Effectiveness of Congesting Cuffs ("Rotating Tourniquets") in Patients with Left Heart Failure

By Philip A. Habak, M.D., Allyn L. Mark, M.D., J. Michael Kioschos, M.D., Donald R. McRaven, M.D., and Francois M. Abboud, M.D.

SUMMARY

Congesting cuffs or "rotating tourniquets" are often used to treat patients with acute pulmonary edema secondary to left heart failure. The purpose of this study was to evaluate the effectiveness of congesting cuffs in pooling blood in the extremities and decreasing pulmonary congesting pressures in patients with left heart failure and to define optimal congesting cuff pressures. Congesting cuffs on three extremities were inflated to 20, 40, 60, and 80 mm Hg in 16 patients with left heart failure and in 7 normal subjects. The extremities were elevated to collapse the veins before inflating the cuffs. The amount of venous pooling was measured with mercury-in-silastic strain gauge plethysmographs. Inflation of the cuffs produced significantly less venous pooling in patients with heart failure than in normal subjects. Decreases in right atrial pressure during inflation of the cuffs were also significantly less in the patients with heart failure than in the normal subjects. In patients with heart failure, pulmonary congesting pressure (left ventricular diastolic pressure or mean pulmonary arterial wedge pressure) averaged 24.2 ± 1.5 (SEM) mm Hg in the control period and 24.6 ± 2.2, 22.9 ± 1.9, 23.2 ± 2.1 and 20.3 ± 1.6 mm Hg during inflation of the cuffs at 20, 40, 60, and 80 mm Hg, respectively. The decreases in pulmonary congesting pressure (PCP) were not significant (F > 0.05) at cuff pressures of 20, 40 and 60 mm Hg. Decreases in PCP were statistically significant at a cuff pressure of 80 mm Hg, but only 6 of 16 patients had decreases greater than 4 mm Hg. The results suggest that in patients with heart failure the effectiveness of congesting cuffs is limited by decreases in venous distensibility which are characteristic of heart failure.

Additional Indexing Words:

Plethysmography
Venous distensibility
Pulmonary arterial wedge pressure
Left ventricular end diastolic pressure
Capillary filtration coefficient

CONGESTING CUFFS are frequently used in the treatment of patients with pulmonary edema resulting from left heart failure in order to pool blood in the veins and decrease venous return and pulmonary congestion. Despite the fact that this form of treatment has been used for many years\(^1,2\) there has been no systematic study either of its effectiveness in decreasing pulmonary congesting pressures in patients with heart failure or of the optimal con-

gesting pressures. Ebert and Stead\(^3\) demonstrated that 580-980 ml of blood may be impounded in three extremities when congesting cuffs are inflated to diastolic pressure in normal subjects, but these investigators did not study patients with heart failure or evaluate effectiveness of lower congesting pressures.

There are several reasons why patients with heart failure might respond differently from normal subjects to congesting cuffs. Wood\(^4\) has demonstrated that the systemic veins of patients with congestive heart failure are less distensible than normal. This suggests that congesting cuffs might be less effective in pooling blood in the veins and decreasing venous return and pulmonary congestion in these patients. On the other hand, pulmonary vascular pressure-volume curves are thought to be steep in patients with heart failure, so that small changes in pulmonary blood volume result in large changes in pulmonary congesting pressure.\(^5\) This suggests that even small amounts of pooling and decreases in venous return might be associated with large decreases in pulmonary congesting pressures.

The systemic veins are more distensible at low dis-
tending pressures than at high pressures.⁶ Consequently, lower congesting pressures might pool almost as much blood as higher pressures without some of the undesirable effects which might result from the latter. For example, hypotension has been reported during application of high congesting pressures.⁷ High congesting pressures also may produce excessive capillary filtration⁸ and, thereby, increase blood viscosity and vascular resistance. These considerations suggest that lower congesting pressures, if effective in decreasing pulmonary congesting pressure, might be more desirable than high pressures in the treatment of patients with pulmonary edema.

The purpose of the study was, therefore, to evaluate the effectiveness of congesting cuffs in patients with left heart failure and to define optimal congesting cuff pressures. We compared responses to lower congesting cuff pressures (20 and 40 mm Hg) with responses to higher pressures (60 and 80 mm Hg) in normal subjects and in patients with left heart failure.

**Methods**

Fourteen patients were studied during diagnostic cardiac catheterization. Two other patients were studied in the coronary care unit. The diagnoses and some of the hemodynamic data on these patients are shown in table 1. All sixteen patients had evidence of left ventricular failure based on a recent history of orthopnea and paroxysmal nocturnal dyspnea and elevated left ventricular end diastolic or mean pulmonary arterial wedge pressure (> 15 mm Hg). All but one had large left ventricular end diastolic volumes (> 110 ml/m² BSA), and all but two had ejection fractions less than 50%. In four of the patients studied in the catheterization laboratory and one of the patients (KM) studied in the coronary care unit, the onset of cardiac decompensation was recent. None of the patients had pitting edema. The majority of the patients were taking digitalis and diuretics, and no change in therapy was made for the study.

The patients were studied in the supine position with both legs and the left forearm elevated 15 cm above the level of the right atrium to collapse the veins. The ambient temperature was 24°C. Sphygmomanometer cuffs, 13 cm wide for the arm and 20 cm wide for the thighs, were used as congesting cuffs. The cuffs were fitted around the left arm and both thighs close to the trunk and taped to facilitate transmission of pressure in the cuff to the extremity. A cuff pressure of 5-7 mm Hg was necessary to initiate venous congestion and increases in limb volume. This pressure, which has previously been referred to as "cuff zero,"⁹ was taken as the zero level for subsequent inflation to 20, 40, 60 and 80 mm Hg. The increase in volume of the forearm (15 patients)

**Table 1**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Diagnosis</th>
<th>MRAP (mm Hg)</th>
<th>PCP (mm Hg)</th>
<th>LVEDV (ml/m²)</th>
<th>LVSEF (%)</th>
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<td>6</td>
<td>15</td>
<td>161</td>
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<td>Coronary artery disease</td>
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</table>

Abbreviations: MRAP = mean right atrial pressure; PCP = pulmonary congesting pressure. Entries for PCP represent left ventricular end diastolic pressure except for entries indicated by * which represent left ventricular diastolic pressure before a large atrial wave and entries indicated by ‡ which represent mean pulmonary arterial wedge pressure; LVEDV = left ventricular end diastolic volume: normal = 30-110 ml/m² BSA; LVSEF = left ventricular systolic ejection fraction: normal = > 50%.

‡Patients with recent onset decompensation.
and calf (6 patients) during congestion was measured with mercury-in-silastic strain gauge plethysmographs and expressed in ml/100 ml limb volume. Capillary filtration during congestion was estimated from the increase in limb volume above control volume after the cuffs were deflated and was expressed in ml/100 ml of limb volume.

Right atrial, systemic arterial and pulmonary congesting pressures were measured with conventional pressure transducers and were recorded on a physiologic recorder. The reference point for these pressures was the mid-chest. Values for pulmonary congesting pressure consist of measurements of mean pulmonary arterial wedge pressure in the two patients studied in the coronary care unit, left ventricular end diastolic pressure in 10 patients, and left ventricular diastolic pressure just before a large atrial wave in 4 patients. In these 4 patients it was found that because of the large atrial wave, measurements of left ventricular diastolic pressure just before the atrial wave provided better correlation with mean pulmonary arterial wedge pressure than did end diastolic pressure. None of the patients had mitral stenosis. In four patients in whom end diastolic and pulmonary wedge pressure were measured simultaneously during congestion, the changes in mean pulmonary wedge pressure paralleled changes in end diastolic pressure. Consequently, left ventricular end diastolic pressure, diastolic pressure before the atrial wave, or mean pulmonary arterial wedge pressure are referred to as pulmonary congesting pressure.

After obtaining control measurements, the cuffs were inflated consecutively to 20, 40, 60 and 80 mm Hg for three minutes each without releasing congestion between each level and the subsequent one. Measurements of limb volume and pressures were taken at the end of each period of congestion. Similar studies were performed on seven normal subjects, except that left ventricular and pulmonary arterial wedge pressures were not measured.

In two normal subjects, we measured the total volume of the extremities (2 lower and 1 upper) distal to the cuffs by measuring the amount of water displaced and found that it was approximately 12 liters (5 liters for each lower and 2 liters for the upper). These values for total limb volume were then used to calculate the estimated total amount of fluid pooled at each level in the rest of the subjects. The total amount of fluid pooled at each level was calculated by multiplying the amount pooled in ml/100 ml by the volume of the extremities expressed in 100 ml units. For example, increases in calf volume of 4.3 ml/100 ml represent an estimated pooling of 430 ml (4.3 x 100) and increases in forearm volume of 4.4 ml/100 ml represent estimated pooling of 88 ml (4.4 x 20) for a total estimated pooling in the three extremities of 518 ml. It might be noted here that this calculation is not a critical part of the study and was performed only to provide an estimate of the total amount of pooling; it almost certainly overestimates the total amount of pooling for the following reasons. The pooling presumably occurs in skin and skeletal muscle and not in bone, but the calculation is expressed in ml/100 ml of total limb volume (skin, muscle and bone). Since the actual measurements were made at the level of the extremity (mid-calf or upper forearm) where the ratio of skin and muscle volume to total limb volume is higher than at any other level of the limb distal to the cuffs, the calculation of total amount of pooling derived from the measurement at this level probably overestimates the total amount of pooling.

Analysis of variance and Dunnett's test for multiple comparisons were used for statistical analysis.* Values of $P < 0.05$ were taken as statistically significant.

**Results**

**Limb Volumes**

Increases in forearm and calf volume during inflation of the congesting cuffs were significantly less in patients with heart failure than in normal subjects (table 2). The increases in forearm volume expressed in ml/100 ml limb volume did not differ significantly from the increases in calf volume in either group (table 2).

The average of the estimated total amount pooled in the three extremities at a congesting pressure of 40 mm Hg was 518 ml in the normal subjects and 366 ml in the patients with heart failure. Corresponding values at a congesting pressure of 80 mm Hg were 736 ml in normal subjects and 596 ml in patients with heart failure.

The amount of pooling in the forearm of the patients with recent cardiac decompensation did not differ significantly from that in patients with a history of chronic heart failure. Increases in the former group averaged $1.7 \pm 0.3$, $2.5 \pm 0.2$, $3.8 \pm 0.4$ and $5.1 \pm 0.4$ (se) ml/100 ml limb volume compared with increases in the latter group of $1.8 \pm 0.2$, $2.9 \pm 0.3$.

**Table 2**

*Increases in Limb Volumes During Inflation of Congesting Cuffs*

<table>
<thead>
<tr>
<th>Congesting cuff pressures (mm Hg):</th>
<th>Forearm volume (ml/100 ml)</th>
<th>Calf volume (ml/100 ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>20</td>
<td>40</td>
</tr>
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<td>Normal subjects</td>
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<td></td>
</tr>
<tr>
<td>X</td>
<td>3.0</td>
<td>4.4</td>
</tr>
<tr>
<td>SE</td>
<td>0.3</td>
<td>0.5</td>
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<tr>
<td>N</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td>Patients with left heart failure</td>
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<td></td>
</tr>
<tr>
<td>X</td>
<td>1.8</td>
<td>2.8</td>
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<tr>
<td>SE</td>
<td>0.2</td>
<td>0.2</td>
</tr>
<tr>
<td>N</td>
<td>14</td>
<td>14</td>
</tr>
</tbody>
</table>

*R* represents the difference in limb volume in the recovery period after deflation of the cuffs minus limb volume in the control period. This value represents loss of intravascular fluid by capillary filtration.

Increases in forearm and calf volumes at each level of congesting pressure were statistically significant ($P < 0.05$) in both groups.
CONGESTING CUFFS IN LEFT HEART FAILURE

4.0 ± 0.3, 5.3 ± 0.4 ml/100 ml at 20, 40, 60 and 80 mm Hg, respectively.

Capillary filtration at the end of congestion averaged 0.9 ± 0.1 and 0.6 ± 0.1 ml/100 ml in forearm and calf, respectively, in the normal subjects and 0.3 ± 0.1 and 0.1 ± 0.1 ml/100 ml in the forearm and calf, respectively, in the patients with heart failure. Thus, the patients with heart failure not only pooled less blood, but also had less estimated capillary filtration during congestion.

Right Atrial Pressure

Control values for right atrial pressure did not differ significantly in the two groups (table 3), but decreases in right atrial pressure with inflation of the cuffs were significantly less in patients with heart failure than in normal subjects (table 3).

Pulmonary Congestive Pressure

This variable was not measured in normal subjects. In patients with heart failure, pulmonary congestive pressure did not decrease significantly at cuff pressures of 20, 40 and 60 mm Hg, but did decrease significantly at a cuff pressure of 80 mm Hg (table 3). At 40 mm Hg, 3 of 16 patients had a decrease in pulmonary congestive pressure greater than 4 mm Hg. At 60 mm Hg, 3 of 11 patients had decreases greater than 4 mm Hg. At 80 mm Hg, 6 of 16 patients had decreases greater than 4 mm Hg.

Arterial Pressure

In patients with heart failure, mean arterial pressure averaged 82 ± 4.1 mm Hg before congestion and 84 ± 3.7 and 86 ± 3.9 mm Hg at congesting pressures of 40 and 80 mm Hg, respectively. In normal subjects, arterial pressure averaged 82 ± 3.3 mm Hg before congestion and 86 ± 2.2 and 91 ± 3.1 mm Hg at 40 and 80 mm Hg, respectively. Changes in arterial pressure were not significant at 40 mm Hg, but were significantly greater than control (P < 0.05) at 80 mm Hg in both groups.

Discussion

Previous studies have demonstrated that venous distensibility is decreased in patients with heart failure. Consequently, it was not surprising that congesting cuffs produced less venous pooling in patients with heart failure than in normal subjects (table 2). But because of reports that pulmonary vascular pressure-volume curves are steep in heart failure, we anticipated that even small amounts of venous pooling might cause large decreases in pulmonary congesting pressure and cardiac filling pressures. In contrast, the results demonstrated that decreases in right atrial pressure were less in patients with heart failure than in normals during inflation of congesting cuffs (table 2). Although limb volumes increased and right atrial pressure decreased with increasing cuff pressure from 20 to 80 mm Hg, these changes were not reflected in significant decreases in pulmonary congesting pressure except at the highest level of congestion. Thus, in patients with heart failure congesting cuffs produced statistically significant decreases in pulmonary congesting pressure only when the cuffs were inflated to 80 mm Hg, and even at this high cuff pressure, only 6 of 16 patients had decreases in pulmonary congesting pressure of greater than 4 mm Hg. At congesting cuff pressures of 40 and 60 mm Hg, 3 patients had decreases greater than 4 mm Hg.

The smaller amounts of venous pooling in the patients with heart failure were not the result of pre-existing venous distension from elevated venous pressure. Only five of the patients had mean right atrial pressure higher than 10 mm Hg and there was no significant difference between right atrial pressure

Table 3

<table>
<thead>
<tr>
<th>Cardiac Filling Pressures at Various Congesting Cuff Pressures</th>
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<tbody>
<tr>
<td></td>
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<tr>
<td>Normal subjects</td>
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<td></td>
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<tr>
<td>Patients with left heart failure</td>
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Abbreviations: C = control values before inflation of the cuffs; R = pressure recorded in the recovery period three minutes after release of the congesting cuffs; P < 0.05 = values which were significantly different from control values; N = number of patients studied.
in the normal subjects and the patients with left heart failure. Furthermore, before the cuffs were inflated,
the extremities were elevated to collapse the veins. The mechanism of decreased resting venous disten-
sibility in patients with heart failure is not clear, but it is not dependent on elevations in peripheral venous
pressure.\textsuperscript{4} Parenthetically, we might note here that the baseline values for mean right atrial pressure in
the normal subjects are slightly higher than previously reported values. Although this may have resulted
from elevation of the lower extremities, we would note that the values in this study are similar to values
observed in healthy young normal subjects without elevation of the legs in a previous study in our
laboratory.\textsuperscript{10} The important point is that the lower extre-

mities were elevated to collapse the veins in both groups in the present study and baseline right atrial pressures before congestion were similar in the two
groups so that lesser venous pooling in the patients with heart failure cannot be explained by pre-existing
venous distension.

We cannot explain why some patients had appreciable decreases (> 4 mm Hg) in pulmonary con-
gesturing pressure and others did not. We compared one
group which had decreases in pulmonary congesting
pressure ranging from −5 to −14 mm Hg (average
−8; \( N = 6 \)) and another group which had changes
ranging from −1 to +4 mm Hg (average +1; \( N = 6 \)).
There were no significant differences between the two
groups in resting pulmonary congesting pressure,
arterial pressure and right atrial pressure. There were
also no significant differences between the two groups
in the amount of venous pooling or the changes in
right atrial and arterial pressures. Since the amount of
pooling was not different, we might suggest that the
decreases in pulmonary blood volume in “responders”
and “nonresponders” were not different, and that,
therefore, the patients who had appreciable decreases
in congesting pressures had steeper pulmonary
vascular pressure-volume curves. This explanation is
speculative, however, since pulmonary blood volume
was not measured in these patients.

Increases in systemic arterial pressure occurred
during congestion in both normal subjects and
patients. The mechanism is not clear but might in-
volve vasoconstriction produced by decreases in car-
diac filling pressures and activation of reflexes
originating in low pressure cardiopulmonary
baroreceptors.\textsuperscript{10} In addition, at high levels of con-
gesturing pressure the cuffs may have produced a
mechanical increase in vascular resistance or a sym-
pathetic discharge related to discomfort. We con-
sidered the possibility that increases in arterial
pressure and afterload may have prevented decreases
in pulmonary pressure during decreases in preload,

but the magnitude of the increase in arterial pressure
did not correlate with the decrease in pulmonary

pressure.

Limb volumes tended to plateau during the three
minutes of congestion at 20 mm Hg and 40 mm Hg.
This is consistent with previous studies\textsuperscript{11} which ind-
cate that venous volume and distending pressure
reach a plateau during this period. Therefore, longer
periods of congestion would not produce significantly
greater venous pooling at 40 mm Hg. However, at 60
and 80 mm Hg limb volume did not plateau after the
initial increment, but instead continued to increase
slowly. This slow increase represented capillary
filtration since the volume of the extremities remained
above control values after the cuffs were deflated.
The estimated value for capillary filtration coefficient in
the normal subjects (0.0033 ml/min \( \times \) mm Hg \( \times \) 100
ml) was similar to that reported by Landis,\textsuperscript{4} but
capillary filtration coefficient in patients with heart
failure was 0.0011 ml/min \( \times \) mm Hg \( \times \) 100 ml, or
about 33% of the normal value.*

These data suggest that the rate of capillary filtra-
tion may be reduced in patients with heart failure,
perhaps because of increased tone of precapillary
vessels and decreased capillary surface area or perhaps
because of occult edema and increased tissue pressure.
Despite decreased capillary filtration, prolonged in-
flation of congesting cuffs at 80 mm Hg as is usually per-
formed in the clinical setting of left heart failure
might produce additional sequestration of fluid in the
extremities. It should be noted, however, that the rate
of capillary filtration decreases rapidly with prolonged
congestion,\textsuperscript{8} presumably because of increases in
interstitial fluid and pressure which oppose filtration.

The patients in this study had chronic or subacute
left heart failure, but when studied did not have frank
pulmonary edema. We should, therefore, consider the
limitations of applying these results to use of con-
gesturing cuffs in patients with acute pulmonary edema.
Many patients who develop acute pulmonary edema
have subacute or chronic heart failure, but some

*In calculating capillary filtration coefficient (CFC), capillary filtra-
tion in ml/100 was the increase in limb volume above control after
deflation of the cuffs. The increment in postcapillary pressure was
reported assuming that resting postcapillary pressure ap-
norimated 30 mm Hg and that increases in congesting pressure
below 30 mm Hg were not transmitted to the capillaries. Therefore,
the calculated increment in capillary pressure during congestion at
20, 40, 60 and 80 mm Hg was zero, 10, 30 and 50 mm Hg, respec-
tively. Using this approach the increment in capillary pressure dur-
ing the 9 minutes of inflation (congesting period at 20 mm Hg ex-
cluded) averaged 30 mm Hg. The validity of this approach is sup-
ported by two observations: first, the value of CFC in forearm of
normal subjects is similar to that reported by Landis, and second,
limb volumes plateaued during congestion at 20 and 40 mm Hg in-
dicating little or no capillary filtration at these pressures.
patients develop heart failure and pulmonary edema abruptly. It is possible that decreases in venous distensibility, particularly those decreases which result from local factors, may not have developed in patients with abrupt onset of heart failure. If this is true, then patients with abrupt onset of heart failure and pulmonary edema might pool more blood and have greater decreases in pulmonary congesting pressures than the patients in our study. In contrast, there are several factors which might contribute to less venous pooling in patients with acute pulmonary edema. First, in many patients with pulmonary edema there are potent reflex vasoconstrictor stimuli such as hyperventilation, apprehension or pain which tend to decrease the amount of pooling. A second factor is the amount of blood in the veins before inflation of the cuffs. In our studies, the extremities were elevated in order to collapse the veins before congestion, but in treating patients with acute pulmonary edema it is unusual and inadvisable to elevate the extremities and collapse the veins before inflating the cuffs. With the extremities at or below the level of the right atrium, as is usual in the clinical setting, the veins are partially distended before the cuffs are inflated. Consequently, the increment in venous pooling might be less in the clinical setting than in our study. On the other hand, it is possible that pulmonary vascular pressure-volume relationship may be steeper in patients with acute pulmonary edema than in our patients with chronic left heart failure so that small decreases in pulmonary blood volume might produce greater benefit in patients with acute pulmonary edema than in patients with chronic left heart failure. The results of this study should, therefore, be applied cautiously to consideration of congesting cuffs in the treatment of patients with acute pulmonary edema. The study indicates, however, that the decreases in venous distensibility which are characteristic of heart failure limit the amount of venous pooling produced by congesting cuffs.

Acknowledgment
We wish to thank Ms. Julianne Garvey for secretarial assistance.

References
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Circulation. 1974;50:366-371
doi: 10.1161/01.CIR.50.2.366

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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