Circulatory changes in acute glomerulonephritis at rest and during exercise

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In order to evaluate the effects of acute glomerulonephritis on the circulation, 6 patients were investigated at rest and during moderate exercise. With the patients in a state of rest the cardiac index and the stroke volume index were significantly higher in acute glomerulonephritis than normal, despite significantly raised right and left atrial pressures. Oxygen consumption was significantly increased (P < 0.01) and the arteriovenous oxygen difference was narrowed significantly (P < 0.001) in acute glomerulonephritis as compared to normal subjects. The calculated increase in cardiac output was due to both a rise in oxygen consumption and a narrowing of arteriovenous oxygen difference, the latter being more significant.

The exercise-induced changes in cardiac output in the patients with glomerulonephritis were not different from those in normal subjects.

These results showed that the circulatory changes in the oliguric stage of acute glomerulonephritis resemble those in the hyperkinetic states; the raised mean right atrial and pulmonary wedge pressures do not indicate the presence of heart failure when resting cardiac output is above normal level and response to exercise is normal.

The manifestations of circulatory congestion encountered in the oliguric phase of acute glomerulonephritis are considered by some authors (Wilson, 1967; Gore and Saphir, 1948; Master, Jaffe, and Dack, 1937; Whitehill, Longcope, and Williams, 1939) to be an expression of heart failure. On the other hand, Davies (1951) and Eichna et al. (1954) stressed that the syndrome suggesting heart failure seen in acute glomerulonephritis was an extra-cardiac circulatory congestion caused by water and salt retention.

Haemodynamic studies in acute glomerulonephritis have revealed that performance of the heart as a pump is not impaired. Davies (1951) and Eichna et al. (1954) reported normal values for cardiac output and stroke volume at rest. DeFazio et al. (1959) found that cardiac output and stroke volume were raised despite high right and left atrial pressures at rest.

Circulatory changes during exercise in acute glomerulonephritis have not yet been investigated thoroughly. DeFazio et al. (1959) found normal response to exercise in one patient and Guz et al. (1966) found the slope of the cardiac output/oxygen consumption relation to be increased during exercise in one of his patients.

Since it is known that the evaluation of cardiac performance requires exercise data, it was considered that haemodynamic response to exercise in acute glomerulonephritis would provide additional information concerning myocardial function in this particular syndrome. These considerations form the background of this haemodynamic study in 6 patients with acute glomerulonephritis.

Subjects and methods

Studies were made of 6 patients with typical clinical and laboratory findings of post-streptococcal acute glomerulonephritis and of 6 normal subjects as controls. Informed consent for the study was obtained from the patients as well as from the control group. The important clinical and laboratory findings in the patients with acute glomerulonephritis are shown in Tables 1 and 2. All the patients were male, ranging in age from 14 to 24 years. Patients were studied before the onset of natural diuresis. As a result of diuresis patients lost about 2 to 6 kg in the days after the haemodynamic study. Pyrexia was not present in any of the patients. All cases had palpebral and 3 cases also had pretribial pitting oedema (Cases 1, 5, and 6). Six patients had dyspnoea, 4 patients
TABLE 1  Clinical findings*

<table>
<thead>
<tr>
<th>Case</th>
<th>Duration of disease (days)</th>
<th>Dyspnoea</th>
<th>Orthopnoea</th>
<th>Râles</th>
<th>Oedema</th>
<th>Weight loss (kg)</th>
<th>Transverse diameter % decrease‡</th>
<th>Murmurs</th>
<th>Gallop rhythm</th>
<th>Arterial pressure (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M 19</td>
<td>7</td>
<td>±</td>
<td>±</td>
<td>2</td>
<td>20</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>180/90</td>
</tr>
<tr>
<td>2</td>
<td>M 15</td>
<td>6</td>
<td>+</td>
<td>+</td>
<td>3</td>
<td>18</td>
<td>Grade I systolic</td>
<td>—</td>
<td>—</td>
<td>160/90</td>
</tr>
<tr>
<td>3</td>
<td>M 16</td>
<td>7</td>
<td>+</td>
<td>+</td>
<td>3</td>
<td>16</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>150/85</td>
</tr>
<tr>
<td>4</td>
<td>M 16</td>
<td>7</td>
<td>++</td>
<td>++</td>
<td>4</td>
<td>22</td>
<td>Grade I systolic</td>
<td>—</td>
<td>—</td>
<td>185/100</td>
</tr>
<tr>
<td>5</td>
<td>M 14</td>
<td>3</td>
<td>+++</td>
<td>+</td>
<td>5</td>
<td>23</td>
<td>Grade I systolic</td>
<td>+</td>
<td>165/95</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>M 24</td>
<td>4</td>
<td>++</td>
<td>++</td>
<td>6</td>
<td>25</td>
<td>Grade I systolic</td>
<td>+</td>
<td>195/95</td>
<td></td>
</tr>
</tbody>
</table>

Conversion factor from Traditional to SI Units: 1 mmHg = 0.133 kPa; 1 cmH2O = 0.98 kPa.
* Most of the clinical findings are graded on a scale of + to +++.
† Duration of the disease when catheterization was performed.
‡ Decrease of the transverse diameter of the heart after natural diuresis.

had mild orthopnoea, and 2 patients had apical diastolic gallop rhythm (S2). Pulmonary basal râles were heard in 5 patients, jugular venous pressure was increased in all patients, and the circulation time was in the normal range before the study. The transverse diameter of the heart was increased in all patients during the oliguric phase. After diuresis transverse diameters of the heart decreased appreciably, the mean decrease being 21 per cent. Transverse diameter of the heart was measured on standard 6 foot posteroanterior chest films of the patients before and after diuresis was initiated. Slight to moderate arterial hypertension was present and urinary findings consisted of + to ++ albumin, and red blood cells; some of the patients had cylindruria. Blood urea nitrogen concentrations were between 39.3 and 74.9 mmol/l (55 and 105 mg/dl) in the patients. The total serum protein level was normal in 3 and at lower limits in 3 cases and serum albumin ranged between 30 to 40 g/l. Plasma sodium levels were within normal limits in all cases, plasma potassium level being slightly raised in one case.

Right heart catheterization was performed on the

TABLE 2  Laboratory findings

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Urine Protein</th>
<th>Red cells</th>
<th>Casts</th>
<th>Blood urea nitrogen (mmol/l)</th>
<th>Serum proteins (g/l) Total</th>
<th>Albumin</th>
<th>Globulin</th>
<th>Plasma sodium (mmol/l)</th>
<th>Plasma potassium (mmol/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>+</td>
<td>++ ++</td>
<td>+</td>
<td>39.3</td>
<td>70</td>
<td>40</td>
<td>30</td>
<td>142</td>
<td>5.0</td>
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<tr>
<td>2</td>
<td>+</td>
<td>++ ++</td>
<td>+</td>
<td>63.5</td>
<td>69</td>
<td>40</td>
<td>29</td>
<td>140</td>
<td>5.0</td>
</tr>
<tr>
<td>3</td>
<td>±</td>
<td>++ ++</td>
<td>+</td>
<td>31.4</td>
<td>69</td>
<td>39</td>
<td>30</td>
<td>140</td>
<td>5.2</td>
</tr>
<tr>
<td>4</td>
<td>±</td>
<td>++ ++</td>
<td>+</td>
<td>74.9</td>
<td>58</td>
<td>34</td>
<td>24</td>
<td>150</td>
<td>6.1</td>
</tr>
<tr>
<td>5</td>
<td>+</td>
<td>++ ++</td>
<td>+</td>
<td>41.4</td>
<td>60</td>
<td>34</td>
<td>28</td>
<td>150</td>
<td>4.8</td>
</tr>
<tr>
<td>6</td>
<td>++</td>
<td>++ ++</td>
<td>+</td>
<td>62.8</td>
<td>59</td>
<td>31</td>
<td>28</td>
<td>148</td>
<td>4.4</td>
</tr>
</tbody>
</table>

N normal range.
Circulatory changes in acute glomerulonephritis at rest and during exercise

<table>
<thead>
<tr>
<th>Venous pressure (cmH2O)</th>
<th>Circulation time (s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>14</td>
<td>10</td>
</tr>
<tr>
<td>16</td>
<td>13</td>
</tr>
<tr>
<td>20</td>
<td>12</td>
</tr>
<tr>
<td>20</td>
<td>10</td>
</tr>
<tr>
<td>22</td>
<td>12</td>
</tr>
</tbody>
</table>

and the pulmonary artery, and their oxygen content, capacity, and saturation were determined in duplicate, errors being less than 0.2 per cent vol. O2 by the method of Van Slyke and Neill (1924) and Peters and Van Slyke (1943). Immediately after the determination of cardiac output, simultaneous pulmonary arterial and brachial arterial pressures were recorded by a Sanborn strain gauge transducer and photographic writing polyviso (Electronics for Medicine). The zero reference point for supine position was taken as the midpoint of the antero-posterior diameter of the chest at the level of the third interspace at the sternum. Mean pressures were obtained by electrical integration.

After observations at rest, haemodynamic response to moderate exercise was studied. Higher and repeated work loads were not attempted in order to avoid the potential risk of dyspnoea which can initiate anxiety, resulting in changes in cardiac output. Exercise studies involved pedalling a bicycle ergometer in the recumbent position at a rate of 60 revolutions per minute at the same load (50 watts) for 5 minutes, all parameters being measured during the last minute of exercise. From the results the following calculations were made:

Total peripheral resistance in dynes s cm⁻⁵ =

\[
\frac{BAm \times 1332}{CO}
\]

Pulmonary arteriolar resistance in dynes s cm⁻⁵ =

\[
\frac{(PAm-PWm) \times 1332}{CO}
\]

Left ventricular work index in kg m/min per m² BSA =

\[
\frac{CI \times 1.055 \times (BAm-PWm) \times 13.6}{1000}
\]

Right ventricular work index in kg m/min per m² BSA =

\[
\frac{CI \times 1.055 \times (PAm-RAm) \times 13.6}{1000}
\]

Where: \(BAm\) = mean brachial artery pressure in mmHg; \(PAm\) = mean pulmonary artery pressure in mmHg; \(PWM\) = mean pulmonary artery wedge pressure in mmHg; \(RAm\) = mean right atrial pressure in mmHg; \(CO\) = cardiac output in ml per second; \(CI\) = cardiac index in litre/min per m² BSA; 1332 = conversion factor for mmHg to dynes/cm²; 1055 = specific gravity of blood and 13.6 = specific gravity of mercury.

For statistical evaluation of the haemodynamic results at rest, data obtained from patients with acute glomerulonephritis have been compared with those obtained from normal subjects. For each resting haemodynamic parameter, the significance of the difference between the means of normal and acute glomerulonephritis patients has been evaluated by the utilization of the t test (Schwartz and Lazar, 1964).

Response to exercise was evaluated by comparing the percentage changes in pre-exercise and post-exercise values for normal subjects with those for patients with acute glomerulonephritis.

normal subjects and patients in the overnight fasting state, and to obtain a reasonable sedation 50 to 100 mg pethidine ('demerol') was given one hour before the start of the procedure. A double lumen catheter was employed; the distal opening was wedged in a branch of the pulmonary artery in order to obtain pulmonary wedge pressure and the proximal opening was in the pulmonary artery. An indwelling Cournand needle was placed in the brachial artery to obtain arterial pressures and blood samples.

Cardiac outputs were determined by the direct Fick principle (Fick, 1870). Measurement of cardiac output by the oxygen Fick method can sometimes be in error by more than 100 per cent. This, however, is not caused by any basic error in the principle itself, but instead by misapplication of the principle (Guyton, 1963; Visscher and Johnson, 1953). The types of errors involved can be classified into three categories: 1) errors in sampling and analysis; 2) errors caused by unstable cardiac outputs; 3) errors caused by a changing respiratory condition.

To minimize the potential errors in the measurement of cardiac output which are listed above, the following method was applied. Oxygen consumption was determined by analysis of expired air which was collected in a Tissot spirometer and of room air for oxygen content on a Pauling oxygen analyser. During the collection of expired air, blood samples were slowly and simultaneously withdrawn under oil from the brachial artery.
TABLE 3
Haemodynamic changes at rest and during exercise in normal subjects

<table>
<thead>
<tr>
<th>Case No., sex, age</th>
<th>Body surface area (m²)</th>
<th>O₂ consumption (ml/min per m² BSA)</th>
<th>O₂ AV difference (m/l)</th>
<th>Cardiac index (lm/min per m² BSA)</th>
<th>Heart rate</th>
<th>Stroke index (ml/beat per m² BSA)</th>
<th>Mean pressure (mmHg)</th>
<th>Resistance (dynes cm⁻⁵)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1, F, 25</td>
<td>R 1.69</td>
<td>140</td>
<td>35</td>
<td>4.00</td>
<td>92</td>
<td>43</td>
<td>92 16 7 3</td>
<td>1090</td>
</tr>
<tr>
<td>2, F, 25</td>
<td>E 1.69</td>
<td>140</td>
<td>38</td>
<td>3.69</td>
<td>95</td>
<td>39</td>
<td>102 17 6 4</td>
<td>965</td>
</tr>
<tr>
<td>3, M, 20</td>
<td>E 1.62</td>
<td>200</td>
<td>40</td>
<td>5.00</td>
<td>80</td>
<td>60</td>
<td>100 18 7 4</td>
<td>990</td>
</tr>
<tr>
<td>4, M, 30</td>
<td>E 1.59</td>
<td>198</td>
<td>39</td>
<td>5.08</td>
<td>88</td>
<td>58</td>
<td>80 18 7 2</td>
<td>789</td>
</tr>
<tr>
<td>5, M, 22</td>
<td>E 1.56</td>
<td>170</td>
<td>38</td>
<td>4.47</td>
<td>89</td>
<td>50</td>
<td>93 17 7 3</td>
<td>1068</td>
</tr>
<tr>
<td>6, M, 24</td>
<td>E 1.50</td>
<td>150</td>
<td>42</td>
<td>3.57</td>
<td>70</td>
<td>51</td>
<td>92 17 6 3</td>
<td>1160</td>
</tr>
<tr>
<td>Mean</td>
<td>E 1.61</td>
<td>166</td>
<td>39</td>
<td>4.30</td>
<td>86</td>
<td>50</td>
<td>93 17 7 3</td>
<td>1043</td>
</tr>
</tbody>
</table>

Conversion factor from Traditional to SI units: 1 mmHg ≈ 0.133 kPa; 10 dynes cm⁻⁵ ≈ 1 MPa m⁻³.

TABLE 4
Haemodynamic changes at rest and during exercise in patients with acute glomerulonephritis

<table>
<thead>
<tr>
<th>Case No., sex, age</th>
<th>Body surface area (m²)</th>
<th>O₂ consumption (ml/min per m² BSA)</th>
<th>O₂ AV difference (m/l)</th>
<th>Cardiac index (lm/min per m² BSA)</th>
<th>Heart rate</th>
<th>Stroke index (ml/beat per m² BSA)</th>
<th>Mean pressure (mmHg)</th>
<th>Resistance (dynes cm⁻⁵)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1, M, 19</td>
<td>R 1.50</td>
<td>144</td>
<td>30</td>
<td>4.80</td>
<td>75</td>
<td>55</td>
<td>125 20 13 6</td>
<td>1386</td>
</tr>
<tr>
<td>2, M, 15</td>
<td>E 1.39</td>
<td>240</td>
<td>41</td>
<td>5.85</td>
<td>90</td>
<td>65</td>
<td>135 22 16 6</td>
<td>1225</td>
</tr>
<tr>
<td>3, M, 16</td>
<td>E 1.36</td>
<td>194</td>
<td>35</td>
<td>5.54</td>
<td>80</td>
<td>69</td>
<td>120 22 13 6</td>
<td>1245</td>
</tr>
<tr>
<td>4, M, 16</td>
<td>E 1.50</td>
<td>400</td>
<td>50</td>
<td>8.00</td>
<td>95</td>
<td>84</td>
<td>128 24 16 5</td>
<td>922</td>
</tr>
<tr>
<td>5, M, 14</td>
<td>E 1.20</td>
<td>160</td>
<td>34</td>
<td>4.71</td>
<td>100</td>
<td>40</td>
<td>110 22 16 4</td>
<td>1344</td>
</tr>
<tr>
<td>6, M, 24</td>
<td>E 1.60</td>
<td>316</td>
<td>50</td>
<td>6.32</td>
<td>115</td>
<td>51</td>
<td>116 24 19 4</td>
<td>1081</td>
</tr>
<tr>
<td>Mean</td>
<td>E 1.42</td>
<td>180</td>
<td>34</td>
<td>5.30</td>
<td>52</td>
<td>89</td>
<td>140 25 14 7</td>
<td>1402</td>
</tr>
</tbody>
</table>

Conversion factor from Traditional to SI units: 1 mmHg ≈ 0.133 kPa; 10 dynes cm⁻⁵ ≈ 1 MPa m⁻³.

Results

The haemodynamic data in normal subjects and in patients with acute glomerulonephritis at rest and during exercise are presented in Tables 3 and 4, respectively. Table 5 shows the mean values in normal subjects and patients and the P values of the difference of the means. Haemodynamic changes during exercise in normal subjects and in acute glomerulonephritis are presented in Table 6.

With the patients at rest, the cardiac index was higher than the highest value of normal subjects in 3 patients, with a range from 4.71 to 6.38 l/min per m² BSA. The mean of the patient group was 5.36 l/min per m² BSA compared with the normal mean of 4.30 l/min per m² BSA. The difference is significant at a level of less than 2 per cent. Exercise-induced changes in cardiac index were similar in normal subjects and in patients with glomerulonephritis, the mean increment being 39 per cent and 40 per cent, respectively.

Oxygen consumption in patients at rest was significantly increased at a level of less than 1 per cent; it
Pulmonary Left
were
80 normals the
increased
tion
venous
oxxygen
mean
normal
arteriolar
in
widened
changes
7.36
I04
152
4.86
Pulmonary
Left
6.65
io8
152
4.39
I60
t0
144
5.30
II3
9.55
5.00
80
8.42
75
13.88
42
8.10
70
12.86
50
8.10
I.9
54
11.97
1.88

---

averaged 181 ml/min per m² BSA with a range of 144 to 217 ml/min per m² BSA, as compared to the normal mean of 166 ml/min per m² BSA. Arterio-
venous oxygen difference were narrowed signifi-
cantly (P <0.001) in all patients, ranging from 30 to
35 ml per litre. During exercise oxygen consump-
tion increased and arteriovenous oxygen difference
widened in acute glomerulonephritis, the average
changes being 93 and 35 per cent, respectively. In
normals the average changes in these measurements
were 80 and 28 per cent, respectively.

Pulse rates were generally within normal limits,
with a range of 52 to 100 per minute in acute
glomerulonephritis and they increased 23 per cent
during exercise. In normals the increase was 27
per cent.

The stroke volume index was significantly
greater than normal (P <0.02), the mean stroke volume
index for the acute glomerulonephritis being 66 ml
per beat per m² BSA, as compared to the normal
mean of 50 ml per beat per m² BSA. Stroke volume
index increased 14 per cent in normals and 19 per
cent in patients during exercise.

The pulmonary wedge pressures were signifi-
cantly raised in all patients (P <0.001), ranging
from 13 to 16 mmHg (1.7 to 2.1 kPa), with a mean
of 15 mmHg (2.0 kPa), as compared to the normal
mean of 7 mmHg (0.9 kPa). The same significant
increase (P <0.001) was observed in right atrial
pressure in all patients except one (Case 3), ranging
from 4 to 8 mmHg (0.5 to 1.1 kPa), with a mean of
6 mmHg (0.8 kPa), as compared to the normal sub-
jects. Both pulmonary capillary wedge and right
atrial mean pressures were raised during exercise,
the increment being 29 per cent and 33 per cent in
normal subjects and 20 per cent and 17 per cent in
acute glomerulonephritis, respectively.

Significant increase in the pulmonary artery
mean pressure was observed in all patients with a
mean of 22 mmHg (2.9 kPa), as compared to the
normal mean of 17 mmHg (2.7 kPa). In the patients,
the brachial arterial mean pressures ranged from
120 to 140 mmHg (16.0 to 18.6 kPa), with a mean
of 122 mmHg (16.2 kPa), which is significantly
higher than the normal mean of 93 mmHg (12.4
kPa) (P <0.001). Both left and right ventricular
work indices were significantly higher than normal
(P <0.001). The calculated total peripheral resis-
tance was significantly higher than normal (P <0.001),
and pulmonary arteriolar resistance was lower than
normal (P <0.02). In both groups, pulmonary and
brachial arterial pressures, and left and right ven-
tricular work indices increased while total per-
ipheral and pulmonary arteriolar resistance de-
creased appreciably during exercise.

Discussion
A particular haemodynamic pattern has been
observed in our cases of acute glomerulonephritis: a
high cardiac output and stroke volume, a re-
duced arteriovenous oxygen difference, a normal to
high oxygen consumption, raised left and right atrial
pressures, a high total peripheral resistance, a normal
to low pulmonary arteriolar resistance, and high
systemic arterial and pulmonary arterial pressures
at rest. This is in accord with the observations of Guz et al. (1966), Bradley, Jenkins, and Branthwaite (1971), and DeFazio et al. (1959). The supernormal response to exercise, alleged to exist in some of the hyperkinetic states, was not observed in our cases. This finding conflicts with the findings of Guz et al. (1966) who found that cardiac output was high for the prevailing oxygen consumption in one case of acute glomerulonephritis during exercise.

This high cardiac output at rest and normal response to exercise places these patients in the group of hyperkinetic states which includes patients with chronic anaemia, hyperthyroidism, and chronic arteriovenous fistula. Since none of these diseases were present in our cases, it is supposed that hypervolaemia caused by salt and water retention, which occurs in most patients in the oliguric stage of acute glomerulonephritis (Eichna, 1960; Eisenberg, 1958; Cardozo, 1946), is responsible for the hyperkinetic state.

Right atrial mean pressure and pulmonary wedge mean pressure were raised to a level which was not high enough to produce signs and symptoms of pulmonary congestion and peripheral oedema. But the low serum albumin concentration observed in our 3 cases, together with salt and water retention, might have contributed to the development of pulmonary congestion and peripheral oedema, in spite of relatively low values for left and right atrial pressures.

Increased ventricular end-diastolic pressures, in the face of the high cardiac output at rest, cannot be regarded as evidence in these patients that they were in heart failure. But, according to Guz et al. (1966), high cardiac output at rest does

### Table 5: Mean values in normal subjects and patients with glomerulonephritis at rest

<table>
<thead>
<tr>
<th>Group</th>
<th>O$_2$ consumption (ml/min per m$^2$ BSA)</th>
<th>Cardiac index (l/min per m$^2$ BSA)</th>
<th>Heart rate</th>
<th>Stroke index (ml/beat per m$^2$ BSA)</th>
<th>Mean pressures (mmHg)</th>
<th>Resistance (dynes s cm$^{-1}$)</th>
<th>Work index (kg m/min per m$^2$ BSA)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>166 39 4.30 86 50 93 17 7 3</td>
<td>1043 122 5.30 0.87</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AGN 181</td>
<td>34 5.36 80 66 122 22 15 6</td>
<td>1284 75 8.42 1.19</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>t 3.2 13.0 3.0 1.7 3.0 10.5 13.0 12.0 11.0</td>
<td>4.0 2.6 6.8 14.0</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P &lt;0.01 &lt;0.001 &lt;0.02 &lt;0.10 &lt;0.02 &lt;0.001 &lt;0.001 &lt;0.001 &lt;0.001 &lt;0.001</td>
<td></td>
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</tbody>
</table>

BA, brachial artery; PA, pulmonary artery; PW, pulmonary artery wedge; RA, right atrium; TPR, total peripheral resistance; PAR, pulmonary arteriolar resistance; LV, left ventricular; RV, right ventricular; AGN, acute glomerulonephritis.

Conversion from Traditional to SI units: 1 mmHg $\approx$ 0.133 kPa; 10 dynes s cm$^{-1}$ $\approx$ 1 MPa s m$^{-2}$.

### Table 6: Haemodynamic changes at rest and during exercise in normal subjects and in patients with acute glomerulonephritis (mean values)

<table>
<thead>
<tr>
<th>Group</th>
<th>Experimental condition</th>
<th>O$_2$ consumption (ml/min per m$^2$ BSA)</th>
<th>Cardiac index (l/min per m$^2$ BSA)</th>
<th>Heart rate</th>
<th>Stroke index (ml/beat per m$^2$ BSA)</th>
<th>Mean pressures (mmHg)</th>
<th>BA</th>
<th>PA</th>
<th>PW</th>
<th>RA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>R</td>
<td>166 39 4.30 86 50 93 17 7 3</td>
<td>50 93 17 7 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>E</td>
<td>299 50 5.98 109 57 99 19 9 4</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>% change</td>
<td>+80</td>
<td>+28</td>
<td>+39</td>
<td>+27</td>
<td>+14</td>
<td>+60</td>
<td>+12</td>
<td>+29</td>
<td>+33</td>
<td></td>
</tr>
<tr>
<td>Acute</td>
<td>R</td>
<td>181 34 5.36 80 66 122 22 15 6</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>glomerulonephritis</td>
<td>E</td>
<td>349 46 7.48 98 78 131 24 18 7</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>% change</td>
<td>+93</td>
<td>+35</td>
<td>+40</td>
<td>+23</td>
<td>+19</td>
<td>+8</td>
<td>+9</td>
<td>+20</td>
<td>+17</td>
<td></td>
</tr>
</tbody>
</table>

Conversion from Traditional to SI units: 1 mmHg $\approx$ 0.133 kPa; 10 dynes s cm$^{-1}$ $\approx$ 1 MPa s m$^{-2}$.
not necessarily indicate that the heart is functioning normally and the reserve capacity of the heart should be tested with exercise. When the mean pulmonary wedge pressures were related to stroke volume index it was seen that stroke volume increased more than the pulmonary wedge pressure on exercise as compared to normal subjects. Since in each of our cases the pulmonary wedge pressure was not related to the stroke volume, an increase in the latter could be explained either by an increase in diastolic volume without change in pulmonary wedge pressure or by a more complete emptying of the ventricles because of the increased ventricular contractility. The increase in cardiac size during the oliguric phase and its decrease after natural diuresis or after frusemide injection (Santibenez, Rosendo, and Atherton, 1971) suggest that the diastolic volume is increased in acute glomerulonephritis. Since contractility of the ventricles was not measured in this series, it is a matter of conjecture as to whether or not an increase in contractility, during rest and exercise, contributed to the increase in cardiac output.

When the overall results obtained in our cases were evaluated, it was clearly seen that the circulatory changes in acute glomerulonephritis resembled those in hyperkinetic states and the increase in atrial pressures did not indicate the presence of heart failure when resting cardiac output was above normal level and response to exercise was normal.

### Circulatory changes in acute glomerulonephritis at rest and during exercise

<table>
<thead>
<tr>
<th>Resistance (dynes s cm⁻⁵)</th>
<th>Work index (kg m/min per m² BSA)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulmonary arteriolar</td>
<td>Left ventricular</td>
</tr>
<tr>
<td>1043</td>
<td>122</td>
</tr>
<tr>
<td>808</td>
<td>80</td>
</tr>
<tr>
<td>1284</td>
<td>-34</td>
</tr>
<tr>
<td>1005</td>
<td>75</td>
</tr>
<tr>
<td>-22</td>
<td>54</td>
</tr>
<tr>
<td>-28</td>
<td>+4.2</td>
</tr>
</tbody>
</table>

### References


Requests for reprints to Professor Kenan Binak, Taksim, Topcu Caddesi, Elbir Apt. 22/2, Istanbul, Turkey.
Circulatory changes in acute glomerulonephritis at rest and during exercise.

K Binak, N Sirmaci, D Uçak and N Harmanci

*Br Heart J* 1975 37: 833-839
doi: 10.1136/hrt.37.8.833

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