</td>
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<tr>
<td>10/16/92</td>
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<td>3.37</td>
<td>8.70</td>
<td>31.49</td>
<td>&lt;24.5</td>
<td>238.31</td>
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<td>4/24/92</td>
<td>6.46</td>
<td>2.99</td>
<td>8.82</td>
<td>26.26</td>
<td>19.07</td>
<td>&lt;400</td>
<td>...</td>
</tr>
</tbody>
</table>

Reference range: 3.9-11.7 for T4, 0.4-3.8 for TSH, 10.8-61.4 for LH, 5.8 for FSH, 22.1 for Estradiol, 24.68 for PRL, and 30.62 for Peak Cortisol.

The stroke occurred in March 1991.

T4 indicates thyroxine; TSH, thyroid-stimulating hormone; LH, luteinizing hormone; FSH, Follicle stimulating hormone; and PRL, prolactin.

*Patient was put on T4 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. 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. As corroboration, it would have been interesting to learn whether Dr Jolobe’s patient had an injury in this area.

### Microembolism and Hemodynamic Changes in the Brain During Carotid Endarterectomy

To the Editor:

We were interested to read the article by Jansen and colleagues 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. 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 al. 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. In addition, the last 50 consecutive patients also had MRI brain scans.

Unlike Jansen et al, 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. Differentiation is not totally specific with
fast Fourier spectral analysis, but greater reliability has be
achieved experimentally using the Wigner method of spectral analysis. Nevertheless, fast Fourier analysis does allow a differentia
tion 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.

M.E. Gaunt, FRCS P.R.F. Bell, MD, FRCS Department of Surgery P.J. Martin, MRCP Department of Neurology Leicester Royal Infirmary Leicester, England A.R. Naylor, MD, FRCS Department of Surgery Aberdeen Royal Infirmary Aberdeen, Scotland

In our opinion, the analysis of embolic events is still hindered by inadequate signal processing techniques. The stage of the operation can suggest the nature of microemboli, air bubbles being extremely unlikely when the artery has not yet been opened. Because the fundamental paradox of the Fourier transform technique is that the best temporal resolution that can be achieved is inversely propor
tional to the frequency resolution, the Wigner method seems promis
ing, but the technique is still under study. In our hospital we were not equipped to discriminate between different microemboli.

Persistent particulate embolization in the early postoperative pe
riod is an important observation. In some cases this phenomenon heralds an ischemic complication during cardiopulmonary bypass immediately after the operation should therefore prompt reassessment of the operated artery, although an increase in the number of microemboli in the first postoperative hours, without clinical consequences, has been described. For this reason, postoperative embolization should be interpreted carefully to avoid unnecessary reexploration. We agree with the statement made by Gaunt et al in their letter that postoperative TCD monitoring should be undertaken to study the cause of morbidity in the first days after 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 al1 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 differen
tiation; there are physiological variables affecting transcranial Doppler (TCD) velocities that were not taken into consideration in the statistical analysis.

According to Hagen-Poiseille's law, blood viscosity has an important influence on cerebral vasomotion2 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 al3 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 PO2 and PCO2. Cerebral blood flow does not begin to rise because of hypoxia until

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C. Jansen, MD, PhD R.G.A. Ackerstaff, MD, PhD Department of Clinical Neurophysiology St Antoniushospital Nieuwegein, The Netherlands

References
5. Van Zuilen EV, Moll FL, Vermeulen FEE, Mauser HW, van Gijn J, Ackerstaff RGA. Detection of cerebral microemboli by means of transcranial Doppler monitoring before and after carotid endarter
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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