Effect of Acute Cardiac Tamponade on Red Cell Volume, Total Plasma Protein and Plasma Volume

By Rex J. Underwood, M.D., Herbert E. Griswold, Jr., M.D. and William W. Hurst, M.D.

Acute heart failure was produced by cardiac tamponade in unsplenectomized and splenectomized dogs. The results indicate that splenic contraction was entirely responsible for the rise in hematocrit observed following tamponade in the unsplenectomized dogs. A fall in the hematocrit values of the splenectomized dogs and a reduction in total plasma proteins of both groups indicates an increase in plasma volume following tamponade. There was no evidence of sequestration of red cells.

Reports indicate that experimental acute failure produced in dogs by coronary artery ligation, embolization of the coronary circulation, or by injection of a necrosing substance in the myocardium result in a rise in hematocrit and a fall in plasma volume as measured by dye substances. The rise in hematocrit readings has been considered to be consistent with the hemoconcentration evidenced by the fall in plasma volume. On the other hand, there is experimental evidence that acute heart failure in cats produced by cardiac tamponade reduces the cell-plasma ratio, suggesting hemodilution due to an increase in plasma volume.

The hemodynamic consequences of cardiac tamponade are similar to those resulting from acute myocardial failure and have been described by Starling and others. The purpose of this investigation was to determine whether or not cardiac tamponade in dogs produces changes in blood volume which are similar to those produced by other types of acute heart failure. Changes in the hematocrit values, red cell volume and plasma protein concentration were used as indications of the direction and amount of any alteration in blood volume which might occur immediately following tamponade.

METHODS

Experiments were performed on 22 mongrel dogs weighing between 9 and 15 Kg. and anesthetized with 30 mg./Kg. of sodium pentobarbital. Seven of the dogs were splenectomized 5 to 10 days before the experiment.

The chest was opened and a polyethylene catheter inserted into the pericardial sac and sutured securely in place. The distal end of the catheter was brought out through a stab wound in the anterior chest wall, the chest incision closed and the dog allowed to breathe unassisted for the duration of the experiment.

After the establishment of control measurements, normal saline was injected into the catheter until mean arterial pressure was approximately 50 per cent of the control level.

Hematocrit ratios and total plasma proteins were determined on arterial blood samples from all dogs at various time intervals before and during tamponade. Total plasma protein was determined by Weichselbaum’s modification of the biuret method.

Red cell volume was measured with Cr tagged cells in 3 unsplenectomized dogs and 3 splenectomized dogs. The cells were labeled using the procedure described by Gray and Sterling. The dose of tagged cells was determined by a method which does not require the use of calibrated syringes (unpublished method of Dr. T. T. Hutchens). Whole blood samples were counted in a well type scintillation counter.

Red cells volume was measured before tamponade by injecting a dose of tagged cells and allowing a 30 min. period for mixing before withdrawing a sample for counting. Tagged cells were reinfected 20 min. after starting tamponade. Mixing after this reinfusion was assumed to be complete when the
counting rates of at least 2 successive samples drawn at 10 to 15 min. intervals agreed within 0.5 per cent. All volume determinations were corrected for blood removal.

**RESULTS**

The measurement of red cell volume during tamponade by reinjection of tagged cells in 3 unsplenectomized and 3 splenectomized dogs showed no significant change from control values in either group. In unsplenectomized dogs the mean red cell volumes were respectively, 34 and 31 ml./Kg. before and after cardiac tamponade and in the splenectomized animals they were respectively, 31 and 30 ml./Kg. The time required for reaching a constant number of counts per second per cc of red cells in arterial blood ranged between 15 and 35 minutes (average 25 minutes).

Following tamponade the hematocrit values in the unsplenectomized dogs increased over control levels by an average of 20 per cent, with a range of 11 to 55 per cent. The mean control hematocrit was 42.5 per cent. On the other hand hematocrit values in the splenectomized dogs fell slightly during tamponade. The mean control hematocrit was 40 per cent and the decrease ranged from 2 to 12 per cent of control values with a mean decrease of 6 per cent. This change was significant at the 0.05 level. In both groups of dogs, the changes in hematocrit values were maximum by 10 min. after starting the tamponade.

Figure 1 shows the comparison of changes in the hematocrit readings of the splenectomized and the unsplenectomized dogs at various time intervals during and after tamponade. The difference between the two groups is significant at the 0.01 level at all time intervals.

Total plasma proteins in the unsplenectomized dogs decreased during tamponade by an average of 0.4 Gm. per cent, with a range of 0.2 to 0.6 Gm. per cent. The mean control protein value was 5.50 Gm. per cent. Total protein in the splenectomized dogs also decreased by an average of 0.4 Gm. per cent, with a range of 0.2 to 0.5 Gm. per cent. The mean control protein value in this group was 4.45 Gm. per cent.

In both groups of dogs, the change in plasma protein concentration was at or near maximum 10 min. after starting tamponade. Samples were drawn for protein determination at 20 min. intervals for 1 hour during tamponade. There was no significant difference between the mean change in protein concentrations in the two groups at any of the time intervals.

The reason for the decline of hematocrit readings and of plasma-protein in splenectomized dogs was sought by comparing the fall in plasma-protein concentration which would result from the observed reduction in cell-plasma ratio and the observed protein concentration. Table 1 provides a tabulation of data

<table>
<thead>
<tr>
<th>Tag no.</th>
<th>Control hematocrit (%)</th>
<th>Control total protein (Gm.%)</th>
<th>Average hematocrit during tamponade (%)</th>
<th>Average observed total protein during tamponade (Gm.%)</th>
<th>Predicted* total protein (Gm.%)</th>
<th>Observed minus predicted total protein (Gm.%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>22</td>
<td>43.0</td>
<td>4.50</td>
<td>42.0</td>
<td>4.15</td>
<td>4.33</td>
<td>-0.18</td>
</tr>
<tr>
<td>24</td>
<td>37.5</td>
<td>3.85</td>
<td>36.0</td>
<td>3.62</td>
<td>3.62</td>
<td>0.00</td>
</tr>
<tr>
<td>25</td>
<td>30.5</td>
<td>4.15</td>
<td>29.5</td>
<td>4.00</td>
<td>3.96</td>
<td>+0.04</td>
</tr>
<tr>
<td>26</td>
<td>30.5</td>
<td>4.33</td>
<td>35.0</td>
<td>3.50</td>
<td>3.60</td>
<td>-0.10</td>
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<td>29</td>
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<td>4.80</td>
<td>38.0</td>
<td>4.40</td>
<td>4.42</td>
<td>-0.02</td>
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<tr>
<td>31</td>
<td>50.5</td>
<td>5.00</td>
<td>48.0</td>
<td>4.38</td>
<td>4.53</td>
<td>-0.15</td>
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<tr>
<td>Mean</td>
<td>40.0</td>
<td>4.44</td>
<td>38.6</td>
<td>4.01</td>
<td>4.08</td>
<td>-0.07</td>
</tr>
</tbody>
</table>

* Protein value predicted on the basis of change in hematocrit.
† 95 per cent confidence limits. Mean difference not significant at 0.05 level.
from 6 splenectomized dogs. The next to the last column shows the predicted values and the last column the difference between the mean observed and the predicted protein concentrations. The mean difference of 0.07 Gm. has 95 per cent confidence limits of ±0.10 Gm. and is, therefore, essentially zero. Consequently, the decrease in hematocrit readings that splenectomized dogs sustain after cardiac tamponade appears to be due to an increase in plasma volume. This conclusion was supported by the findings that plasma volumes calculated from red cell volumes and hematocrit readings of arterial blood were increased from 46 to 52 ml./Kg.; differences which are significant at the 0.05 level. It is recognized that such calculations do not accurately reflect plasma volumes, but they do indicate directional changes.

**DISCUSSION**

In view of the observations on splenectomized animals it appears highly probable that the hemoconcentration following cardiac tamponade in barbitalized, unsplenectomized dogs can be explained by contraction of an engorged spleen.

Since tamponade and acute heart failure produced by myocardial damage both result in a reduction of cardiac output, a fall in systemic and pulmonary arterial pressures, and a rise in systemic and pulmonary venous pressure, hydrostatic pressure changes in the capillary beds should be in the same direction in both instances. Hence, one would predict that shifts in plasma volume would also occur in the same direction and that our data are comparable to those of previous workers.

Previous investigators have concluded that experimental acute heart failure in the dog results in hemoconcentration. Since they employed unsplenectomized dogs it is possible that this hemoconcentration was not due to sequestration of red cells, but to a combination of splenic contraction and the prolonged mixing time of dye substances used to measure plasma volume. The slowing of the circulation during tamponade would appear to increase appreciably the time required for mixing of tagged cells over the generally accepted mixing time of ten minutes. This prolonged mixing time could cause errors in the dye dilution estimations of plasma volume since incompletely mixed dye samples would cause plasma volume to appear falsely low.

**SUMMARY**

Acute cardiac tamponade was produced in unsplenectomized dogs in an effort to determine whether or not tamponade produces the same type of change in blood volume as has been reported in other types of acute heart failure in dogs. Hematocrit readings, Cr51 red cell volumes and plasma protein concentrations were determined before and during tamponade. The following conclusions were reached:

Cardiac tamponade in the unsplenectomized dog is followed by splenic contraction, resulting in the discharge of cell-rich blood into the general circulation and a rise in arterial hematocrit readings.

In splenectomized dogs plasma volume increases by about 6 ml./Kg. following tamponade as shown by the fall in the hematocrit values and the reduction in plasma protein concentration in both groups.

There was no evidence for the sequestration of red cells during tamponade in the time period studied.

**ACKNOWLEDGMENT**

We are grateful to Dr. John M. Brookhart for his helpful advice and criticism; and to Miss Ruth Bawden, Miss Betty Dornbusch and Mrs. Kay Elliott for technical assistance.

**SUMMARY IN INTERLINGUA**

Acute tamponamento cardiac esseva producite in non-splenectomisate canes pro determinar si o non tamponamento produce in iste animales un typo de alteration del volumine sanguine idetico con illo reportate previemente como effecto de altere formas de disfallimento cardiac acute in canes. Lecturas de hematocrite, determinaciones a Cr51 del volumine erythrocytic, e estimationes del proteina plasmatic esseva obtenite ante a durante le tamponamento. Esseva formulate le sequente conclusiones:

Tamponamento cardiac in non-splenectomy-
sate canes es sequite per contraction splenic que resulta in le discarga de sanguine ric in cellulas a in le circulation general e in un elevation del lecturas de hematocrite arterial. Le volumine de plasma cresce per circa 6 ml/kg post tamponamento. Iste facto es manifeste in le reduction del valores de hematocrite in canes splenectomisate e le reduction del concentration de proteina plasmatic in ambe gruppos.

Le studio non indica que il occurre sequestration of erythrocytos in tamponamento durante periodos del typo studiate.

REFERENCES

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