The Effect of Intracardial Pacemaker Therapy on Cerebral Blood Flow and Electroencephalogram in Patients with Complete Atrioventricular Block

By I. A. Sulg, M.D., S. Cronqvist, M.D., H. Schüller, M.D., and D. H. Ingvar, M.D.

SUMMARY

In seven patients with complete A-V block of long duration (five suffering from Adams-Stokes attacks), the cerebral blood flow was found to be significantly decreased and there was also moderate slowing of the electroencephalogram. Artificial pacing increased the heart rate from 39 to 72/min. Postoperative studies showed that in six of the seven cases the cerebral blood flow had increased and that the mean spectral frequency of the EEG had increased. In two patients follow-up studies suggest that the normalization of the EEG and cerebral blood flow may take some time to develop fully. The changes in EEG and cerebral blood flow paralleled the clinical improvement which accompanied the pacemaker therapy in all the patients.

Additional Indexing Words:
Adams-Stokes attacks
Hemisphere blood flow
Heart rate
Blood pressure
EEG

Several authors have demonstrated that cardiac arrhythmia may have a profound effect on the systemic circulation as a whole.1-4 Disturbances of the heart rate and heart rhythm, due to atrioventricular block (A-V block) for example may also induce cerebral symptoms such as syncopal-convulsive episodes, as described by Adams and Stokes.5,6

The treatment of an irregular heart rhythm by implantation of an artificial pacemaker has proved successful in relieving such syncopal symptoms.5-9

The purpose of the present study was to measure the effects of pacemaker therapy on the cerebral blood flow and EEG in patients with A-V block.

Methods

Seven male patients with complete A-V block and Adams-Stokes syndrome were studied with measurements of regional cerebral blood flow (rCBF) and EEG before and after implantation of a unipolar intracardiac pacemaker. The ages of the patients varied between 60 and 77 years (mean, 69 years). The interval between the operation and the second examination varied in six cases between 2 and 17 days. In one patient (H. S.) the EEG and rCBF were recorded immediately before and within a few minutes following the start of the artificial pacemaker. This patient also underwent a third study about 6 months later.

The duration of the A-V block prior to the determination of first cerebral blood flow and EEG examination varied between 6 months and 9 years (mean, 3 years). All but two patients had Adams-Stokes attacks. Signs of more pronounced

---

This study was sponsored by grants to Dr. Ingvar from the Swedish Medical Research Council (project number B67-21X-84-03) and from the Wallenberg Foundation, Stockholm. Dr. Sulg was aided by grants from the Medical Faculty of Lund. Grants to Dr. Cronqvist from "Riksföreningen mot Cancer" also aided the study.

*Type EM 139, Elema Schöntander, Stockholm, Sweden.
Table 1

Clinical Findings in Seven Patients with Total Heart Block Before and After Implantation of an Artificial Pacemaker

<table>
<thead>
<tr>
<th>Case</th>
<th>Age (yr)</th>
<th>A-V block for (yr)</th>
<th>Time of examination</th>
<th>Adverse-</th>
<th>Cardiac</th>
<th>Orthostatic</th>
<th>Heart</th>
<th>Heart</th>
<th>Blood pressure (external measure) (mm Hg)</th>
<th>Arterial</th>
<th>Hemoglobin (g%)</th>
<th>Rating of</th>
<th>General</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Stokes</td>
<td>decompen-</td>
<td>symptoms</td>
<td>volume (ml/m²)</td>
<td>rate</td>
<td>(beats/min)</td>
<td>Syst./Diast.</td>
<td>Mean</td>
<td>PO₂ (mm Hg)</td>
<td>improvement</td>
</tr>
<tr>
<td>S.T.</td>
<td>77</td>
<td>½</td>
<td>Preop.</td>
<td>+</td>
<td>—</td>
<td>—</td>
<td>480</td>
<td>42</td>
<td>270/100</td>
<td>164</td>
<td>42.8</td>
<td>12.8</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>P.O.</td>
<td>0</td>
<td>—</td>
<td>—</td>
<td>Not ex.</td>
<td>76</td>
<td>160/90</td>
<td>120</td>
<td>50.3</td>
<td>12.0</td>
<td></td>
</tr>
<tr>
<td>H.P.</td>
<td>74</td>
<td>9</td>
<td>Preop.</td>
<td>+</td>
<td>+</td>
<td>—</td>
<td>Not ex.</td>
<td>34</td>
<td>210/70</td>
<td>121</td>
<td>28.0</td>
<td>12.5</td>
<td>++</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>P.O.</td>
<td>0</td>
<td>—</td>
<td>—</td>
<td>Not ex.</td>
<td>72</td>
<td>125/85</td>
<td>103</td>
<td>28.0</td>
<td>12.5</td>
<td>++</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>P.O.</td>
<td>0</td>
<td>—</td>
<td>—</td>
<td>460</td>
<td>72</td>
<td>140/80</td>
<td>106</td>
<td>37.0</td>
<td>8.7</td>
<td>+</td>
</tr>
<tr>
<td>T.D.</td>
<td>71</td>
<td>1</td>
<td>Preop.</td>
<td>0</td>
<td>++</td>
<td>—</td>
<td>705</td>
<td>30</td>
<td>140/70</td>
<td>99</td>
<td>34.5</td>
<td>13.8</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>P.O.</td>
<td>0</td>
<td>+</td>
<td>—</td>
<td>780</td>
<td>72</td>
<td>150/95</td>
<td>119</td>
<td>36.5</td>
<td>13.9</td>
<td>+</td>
</tr>
<tr>
<td>H.H.</td>
<td>64</td>
<td>2</td>
<td>Preop.</td>
<td>0</td>
<td>+</td>
<td>+</td>
<td>635</td>
<td>48</td>
<td>180/85</td>
<td>124</td>
<td>36.0</td>
<td>14.2</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>P.O.</td>
<td>0</td>
<td>—</td>
<td>—</td>
<td>580</td>
<td>72</td>
<td>160/100</td>
<td>126</td>
<td>36.0</td>
<td>13.9</td>
<td>++</td>
</tr>
<tr>
<td>S.J.</td>
<td>61</td>
<td>½</td>
<td>Preop.</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>560</td>
<td>40</td>
<td>160/85</td>
<td>117</td>
<td>38.0</td>
<td>14.6</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>P.O.</td>
<td>0</td>
<td>—</td>
<td>—</td>
<td>520</td>
<td>72</td>
<td>145/70</td>
<td>101</td>
<td>37.0</td>
<td>16.0</td>
<td>+</td>
</tr>
<tr>
<td>H.S.</td>
<td>60</td>
<td>3</td>
<td>Preop.</td>
<td>+</td>
<td>—</td>
<td>+</td>
<td>610</td>
<td>35</td>
<td>130/80</td>
<td>102</td>
<td>37.0</td>
<td>13.5</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>P.O.</td>
<td>0</td>
<td>—</td>
<td>—</td>
<td>440</td>
<td>72</td>
<td>145/85</td>
<td>111</td>
<td>39.0</td>
<td>Not ex.</td>
<td>+++++</td>
</tr>
<tr>
<td>Mean</td>
<td>60</td>
<td>3</td>
<td>Preop.</td>
<td>5</td>
<td>5</td>
<td>4</td>
<td>out of</td>
<td>7</td>
<td>out of</td>
<td>(±101)</td>
<td>(±6.2)</td>
<td>(±11)</td>
<td>(±21)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>P.O.</td>
<td>7</td>
<td>7</td>
<td>7</td>
<td>out of</td>
<td>580</td>
<td>72</td>
<td>146/86</td>
<td>112</td>
<td>37.7</td>
<td>12.8</td>
</tr>
</tbody>
</table>

Abbreviations: Preop. = before surgical installation of an artificial pacemaker; P.O. = after its installation; not ex. = not examined; + = slight; ++ = substantial; +++ = pronounced.
cardiac decompensation were noted in only one patient (T. D.) who also had dyspnea, decline edema, and an enlargement of the heart. In four other patients the cardiac decompensation was only slight, the main symptom being dyspnea on strain. In five cases the heart volume was measured preoperatively and postoperatively (table 1). No significant difference was found. The heart rate varied preoperatively between 30 and 48/min (mean, 39/min). With the pacemaker the heart rate was fixed to a frequency of about 72/min in all cases. Blood pressure was measured by the Riva-Rocci method. From systolic and diastolic measurements a geometric mean pressure was calculated, which, according to Best and Taylor, is a better expression of the average arterial pressure than the arithmetic mean of systolic-diastolic values.

None of the patients showed any focal neurological signs. However, in all of them a certain reduction of physical well-being and mental capacity was noted. The patients felt a constant fatigue and had some difficulties in memorizing and in concentration. There was no evidence of pronounced dementia or signs of psychosis.

Essential clinical findings in the seven patients have been summarized in table 1.

Regional Cerebral Blood Flow (rCBF)

This was measured according to the isotope clearance method introduced by Lassen and associates. The radioactive inert gas 133Xenon, dissolved in saline, is injected into the internal carotid artery. The uptake of the isotope and its clearance from the labeled hemisphere were measured with multiple extracranial scintillation detectors. From the clearance curves, the two rCBF values were calculated: (1) the compartmental rCBF and (2) an average flow according to the so-called 10-minute area method.

To calculate the compartmental rCBF each clearance curve was assumed to consist of two monoexponential functions, representing a fast and a slow type of blood flow in the gray and the white matter, respectively. The flow within these two compartments was calculated for each detector field in terms of ml of blood/100 g of brain tissue/min.

Correction of rCBF for PaCO2

The raw, directly measured rCBF values were recalculated to the values one would expect if the arterial P CO2 (PaCO2) had been 40 mm Hg. This correction was performed according to Reivich and Alexander and associates using the formula:

\[ rCBF_{corr, \text{PaCO2}} = \frac{rCBF}{1 + 0.025(PaCO2 - 40)} \]

In healthy controls the "noncompartmental" mean hemisphere rCBF (mean values from all detectors) was found to be 51.1, sd ± 2.7 ml/100 g/min (not corrected for arterial P CO2). This value is close to the total average CBF value obtained in healthy controls with the Kety nitrous oxide-inhalation method: 53.8, sd ± 12.0 ml/100 g/min. The EEG studies were usually made within 2 hours before or after the rCBF measurements.

Results

One patient (B. S.) showed signs of severe agitation during the first examination, and samples for arterial P CO2 determinations were not obtained. Hence this case, although presented in table 1 (clinical results), has been excluded from the statistical analysis. In table 2 the mean hemisphere rCBF values are shown. In all six patients the flow was found to be significantly below normal values at the first examination with a mean for the whole group of 40.1 ± 3.9 ml/100 g/min, which represents a 21.5% decrease from the normal 51.1 ml/100 g/min. The mean hemisphere gray rCBF for the patients was found to be 60.4 ± 5.1 ml/100 g/min, which was also a significant decrease of

*This normal value is slightly higher than the one given in an earlier paper: 49.8 ± 5.4 ml/100 g/min. This is due to the fact that in the present series it was essential to choose a reference material, in which the EEG was normal. For this reason one subject of the original series (T.P.) was excluded. For the same reason the value for mean hemisphere gray rCBF in a group of six normals (with normal EEG) was recalculated to 82.1 ± 5.8 ml/100 g/min.
Table 2

EEG Index and Mean Hemisphere Blood Flow (Calculated from Multiple Regional Measurements) in Raw and Corrected for PaCO₂ Values, Before and After Implantation of an Artificial Pacemaker

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>S.T.</td>
<td>77</td>
<td>Preop.</td>
<td>Abnormal +</td>
<td>9.3</td>
<td>EEG</td>
<td>60.4</td>
<td>55.9</td>
<td>21.3</td>
<td>20.3</td>
<td>40.1</td>
<td>38.2</td>
<td>43.4</td>
<td>40.7</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>P.O.</td>
<td>Abnormal ++</td>
<td>8.5</td>
<td></td>
<td>62.4</td>
<td>45.2</td>
<td>20.4</td>
<td>14.8</td>
<td>41.7</td>
<td>30.2</td>
<td>45.3</td>
<td>31.4</td>
<td></td>
</tr>
<tr>
<td>H.P.</td>
<td>74</td>
<td>Preop.</td>
<td>Abnormal +</td>
<td>9.6</td>
<td></td>
<td>61.9</td>
<td>81.4</td>
<td>15.3</td>
<td>20.1</td>
<td>35.9</td>
<td>47.1</td>
<td>38.9</td>
<td>51.1</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>P.O.</td>
<td>Normal</td>
<td>9.8</td>
<td></td>
<td>74.8</td>
<td>100.0</td>
<td>20.1</td>
<td>26.8</td>
<td>44.9</td>
<td>59.8</td>
<td>46.3</td>
<td>61.8</td>
<td></td>
</tr>
<tr>
<td>T.D.</td>
<td>71</td>
<td>Preop.</td>
<td>Normal</td>
<td>13.0</td>
<td></td>
<td>55.5</td>
<td>62.9</td>
<td>15.1</td>
<td>17.1</td>
<td>33.8</td>
<td>38.2</td>
<td>38.3</td>
<td>40.9</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>P.O.</td>
<td>Normal</td>
<td>14.1</td>
<td></td>
<td>58.6</td>
<td>63.2</td>
<td>17.9</td>
<td>19.2</td>
<td>36.1</td>
<td>39.0</td>
<td>38.3</td>
<td>41.3</td>
<td></td>
</tr>
<tr>
<td>H.H.</td>
<td>64</td>
<td>Preop.</td>
<td>Abnormal +</td>
<td>7.6</td>
<td></td>
<td>54.2</td>
<td>60.0</td>
<td>17.9</td>
<td>19.9</td>
<td>33.3</td>
<td>36.9</td>
<td>33.6</td>
<td>37.2</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>P.O.</td>
<td>Normal</td>
<td>10.1</td>
<td></td>
<td>64.7</td>
<td>73.4</td>
<td>18.8</td>
<td>21.3</td>
<td>38.4</td>
<td>43.3</td>
<td>38.1</td>
<td>43.2</td>
<td></td>
</tr>
<tr>
<td>S.J.</td>
<td>61</td>
<td>Preop.</td>
<td>Abnormal +</td>
<td>9.6</td>
<td></td>
<td>61.8</td>
<td>65.1</td>
<td>17.9</td>
<td>18.7</td>
<td>39.8</td>
<td>41.6</td>
<td>42.4</td>
<td>44.7</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>P.O.</td>
<td>Normal</td>
<td>11.2</td>
<td></td>
<td>65.3</td>
<td>72.3</td>
<td>16.5</td>
<td>18.3</td>
<td>40.3</td>
<td>44.5</td>
<td>46.1</td>
<td>51.0</td>
<td></td>
</tr>
<tr>
<td>H.S.</td>
<td>60</td>
<td>Preop.</td>
<td>Normal</td>
<td>8.6</td>
<td></td>
<td>68.3</td>
<td>75.6</td>
<td>22.6</td>
<td>25.0</td>
<td>42.9</td>
<td>47.5</td>
<td>43.7</td>
<td>42.8</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>P.O.</td>
<td>Normal</td>
<td>9.0</td>
<td></td>
<td>69.9</td>
<td>75.5</td>
<td>20.6</td>
<td>22.3</td>
<td>47.5</td>
<td>51.4</td>
<td>42.8</td>
<td>52.8</td>
<td></td>
</tr>
</tbody>
</table>

Mean +

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Preop.</td>
<td>4 abnormal/6</td>
<td>EEG</td>
<td>60.4</td>
<td>66.8</td>
<td>18.4</td>
<td>29.2</td>
<td>37.6</td>
<td>41.6</td>
<td>40.1</td>
<td>42.9</td>
</tr>
<tr>
<td>P.O.</td>
<td>1 abnormal/6</td>
<td></td>
<td>66.0</td>
<td>71.6</td>
<td>19.1</td>
<td>29.5</td>
<td>41.5</td>
<td>44.7</td>
<td>43.8</td>
<td>46.9</td>
</tr>
<tr>
<td>Mean</td>
<td>+9.4</td>
<td></td>
<td>6 +3.5</td>
<td>19.7</td>
<td>29.6</td>
<td>43.9</td>
<td>46.7</td>
<td>49.9</td>
<td>48.8</td>
<td>52.9</td>
</tr>
</tbody>
</table>

% difference of Preop./P.O.
Significance of difference tested by
"Student's" t-distribution P in %

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Preop.</td>
<td>9.8</td>
<td>82.1</td>
<td>82.7</td>
<td>21.5</td>
<td>21.7</td>
<td>50.9</td>
<td>51.3</td>
<td>51.1</td>
<td>51.4</td>
</tr>
<tr>
<td>P.O.</td>
<td>0.8</td>
<td>5.8</td>
<td>13.1</td>
<td>16.6</td>
<td>2.4</td>
<td>3.1</td>
<td>6.0</td>
<td>2.7</td>
<td>5.9</td>
</tr>
</tbody>
</table>

Abbreviations: Corr. f. PaCO₂ = corrected for arterial PaCO₂ at 40 mm Hg; s = Significant; ns = Not significant; other abbreviations same as in table 1.
about 25.9% below the normal value (82.1 ± 5.8).

On re-examination only a few minutes after the onset of artificial pacing, one patient (H. S.) was found to have a slight decrease of gray matter rCBF (from 68.3 to 57.7 ml/100 g/min), although the cardiac output increased from 3.6 L/min (without pacemaker) to 5.0 L/min (with pacemaker). A third rCBF study in the same patient 6 months later showed a significant increase above the preoperative blood flow values (from 57.7 to 69.9 ml/100 g/min). All six patients showed an increase in hemisphere rCBF averaging 5.6 ml for gray rCBF (+ 9.3%), 0.7 ml for white rCBF (+ 3.8%) and 3.7 ml for the average hemisphere rCBF (+ 9.2%). These mean values refer to the raw rCBF data, not corrected for PaCO2. This does not imply that the primary values from rCBF measurements are not correct. It is not possible, by using regional CBF measurements, to subject the blood of the same regions to gas analysis. As the regional PaCO2 cannot be measured, and as regional PaCO2 alterations may be conceived to occur inde-

pendently of the PaCO2 measured in a large artery,20 there does not exist any correction factor, which can be representative to some circumscribed part of the brain. If, however, the sum of regional CBF values from the same hemisphere are used in calculation of mean hemispherical flow, and if there is no detectable focal ischemia in the hemisphere examined, then it seems probable that the PaCO2 in the carotid artery (as it was measured in the present study) may be taken as representative also for blood in intracerebral vessels. In table 2 the measured rCBF data were given both as primary values and as recalculated values (corrected for PaCO2). The PaCO2 values were given in table 1.

Figure 1
Spectral distribution of electroencephalographic activity (accumulated period time as percentage of the length of analyzed EEG) in frequency classes 1 to 20 c/sec: Shaded area indicates healthy controls; dotted line, patients with A-V block before implantation of pacemaker; heavy line, patients with A-V block after implantation of pacemaker.

Circulation, Volume XXXIX, April 1969

Figure 2
Effect of cardiac pacemaker therapy on the cerebral blood flow and the frequency content in the electroencephalogram. Regional cerebral blood flow calculated as the noncompartmental hemisphere flow, corrected for arterial PaCO2 (left column), and EEG index (mean spectral frequency) in patients with complete A-V block preoperatively and postoperatively (right column). The EEG index from the third examination of S.J., which was higher than the preoperative value, is not included here. (See page 54.)
Analysis of the EEG curves before and after the onset of pacemaker therapy showed a shift in the frequency histogram toward the normal pattern in six cases, that is a decrease of the slow wave content and an increase in fast activity (fig. 1). An increase of the EEG index was noted in the postoperative stage in four cases and a decrease in two (fig. 2). One of these cases (S. J.), however, showed in the preoperative record a regional abnormality, which disappeared in the postoperative record. In the third EEG about 2½ years later the frequency index had risen above the preoperative level, that is from 9.6 to 11.2. In table 2 the results of the EEG analyses and the rCBF measurements are shown together. Thus the postoperative increase of the EEG index was shown in all patients but one. The increase of the group mean value was, however, statistically not significant. If the total activity time through all frequency classes was calculated (= the area of the histogram) and the difference between preoperative and postoperative spectra was tested with a χ² test, then the result χ² = 71 (df* = 18) turned out to be highly significant (P < 0.001). The difference was essentially, but not exclusively, due to the increase of EEG activity within the 10 cycles/second frequency class. The same test carried out for the same patients individually (to test the homogeneity in preoperative and postoperative spectra) gave a total χ² = 300 (df = 108), also a highly significant value.

The correlation between EEG index and rCBF (calculated for differences between preoperative and postoperative values) showed higher coefficients (maximum r = 0.57) for PaCO₂-corrected rCBF values than for raw values (maximum r = 0.34). These values were, however, beyond the statistical significance.

The correlation between the gray flow of the rCBF and EEG index showed to be closer (maximum r = 0.57) than the correlation between noncompartmental rCBF, calculated as the “10-minute area” value (maximum, r = 0.46). There was no correlation between EEG index and white rCBF raw values, but a trend of positive correlation to PaCO₂-corrected values.

Clinical Results

The clinical results of the pacemaker therapy were evaluated objectively and subjectively as seen in table 1. The heart rate increased by means of artificial pacing from a mean of 39 to 72/min. In one case (H. S.) the abnormally enlarged heart volume decreased significantly following pacemaker therapy (610 → 440 ml/m² of body surface), but the group means did not show any significant difference. In one case the cardiac minute volume was measured immediately before and after the starting of the pacemaker. The cardiac output had increased from 3.5 to 5.0 L/min. The average systolic blood pressure in the group and corresponding standard deviation decreased from 182 ± 47 mm Hg preoperatively to 146 ± 12 mm Hg with pacemaker. The diastolic blood pressure did not change significantly.

The group mean values of PaCO₂ and hemoglobin content were essentially identical before and after implantation of the pacemaker. Only one patient (S. J.) had a significant increase in hemoglobin (14.6 to 16 g%) postoperatively. This patient showed a delayed increase of the EEG index postoperatively.

It seems to be a common experience that the patients with total heart block, when treated with artificial pacing, show progressive somatic as well as mental improvement. This was evident in the present material. The improvement was pronounced in one patient, in three it was substantial, and in three slight. All patients had subjective relief and an increasing sense of well-being after institution of pacemaker therapy. Syncopal-convulsive symptoms did not occur during artificial pacing.

Discussion

The main finding of this study is that patients with complete A-V block appear to
suffer from a decrease of cerebral blood flow and a slight slowing of the EEG, and that these two functions tended to be improved by pacemaker therapy. Parallel to this trend general clinical and subjective improvement of all the patients was observed.

The first point to be discussed is the possible role of age in this group of elderly patients. While EEG changes in old age have been reported in a number of investigations, no report on the regional cerebral blood flow in patients treated with complete A-V block has been published. 

Obrist and Henry in 1963, correlated EEG changes with cerebral blood flow studies and oxygen consumption in healthy elderly volunteers and in 20 aged psychiatric patients. They found that the values of average hemisphere cerebral blood flow in aged controls did not differ significantly from those of younger adults. The psychiatric patients, however, had a lower cerebral blood flow and also a lower metabolic rate for oxygen. In another study the same authors showed that the occipital alpha rhythm was significantly slower and the incidence of delta-activity was greater in elderly patients with cardiovascular disease than in normal subjects of the same age. One possibility would be that the cardiovascular abnormality in the present cases had affected other organs of the body, such as the kidneys or the liver and that the brain was affected secondarily by renal or hepatic insufficiency. This explanation does not seem to be a likely one since none of the patients showed signs of renal or hepatic disorder.

A second explanation would be that the preoperative decrease of cerebral blood flow and slowing of EEG could have been due to small "subclinical" ischemic cerebral lesions, possibly suffered during the Adams-Stokes attacks. It is known that focal cerebral lesions may be followed by such a general decrease of the cerebral blood flow. Although this possibility cannot be ruled out completely, it does not seem a likely one, since all patients showed normal neurological findings, and furthermore, since the reduction of CBF and slowing of the EEG were in general found to be reversible.

The third possibility is that hemodynamic factors, such as a low perfusion pressure in the central nervous system, partly created by the cardiac arrhythmia and especially by the very low heart rate, could have caused the diminution of CBF. This condition would then have "exhausted" the normal autoregulatory capacity of the cerebral vascular bed and, hence, caused a slight diminution of the cerebral perfusion. It seems, in fact, possible also that a combination of different causative factors could have been responsible for the development of a chronic encephalopathy in patients with complete A-V block. Frequent Adams-Stokes attacks could have caused prolonged sequences of cerebral anoxia with long-lasting effects on the neuronal metabolism. To this would be added the systemic effects of bradycardia and decreased cardiac output. Following artificial pacing an increase of the heart rate and a considerable improvement in cerebral hemodynamics takes place. At the same time the anoxic attacks are avoided. As a result, there will be a successive improvement in cerebral blood flow, neuronal metabolism, and the EEG. It should be pointed out, however, that in spite of the improvement observed, the rCBF values remained subnormal postoperatively.

In this context emphasis should be given to the case of H. S. who was studied a few minutes after the start of the pacemaker and then re-examined 6 months later. This unique observation indicates that there were no dramatic changes in either the EEG or the cerebral blood flow, when the heart rate was acutely increased at the onset of the pacing. A slight decrease of the cerebral blood flow was in fact shown to take place. At the same time a small drop in the systemic blood pressure was observed. On re-examination 6 months later (table 1), however, a clear-cut increase of cerebral rCBF values was found. This case and the late increase of the EEG-index in S. J. make it probable that a certain time may be needed for the adaptation of the cerebral circulation and metabolism before
the brain achieves the full benefit of pacemaker therapy. The same trend was reflected in the successive improvement of the mental and physical conditions of the patients.

References
The Effect of Intracardial Pacemaker Therapy on Cerebral Blood Flow and Electroencephalogram in Patients with Complete Atrioventricular Block

I. A. SULG, S. CRONQVIST, H. SCHÜLLER and D. H. INGVAR

doi: 10.1161/01.CIR.39.4.487

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1969 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/39/4/487

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/