Studies of the Cerebral Circulation and Metabolism in Congestive Heart Failure

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In a study of the cerebral circulation and metabolism in congestive heart failure, using Kety's nitrous oxide technic, it was found that no significant alterations of the cerebral circulation were present when the quantities measured were compared with values determined for a control group. A decrease in cerebral blood flow and oxygen consumption below values established for normal young individuals was demonstrated to be secondary to the presence of arteriosclerosis and not to cardiac decompensation as had previously been concluded. It was also found that in the series studied the increased venous and cerebrospinal fluid pressures were not of sufficient magnitude to affect the cerebral circulation. The factor of a decreased arterial carbon dioxide tension as a cause for diminished cerebral blood flow in cardiac decompensation was similarly considered and dismissed.

The introduction of the nitrous oxide method for the quantitative measurement of the cerebral circulation in humans has stimulated a renewed interest in the abnormalities of the cerebral hemodynamics and metabolism present in congestive heart failure. The earliest studies pertinent to this problem were the observations of Harrison, who noted that in congestive heart failure the spinal fluid pressure is elevated to approximately the same extent as the venous pressure. It was further found that in such circumstances spinal fluid drainage results in a temporary improvement of orthopnea and dyspnea and a drop in the venous pressure. The implication of this study was that the cerebral circulation was aided by reducing the resistance offered by the increased intracranial pressure with consequent improvement of symptoms attributed to anoxia of the respiratory center.

The first application of the nitrous oxide technic to the actual measurement of the cerebral blood flow in cardiac decompensation resulted in a report that the cerebral blood flow is reduced 39 per cent below the normal. The cerebral metabolism with respect to oxygen and glucose utilization was also found to be significantly reduced. This impairment of the cerebral metabolic rate was considered to result from an inability of the brain to increase its oxygen extraction from the blood in proportion to the drop in blood flow.

Scheinberg inferred that the reduction in cerebral blood flow which he found was related to the 40 per cent reduction in cardiac output found by Stead in a study of a series of patients with a similar degree of cardiac failure. However, the immediate mechanism producing a decreased cerebral blood flow was considered to be an intense cerebrovascular constriction. This was based upon the fact that a mean increase in the cerebrovascular resistance of 100 per cent above normal was found in the patients in congestive heart failure whose cerebral circulations were studied. An explanation for this increased cerebrovascular resistance was, however, wanting for lack of data relevant to the various factors influencing this quantity.

Perhaps the most important single factor in the intrinsic regulation of the cerebrovascular tone is the tension of carbon dioxide (pCO₂) in the blood. An increased arterial blood carbon dioxide tension has been shown to produce...
cerebral vasodilatation with a consequent increase in the cerebral blood flow. Conversely, a decreased arterial blood carbon dioxide tension, such as could occur in the hyperventilation of heart failure, causes cerebral vasconstriction and a decreased cerebral blood flow. It is thus evident that a consideration of the carbon dioxide tension of arterial blood is essential to the understanding of any alteration of the cerebral hemodynamics.

Other physical factors which must also be considered as capable of contributing to an increased cerebrovascular resistance are the venous pressure in the internal jugular vessel and the cerebrospinal fluid pressure. In normal circumstances measurement of these quantities may be neglected. In congestive heart failure both are elevated, and their contributions to the production of increased cerebrovascular resistance must be evaluated. Although neither of these quantities was measured in the first report cited, a more recent publication by Moyer and co-workers mentions that the effect of the increased venous pressure may be considered negligible. These authors confirmed the presence of decreased cerebral blood flow (20 per cent) and an increased cerebrovascular resistance in the presence of congestive heart failure but found cerebral oxygen utilization to be normal. However, neither the blood carbon dioxide tension nor the cerebrospinal fluid pressure was measured in this investigation.

In addition to such physical and chemical changes which may be characteristic of congestive heart failure and thus responsible for the abnormalities of the cerebral hemodynamics and metabolism that have been found, another factor to be considered is the proper selection of controls for the purpose of comparing the parameters measured. In the previous studies cited, the values found for normal individuals were used to assess the changes found in cardiac decompensation. Such a procedure overlooks the possibility that deviations from the normal as observed in the presence of congestive heart failure may be a result of the very vascular abnormalities eventuating in the cardiac decompensation. In other words, the hypertension and arteriosclerosis responsible for the majority of the cases of congestive heart failure studied may have been responsible for the observed abnormalities rather than the heart failure itself being the principal causative factor. In an effort to eliminate this objection, we have used a control group composed of individuals with comparable vascular changes but lacking heart failure.

**Methods**

The cerebral blood flow was measured by the nitrous oxide technic of Kety. The patients were in the fasting state and in the recumbent position.

**Table 1.—Clinical Status, Pulse, and Respirations of Patients with Congestive Heart Failure**

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age</th>
<th>Sex</th>
<th>Diagnosis</th>
<th>Pulse</th>
<th>Resp.</th>
<th>Mental State</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. F.</td>
<td>56</td>
<td>M</td>
<td>ASCVD</td>
<td>90</td>
<td>18</td>
<td>Clear</td>
</tr>
<tr>
<td>M. S.</td>
<td>63</td>
<td>M</td>
<td>CAHD</td>
<td>108</td>
<td>30</td>
<td>Clear</td>
</tr>
<tr>
<td>T. J.</td>
<td>30</td>
<td>M</td>
<td>HHD</td>
<td>108</td>
<td>15</td>
<td>Clear</td>
</tr>
<tr>
<td>A. C.</td>
<td>56</td>
<td>M</td>
<td>ASCVD</td>
<td>72</td>
<td>18</td>
<td>Clear</td>
</tr>
<tr>
<td>E. H.</td>
<td>58</td>
<td>F</td>
<td>CAHD</td>
<td>95</td>
<td>18</td>
<td>Clear</td>
</tr>
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<td>F</td>
<td>HHD</td>
<td>112</td>
<td>21</td>
<td>Clear</td>
</tr>
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<td>M</td>
<td>RHD</td>
<td>72</td>
<td>20</td>
<td>Clear</td>
</tr>
<tr>
<td>G. S.</td>
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<td>F</td>
<td>ASCVD</td>
<td>96</td>
<td>15</td>
<td>Clear</td>
</tr>
<tr>
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<td>M</td>
<td>ASCVD</td>
<td>90</td>
<td>24</td>
<td>Clear</td>
</tr>
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<td>M</td>
<td>ASCVD</td>
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<td>24</td>
<td>Disoriented</td>
</tr>
<tr>
<td>I. M.</td>
<td>54</td>
<td>F</td>
<td>RHD</td>
<td>72</td>
<td>20</td>
<td>Clear</td>
</tr>
<tr>
<td>J. C.</td>
<td>63</td>
<td>F</td>
<td>ASCVD</td>
<td>100</td>
<td>20</td>
<td>Clear</td>
</tr>
<tr>
<td>J. Co.</td>
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<td>M</td>
<td>PHD</td>
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<td>24</td>
<td>Clear</td>
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<td>F</td>
<td>ASCVD</td>
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<td>Clear</td>
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<tr>
<td>B. B.</td>
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<td>F</td>
<td>ASCVD</td>
<td>132</td>
<td>20</td>
<td>Clear</td>
</tr>
</tbody>
</table>

**Abbreviations:** ASCVD—Arteriosclerotic cardiovascular disease; CAHD—Coronary artery heart disease; HHD—Hypertensive heart disease; RHD—Rheumatic heart disease; PHD—Pulmonary heart disease

The jugular venous pressure (JVP) was measured with a spinal fluid manometer, using the level at which the vessel was entered as the reference point. The brachial venous pressure (BVP) was measured in an antecubital tributary with a spinal fluid manometer, the reference level being a plane 5 cm. posterior to the plane of the sternal angle of Louis. The mean arterial blood pressure (MABP) was measured in a femoral artery using a damped mercury manometer. The cerebrospinal fluid pressure (CSFP) was measured in the lumbar region in the supine position, using either a spinal fluid manometer or a Statham strain gage manometer. The reference level used was a plane 3.5 cm. above the mattress on which the patient was lying. The oxygen and carbon dioxide contents of the venous and arterial blood were determined by the method of
Van Slyke, Blood pH was determined anaerobically at room temperature, using a glass electrode. The values were corrected to body temperature by the method of Rosenthal. The carbon dioxide tensions of arterial and venous blood samples were determined by the use of standard nomograms. Blood glucose was determined by the method of Somogyi.

mean arterial blood pressure and the jugular venous pressure, each expressed in mm. Hg.

**Material**

Table 1 presents in outline form a brief summary of the important clinical features of the cases studied. The majority of the patients were in the sixth and seventh decades, the mean age being 60. There were eight males and seven females. Of the 15 cases, eight patients were diagnosed as having arteriosclerotic cardiovascular disease, two each as having coronary artery heart disease, rheumatic heart disease, and hypertensive heart disease, and one as having pulmonary heart disease. All patients were in early phases of therapy for congestive heart failure. None was terminal, but all had, at the time of the study, continuing evidence of cardiac decompensation such

### Table 2.—Cerebral Hemodynamics and Metabolism in Congestive Heart Failure

<table>
<thead>
<tr>
<th>Subject</th>
<th>MABP</th>
<th>BVP</th>
<th>JVP</th>
<th>CSFP</th>
<th>CBF</th>
<th>CVR</th>
<th>CMRO2</th>
<th>CMRG1</th>
<th>R.Q.</th>
</tr>
</thead>
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<tr>
<td>A. F.</td>
<td>81</td>
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<td>58</td>
<td>200</td>
<td>45</td>
<td>1.7</td>
<td>3.5</td>
<td>5</td>
<td>0.98</td>
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<td>86</td>
<td>—</td>
<td>80</td>
<td>220</td>
<td>34</td>
<td>2.4</td>
<td>3.2</td>
<td>—</td>
<td>0.94</td>
</tr>
<tr>
<td>T. J.</td>
<td>149</td>
<td>150</td>
<td>78</td>
<td>260</td>
<td>41</td>
<td>3.5</td>
<td>2.9</td>
<td>2</td>
<td>1.01</td>
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<td>A. C.</td>
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<td>50</td>
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<td>2.9</td>
<td>4</td>
<td>0.95</td>
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<tr>
<td>E. H.</td>
<td>117</td>
<td>90</td>
<td>52</td>
<td>195</td>
<td>42</td>
<td>2.7</td>
<td>1.3</td>
<td>—</td>
<td>0.96</td>
</tr>
<tr>
<td>T. W.</td>
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<td>—</td>
<td>159</td>
<td>300</td>
<td>53</td>
<td>2.9</td>
<td>3.8</td>
<td>—</td>
<td>0.93</td>
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<td>155</td>
<td>260</td>
<td>49</td>
<td>1.8</td>
<td>3.9</td>
<td>—</td>
<td>0.81</td>
</tr>
<tr>
<td>G. S.</td>
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<td>90</td>
<td>90</td>
<td>205</td>
<td>27</td>
<td>3.1</td>
<td>1.9</td>
<td>4</td>
<td>0.90</td>
</tr>
<tr>
<td>H. F.</td>
<td>115</td>
<td>118</td>
<td>80</td>
<td>230</td>
<td>34</td>
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<td>2.6</td>
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<tr>
<td>N. B.</td>
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<td>—</td>
<td>75</td>
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<td>3.1</td>
<td>—</td>
<td>1.01</td>
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<tr>
<td>L. M.</td>
<td>70</td>
<td>210</td>
<td>220</td>
<td>350</td>
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<td>2.2</td>
<td>2.4</td>
<td>3</td>
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</tr>
<tr>
<td>J. C.</td>
<td>143</td>
<td>39</td>
<td>128</td>
<td>250</td>
<td>25</td>
<td>5.4</td>
<td>2.2</td>
<td>4</td>
<td>0.94</td>
</tr>
<tr>
<td>J. Co.</td>
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<td>130</td>
<td>184</td>
<td>150</td>
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<td>2.3</td>
<td>8</td>
<td>0.97</td>
</tr>
<tr>
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<td>100</td>
<td>136</td>
<td>300</td>
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<td>1.7</td>
<td>11</td>
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</tr>
<tr>
<td>B. B.</td>
<td>130</td>
<td>78</td>
<td>110</td>
<td>230</td>
<td>57</td>
<td>2.1</td>
<td>3.1</td>
<td>2</td>
<td>0.85</td>
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**Controla** (18)

<table>
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<tr>
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<th>110</th>
<th>111</th>
<th>120</th>
<th>255</th>
<th>40</th>
<th>2.6</th>
<th>2.7</th>
<th>4.5</th>
<th>0.93</th>
</tr>
</thead>
<tbody>
<tr>
<td>S.E.</td>
<td>±7.2</td>
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<td>±13.7</td>
<td>±15.5</td>
<td>±2.6</td>
<td>±0.24</td>
<td>±0.20</td>
<td>±0.91</td>
<td>±0.017</td>
</tr>
<tr>
<td>p</td>
<td>&gt;.05</td>
<td>&lt;.01</td>
<td>&lt;.02</td>
<td>&lt;.02</td>
<td>&gt;.1</td>
<td>&lt;.05</td>
<td>&gt;.5</td>
<td>&gt;.5</td>
<td>&gt;.9</td>
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</tbody>
</table>

**Normals** (12)

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<th>80</th>
<th>202</th>
<th>48</th>
<th>2.0</th>
<th>2.9</th>
<th>6.2</th>
<th>0.92</th>
</tr>
</thead>
<tbody>
<tr>
<td>S.E.</td>
<td>±2.4</td>
<td>±3.3</td>
<td>±6.9</td>
<td>±15.0</td>
<td>±4.0</td>
<td>±0.15</td>
<td>±0.27</td>
<td>±2.5</td>
<td>±0.25</td>
</tr>
</tbody>
</table>

### Abbreviations: MABP: Mean arterial blood pressure, mm. Hg; BVP: Brachial venous pressure, mm. HgO; JVP: Jugular venous pressure, mm. HgO; CSFP: Cerebrospinal fluid pressure, mm. HgO; CBF: Cerebral blood flow, cc./100 Gm./min.; CVR: Cerebrovascular resistance, mm. Hg/cc./100 Gm./min.; CMRO2: Cerebral metabolic rate, oxygen utilization, cc./100 Gm./min.; CMRG1: Cerebral metabolic rate, glucose utilization, mg./100 Gm./min.; R.Q.: Cerebral respiratory quotient; S.E.: Standard error; p: probability of chance occurrence; *: Statistically significant difference from value found in congestive heart failure.

and Nelson. The cerebral metabolic rate with respect to oxygen utilization (CMRO2) was calculated by multiplying the cerebral blood flow by the arteriovenous oxygen difference. The cerebral metabolic rate with respect to glucose utilization (CMRG1) was calculated by multiplying the cerebral blood flow by the arteriovenous glucose difference. The cerebrovascular resistance (CVR) was calculated by use of the formula: $CVR = P/CBF$, in which $P$ represents the difference between the...
as dyspnea, orthopnea, basal rales, peripheral edema, serous effusions, congestive hepatomegaly, and elevated venous pressure. The presence of an elevated venous pressure generally separates those with predominantly right heart failure from those with predominantly left heart failure. All patients with the exception of N. B. were mentally clear at the time of the study.

Table 3.—Blood Constituents in Congestive Heart Failure

<table>
<thead>
<tr>
<th>Subject</th>
<th>AO₂</th>
<th>ACO₂</th>
<th>ApH</th>
<th>ApCO₂</th>
<th>AGI</th>
<th>VO₂</th>
<th>VCO₂</th>
<th>VpH</th>
<th>VpCO₂</th>
<th>VGl</th>
<th>(A-V)Ο₂</th>
<th>(A-V) CO₂</th>
<th>(A-V) Gl</th>
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</thead>
<tbody>
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<td>52.88</td>
<td>7.34</td>
<td>48</td>
<td>156</td>
<td>7.72</td>
<td>7.41</td>
<td>12</td>
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<tr>
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<td>42.74</td>
<td>7.43</td>
<td>33</td>
<td>67</td>
<td>10.98</td>
<td>49.77</td>
<td>7.40</td>
<td>40</td>
<td>63</td>
<td>6.96</td>
<td>7.03</td>
<td>4</td>
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<td>7.34</td>
<td>44</td>
<td>107</td>
<td>9.81</td>
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<td>7.30</td>
<td>53</td>
<td>100</td>
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<td>7</td>
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<td>7.32</td>
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<td>—</td>
<td>12.00</td>
<td>52.40</td>
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<td>—</td>
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<td>2.93</td>
<td>—</td>
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<td>7.62</td>
<td>24</td>
<td>—</td>
<td>8.41</td>
<td>51.28</td>
<td>7.55</td>
<td>29</td>
<td>—</td>
<td>7.24</td>
<td>6.76</td>
<td>—</td>
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<tr>
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<td>—</td>
<td>13.25</td>
<td>51.50</td>
<td>7.35</td>
<td>47</td>
<td>—</td>
<td>7.91</td>
<td>6.46</td>
<td>—</td>
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<td>54.62</td>
<td>7.38</td>
<td>47</td>
<td>100</td>
<td>8.27</td>
<td>61.92</td>
<td>7.32</td>
<td>60</td>
<td>85</td>
<td>7.00</td>
<td>6.30</td>
<td>15</td>
</tr>
<tr>
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<td>49.40</td>
<td>7.35</td>
<td>46</td>
<td>103</td>
<td>9.21</td>
<td>56.87</td>
<td>7.29</td>
<td>58</td>
<td>98</td>
<td>7.72</td>
<td>7.47</td>
<td>8</td>
</tr>
<tr>
<td>N. B.</td>
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<td>44.01</td>
<td>7.46</td>
<td>35</td>
<td>—</td>
<td>11.61</td>
<td>52.55</td>
<td>7.31</td>
<td>53</td>
<td>—</td>
<td>8.45</td>
<td>8.54</td>
<td>—</td>
</tr>
<tr>
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<td>17.74</td>
<td>41.85</td>
<td>7.40</td>
<td>36</td>
<td>65</td>
<td>8.07</td>
<td>50.76</td>
<td>7.33</td>
<td>45</td>
<td>55</td>
<td>9.67</td>
<td>8.92</td>
<td>10</td>
</tr>
<tr>
<td>J. C.</td>
<td>17.27</td>
<td>48.71</td>
<td>7.33</td>
<td>47</td>
<td>78</td>
<td>8.50</td>
<td>56.96</td>
<td>7.29</td>
<td>55</td>
<td>63</td>
<td>8.77</td>
<td>8.25</td>
<td>15</td>
</tr>
<tr>
<td>J. Co.</td>
<td>17.35</td>
<td>33.13</td>
<td>7.33</td>
<td>33</td>
<td>250</td>
<td>10.91</td>
<td>39.35</td>
<td>7.26</td>
<td>41</td>
<td>227</td>
<td>6.44</td>
<td>6.22</td>
<td>23</td>
</tr>
<tr>
<td>D. N.</td>
<td>14.73</td>
<td>49.26</td>
<td>7.27</td>
<td>53</td>
<td>247</td>
<td>10.93</td>
<td>53.56</td>
<td>7.22</td>
<td>60</td>
<td>223</td>
<td>3.80</td>
<td>4.30</td>
<td>24</td>
</tr>
<tr>
<td>B. B.</td>
<td>20.10</td>
<td>52.67</td>
<td>7.33</td>
<td>52</td>
<td>92</td>
<td>14.65</td>
<td>57.27</td>
<td>7.30</td>
<td>58</td>
<td>88</td>
<td>5.45</td>
<td>4.60</td>
<td>4</td>
</tr>
</tbody>
</table>

Mean | 17.68 | 45.65 | 7.39 | 40 | 128 | 10.65 | 52.36 | 7.32 | 50 | 116 | 7.03 | 6.58 | 12 |
S.E. | ±0.57 | ±1.43 | ±0.01 | ±2.1 | ±22 | ±0.53 | ±1.58 | ±0.03 | ±2.3 | ±20 | ±0.49 | ±0.45 | ±2.3 |
p | >.1 | >.5 | >.2 | >.05 | >.3 | >.3 | >.8 | >.7 | >.3 | >.5 | >.05 | >.1 | >.6 |

"Controls" (18)

Mean | 16.47 | 46.97 | 7.35 | 45 | 103 | 9.76 | 52.63 | 7.31 | 53 | 102 | 6.03 | 5.62 | 15 |
S.E. | ±0.65 | ±1.33 | ±0.04 | ±1.9 | ±11 | ±0.82 | ±1.47 | ±0.03 | ±2.3 | ±13 | ±0.30 | ±0.37 | ±6.2 |

"Normals" (12)

Mean | 16.12 | 47.12 | 7.36 | 43 | 87 | 9.55 | 53.59 | 7.32 | 49 | 75 | 6.57 | 6.47 | 14 |
S.E. | ±0.77 | ±1.37 | ±0.03 | ±1.5 | ±13 | ±0.82 | ±1.23 | ±0.04 | ±1.9 | ±12 | ±0.32 | ±0.45 | ±1.8 |

Abbreviations: A: Arterial (femoral); V: Venous (internal jugular); O₂: Oxygen content, vol.%; CO₂: Carbon dioxide content, vol.%; pCO₂: Partial pressure of carbon dioxide, mm. Hg; (A-V): Arteriovenous difference; Gl: Glucose content, mg. %.

**Results**

Table 2 presents in detail the results obtained in a study of the various parameters concerned with the cerebral hemodynamics and metabolism in the 15 cases of congestive heart failure and, for comparison, a summary of the results obtained in this laboratory in a series of 18 cases classified as "controls" and 12 cases classified as "normal." The control group consisted of individuals between the ages of 50 and 76, the mean age being 62. All had evidence of the presence of systemic arteriosclerosis but none was in congestive heart failure. No cases with a mean arterial blood pressure above 115 mm. Hg were included. The "normal" group included only individuals under the age of 40. Most of these patients were suffering from functional ailments or were convalescing from acute infectious diseases. No hypertensives were included in this group.

The mean arterial blood pressure of 110 mm. Hg for the congestive heart failure series is slightly but significantly higher than the mean value of 95 mm. Hg and 91 mm. Hg observed in the "control" and "normal" groups respectively. This value is not, however, above the
value of 115 mm. Hg which we arbitrarily use
to separate normotensives from definite hypertensives.

The mean brachial venous pressure of 111
mm. H₂O is significantly higher than the mean
values of 61 mm. H₂O for the “controls” and
70 mm. H₂O for the “normals.” Similarly, the
mean jugular venous pressure of 120 mm. H₂O
and the mean cerebrospinal fluid pressure of
255 mm. H₂O for the congestive heart failure
group are both significantly higher than the
respective values for the “control” group which
are 80 mm. H₂O and 202 mm. H₂O. Although
the jugular venous pressure of the decompensation
group is higher than the mean value of
103 mm. H₂O for the “normals,” the difference
is not statistically significant. The mean cerebrospinal fluid pressure of the failure group is,
however, significantly higher than the mean
value of 202 mm. H₂O for the “normals.”

Coming now to the important quantity of
cerebral blood flow, the value of 40 cc. per
100 Gm. per minute in congestive heart failure
is not significantly lower than the mean of 48
cc. per 100 Gm. per minute for the “control”
group. The former value is, however, signifi-
cantly below the mean normal value for the
cerebral blood flow of 53 cc. per 100 Gm. per
minute. The same relationship is seen to hold
in a comparison of the mean values for the
cerebral metabolic rate (oxygen); that is, 2.7
cc. per 100 Gm. per minute (congestive heart
failure) does not differ significantly from the
“control” mean of 2.9 cc. per 100 Gm. per
minute, although it is significantly lower than the “normal” mean value of 3.4 cc. per 100 Gm.
per minute.

A significant increase of the cerebral vascular
resistance in congestive heart failure is noted
when the mean value of 2.6 mm. Hg per cc.
per 100 Gm. per minute is compared with the
“control” value of 2.0 mm. Hg per cc. per 100
Gm. per minute and the “normal” mean of
1.8 mm. Hg per cc. per 100 Gm. per minute.

No significant differences in the cerebral
metabolic rate (glucose) or the cerebral respira-
tory quotient are noted among the three
groups compared.

Table 3 summarizes the values obtained for
the various blood constituents of the three
groups. Without repeating the individual values
noted in the table, it is sufficient to point out
that there are no significant differences among
the three groups for any of the quantities
measured.

**Discussion**

The results of this investigation indicate
that moderate heart failure in itself does not
significantly lower the cerebral blood flow below
the level found in the presence of arterio-
sclerosis without heart failure. The mean value
for the cerebral blood flow of 40 cc. per 100
Gm. per minute in the failure group does not
differ significantly from the value of 48 cc. per
100 Gm. per minute in a control arteriosclerotic
group. The fact that there is a difference, albeit
not a statistically significant one, may be attri-
buted to errors of random sampling and the
probable existence of a severer degree of ar-
eriosclerosis in the failure group than in the
“control” group. Such reduction of the cerebral
blood flow that is observed is principally a func-
tion of organic occlusive changes in the cerebral
vasculature of a group at this age level and is
not directly caused by congestive heart failure.
The finding by Moyer and associates, using
similar technics, of a cerebral blood flow of
45 cc. per 100 Gm. per minute in congestive
heart failure as compared to their normal of
53 cc. per 100 Gm. per minute is in close agree-
ment with our own observations. The signifi-
cance of their findings, as would be true of our
own, is misinterpreted without reference to a
proper “control” group such as we have pre-
sented. The absolute values reported by
Scheinberg are not strictly comparable to our
own because of differences in technic used.

The reduced cerebral metabolic rate (oxygen)
noted in the failure series is also related to the
presence of older age and arteriosclerosis rather
than to the presence of cardiac decompensa-
tion. Both failure and “control” groups show
an approximately 20 per cent reduction of the
cerebral metabolic rate (oxygen) below the
normal value of 3.4 cc. per 100 Gm. per minute.
The exact significance of this reduced metabolic
rate in the presence of arteriosclerosis is not
immediately apparent, although an excellent
correlation exists between the reductions from
normal of the cerebral blood flow and the cerebral metabolic rate in respect to oxygen (correlation coefficient, 0.75). Our results also fail to reveal a significant depression in glucose utilization in either the "control" arteriosclerotic group or the failure group. These relationships are discussed more fully elsewhere.11

On first inspection of the results, it would appear that there actually is an increased cerebral vascular resistance in the presence of congestive heart failure. The value of 2.6 mm. Hg per cc. per 100 Gm. per minute is significantly higher than the "control" value of 2.0 mm. Hg per cubic centimeter per 100 Gm. per minute. However, it must be recalled that an increased cerebral vascular resistance without concomitant change in the cerebral blood flow is a characteristic pattern in essential hypertension.12 If the cerebral vascular resistance of the failure group is recalculated after eliminating the seven instances in which the mean arterial blood pressure is above 115 mm. Hg, a value of 2.2 mm. Hg per cubic centimeter per 100 Gm. per minute is obtained, and this does not differ from the "control" value. It is thus seen that it is unnecessary to implicate changes in the arterial carbon dioxide tensions in order to explain an increased cerebral vascular resistance in congestive heart failure, for there actually is no increased cerebral vascular resistance attributable to the failure, per se.

As implied in the foregoing discussion, no differences among the mean values for the arterial carbon dioxide tension in the three groups compared were found. Furthermore, no correlation was found between the arterial carbon dioxide tension and the cerebral blood flow in the failure series. Thus, whatever instances of a reduced cerebral blood flow were found accompanying congestive heart failure, could not be related to a decreased arterial carbon dioxide tension.

The presence of an elevated venous pressure, both brachial and jugular, and an elevated cerebrospinal fluid pressure are to be expected in the presence of congestive heart failure, and our series is no exception. A general parallelism between the degree of elevation of the venous pressure and the cerebrospinal fluid pressure has been previously noted13 and is confirmed in our data. The degree to which an elevated cerebrospinal fluid pressure might be responsible for an increased cerebral vascular resistance and a decreased cerebral blood flow in this series is probably negligible. Kety, Shenkin, and Schmidt14 have demonstrated that the cerebrospinal fluid pressure must exceed the critical level of 450 mm. HgO before the cerebral blood flow is significantly reduced by increased intracranial tension. Certainly, however, we cannot exclude the possibility that in isolated cases such values are obtained and may thus contribute to cerebral circulatory insufficiency with resultant mental changes. Similarly, although we are unable to correlate decreases in cerebral blood flow and increases in cerebral vascular resistance with the magnitude of the venous pressure elevation or the severity of the failure as judged clinically, we cannot exclude the existence of a critical level for the cardiac output below which the cerebral circulation is embarrassed. Reason would lead us to believe that this probably is the case. However, in a limited series such as has been studied, such factors as degree of failure, height of cerebrospinal fluid pressure and height of venous pressure are of little significance in the etiology of such changes as were found. The general absence of mental symptoms among the failure cases studied precludes the drawing of conclusions concerning the relationship of changes in cerebral blood flow and the cerebral utilization of oxygen to the occurrence of alterations of consciousness or personality. However, as has been implied, in extreme cases of congestive failure, such mental aberrations could be attributed to any or all of the various mechanisms enumerated.

On a purely teleologic basis, what has been demonstrated by actual measurement, the tendency for the cerebral circulation to be maintained in spite of a decreasing cardiac output, might well have been predicted. One would expect such protection to be afforded to the circulation of an organ so sensitive to anoxic and anemic changes as is the brain. Investigation has shown a similar relationship to hold for the coronary circulation in congestive heart failure. Both coronary blood flow
and myocardial oxygen utilization are maintained in the face of marked decreases in the cardiac output. On the other hand, the hepatic flow is decreased in proportion to the drop in cardiac output, and the renal blood flow is decreased to a much greater extent than is the cardiac output. From these various studies there thus emerges a basic physiologic pattern: the redistribution of a reduced cardiac output in congestive failure in such a way as to preserve the circulation of the two organs most essential for immediate survival, the heart and the brain.

**Summary**

1. The cerebral blood flow, metabolism, and vascular resistance have been studied in a group of 15 patients with moderately congestive heart failure. No significant differences were found when the respective values were compared with values obtained for a control group.

2. The cerebral oxygen utilization and blood flow in congestive heart failure were found to be significantly reduced below values established for normal young individuals, but these changes were demonstrated to be a function of increasing age and arteriosclerosis and not of congestive heart failure.

3. Both internal jugular venous pressure and cerebrospinal fluid pressure were found to be elevated in cardiac decompensation. These elevations were of similar magnitudes, but in no instance in the series studied was the degree of elevation of either quantity sufficient to adversely influence the cerebral circulation.

4. Measurement of arterial carbon dioxide tension failed to reveal instances of a reduced cerebral blood flow which could be attributed to a reduced arterial carbon dioxide tension.

5. It is concluded that in congestive heart failure there is a tendency for the cerebral blood flow and metabolism to be maintained in the face of a diminished cardiac output, although the possibility of cerebral circulatory embarrassment in extreme degrees of failure has not been excluded and is even considered quite likely.

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**Sumario Español**

En un estudio de la circulación y metabolismo cerebral en decompensación cardíaca, usando la técnica de Kety con oxígeno de nitrógeno, se encontró que no hubo alteraciones significativas en la circulación cerebral cuando las cantidades medidas fueron comparadas con valores obtenidos en un grupo control. Un decremento en circulación cerebral y consumo de oxígeno bajo los niveles establecidos para sujetos jóvenes normales se demostró ser causado por la presencia de arteriosclerosis y no debido a decompensación cardíaca como previamente se había concluido. También se encontró en la serie estudiada que el aumento en presión venosa y cerebrosaquiña no fue de suficiente magnitud como para afectar la circulación cerebral. El factor de reducción en la tensión del dióxido de carbono arterial como una causa en el decremento en circulación cerebral de decompensación cardíaca fue similarmente considerado y eliminado.

**References**


Studies of the Cerebral Circulation and Metabolism in Congestive Heart Failure
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