The Effect of Venous Occlusion With Tourniquets on Peripheral Blood Pooling and Ventricular Function*

Herman O. Klein, M.D.; Eitan Brodsky, M.D.; Reuven Ninio, M.Sc.; Elieser Kaplinsky, M.D.; and Elie Di Segni, M.D.

Rotating tourniquets were once part of the traditional treatment of acute pulmonary edema. Their effectiveness has been questioned and vasodilator therapy has replaced them, but early favorable results suggested that they may play a beneficial role. A radioisotope technique was used to evaluate blood volume increments in the leg after venous occlusion at 60 mm Hg in 26 patients with left ventricular dysfunction following myocardial infarction. Mean radionuclide counts (reflecting the blood volume distal to the occlusion) increased from the preclosure value. Thus, satisfactory trapping of blood is achieved. However, mean left ventricular ejection fraction (EF) decreased slightly but significantly and this decrease in EF was observed in 18 of 26 patients. Left ventricular end-diastolic and end-systolic volume equivalents tended to decrease slightly but not in all patients. Mean stroke volume and cardiac output equivalents were reduced by 14 percent while peripheral resistance increased significantly. The present study thus fails to support the hypothesis that preload reduction by tourniquets improves left ventricular function; the exact opposite effect may occur because of increased afterload.

(CHEST 1993; 103:521-27)

\[ EF = \text{ejection fraction}, \quad LV = \text{left ventricular} \]

Rotating tourniquets were once an integral part of the traditional treatment of acute pulmonary edema.¹⁹ Their use has largely been replaced by modern pharmacologic therapy, including angiotensin-converting enzyme inhibitors. However, Ebert and Stead² had originally demonstrated that an average of 750 ml of blood could be pooled by venous tourniquets and Judson et al.¹⁰ had documented a 5 mm Hg decrease in pulmonary arterial pressure after venous occlusion. Therefore, it is still important to know whether tourniquets could be of potential benefit as an adjunctive measure to modern pharmacologic treatment.

The effectiveness of tourniquets in patients with left ventricular (LV) dysfunction was therefore reexamined by a noninvasive radionuclide method that serially evaluates directional blood volume changes in the capacitance vessels and the accompanying alterations in LV function.

Two specific questions were asked: (1) Do tourniquets succeed in trapping a significant amount of blood in the limbs of patients with congestive heart failure? (2) Do tourniquets improve LV dynamics by reducing preload?

**METHODS**

**Patients**

Twenty-six patients with depressed LV function as defined by an

---

*From the Departments of Cardiology and Nuclear Medicine, Meir General Hospital, Sapir Medical Center, Kfar-Saba (Mr. Ninio) and Sackler School of Medicine, Tel-Aviv University, Tel-Aviv, Israel. Drs. Kaplinsky and Di Segni are currently at The Heart Institute, Sheba Medical Center, Tel-Hashomer, Israel. Manuscript received May 29; revision accepted June 3.*
sensitivity, collimator (Elscint CE-1 and CCL-3, Israel), as previously described. The camera was positioned in the 45-degree left anterior oblique position, adjusted for optimal separation of the ventricles. The position of the camera was kept constant during control and during venous occlusion. Data were acquired by using the synchronized multigated acquisition mode, using the R wave of the electrocardiogram fed into a minicomputer (Elscint Dykomette). The latter divided the R-R interval into 16 equal "frames." Acquisition for each ventriculography study required 3 min.

Data were displayed on an image display screen, and LV time-activity curves were generated by using a semiautomated detection technique with automatic subtraction of background subtraction. The final, background corrected time-activity was automatically derived by the computer, with the end-diastolic counts represented by the early uppermost point on the curve and end-systole by its nadir. Ejection fraction was calculated by the computer from background-corrected end-diastolic and end-systolic counts. Left ventricular end-diastolic volume equivalents were calculated by the following equation:

\[ \text{Volume} = \frac{\text{nuclide counts} \times 0.40 \, \text{s/heart beats} \times \text{frame time}}{\text{counts in 4 ml whole blood in 60 s}} \]

End-systolic volume equivalents were calculated from the end-diastolic volume equivalents and the EF.

Stroke volume equivalents were calculated by subtracting end-systolic from end-diastolic volume equivalents. The systemic arterial resistance equivalent, expressed as a unit-less measurement, was calculated as the ratio of arterial cuff blood pressure in the contralateral arm divided by the cardiac output equivalent (stroke volume equivalent \times heart rate).

Phase b: The scintillation camera was then focused on the right leg so that the radioactivity below the leg cuff was detected on the image-display screen (Fig 1) and radioactive counts in the leg were measured. The examined area included the knee and the area 20 cm above and below it.

The technique of Rutlen et al was used as follows: Three serial 15-s acquisitions of radioactive counts in the leg distal to the sphygmomanometer cuff ("preocclusion counts") first were obtained. The cuff then was inflated to 60 mm Hg and at least four acquisitions of 15 s each were obtained. Equilibration of the counts at a new level ("occlusion counts") usually took place during the third or fourth acquisition. The cuff was then deflated to obtain new "postocclusion counts." In a subset of ten patients, the effect of incremental degrees of occlusion on peripheral counts was examined by subjecting them to occlusion at 40, 60, and 80 mm Hg for 2 min with cuff deflation between each occlusion.

To determine whether whole blood isotope activity was affected by tourniquet application, 5 ml of whole blood were withdrawn from a vein in the unobstructed contralateral arm in 6 patients, both before and after 1 min of cuff inflation when the leg counts had increased to their maximal values.

**Echocardiographic Studies**

Left ventricular function studies also were performed by M-mode echocardiography in 11 of the previously noted patients, selected strictly on the basis of technically adequate visualization of the subendocardium. The studies were carried out shortly before or after the nuclide studies with the Aloka SSD-720 echocardiograph and a 3.0-MHz transducer. Left ventricular measurements were made in duplicate at the level of the chordae tendineae located first by two-dimensional echocardiography before, during, and after the same three-limb occlusion. Left ventricular end-diastolic dimension measurements were timed at the peak of the R wave of the electrocardiogram; LV end-systolic dimension was measured at the minimal internal systolic dimension. Left ventricular fractional shortening was calculated with the formula:

\[ \frac{(LV \text{ end-diastolic dimension} - LV \text{ end-systolic dimension})}{LV \text{ end-diastolic dimension}} \]

**Statistical Analysis**

All data were analyzed by the two-tailed \( t \) test for paired data.
Results were considered to be statistically significant at the p<0.05 value. Intra-observer variability of baseline measurements was derived for the 26 patients as the standard deviation of the differences between the two measurements divided by the mean value. Increments in peripheral blood counts and changes in LV volume equivalents were normalized by handling the control data as 100 percent values and increments or decrements as percent deviations from this baseline. Changes in EF were calculated as absolute changes. Pearson's coefficient of correlation was calculated to relate changes in EF and LV volumes to increments in peripheral nuclide counts.

RESULTS

Veinous Occlusion and Peripheral Radionuclide Counts

The baseline counts obtained before occlusion were stable. Occlusion at 60 mm Hg pressure resulted almost immediately (within 15 s) in increased radionuclide counts distal to the occlusion. Equilibration of the occlusion count was reached within 45 to 60 s in all 26 patients. For the entire group, the mean radionuclide counts increased by 46 ± 26 percent (p<0.0005) at 60 mm Hg occlusion (range, 8 to 108 percent [Fig 2]).

The mean increment in peripheral radioisotope counts in ten patients sequentially subjected to increasing degrees of occlusion was significantly greater with 60 mm Hg of pressure (56 ± 28 percent increment in counts) than with 40 mm Hg (44 ± 24 percent increment; p<0.05). On the other hand, 80 mm Hg of occlusion was not more effective (and perhaps less so) than 60 mm Hg (50 ± 8 percent increment [Fig 3]). The postocclusion counts (after cuff deflation) returned quickly to the baseline values.

The whole blood radionuclide activity was unaffected by tourniquet application to three limbs (10,700 ± 3,780 vs 10,393 ± 3,800 kilocounts, not significant).

Veinous Occlusion and Left Ventricular Function

As required by the protocol, baseline LV function was impaired in all 26 patients, with the mean EF being 0.23 ± 0.09 (range, 0.10 to 0.42). The coefficient of variability for the duplicate sets of measurements was 1.02 percent. After cuff inflation of three limbs at 60 mm Hg, the mean EF decreased to 0.21 ± 0.10. This modest decrease was statistically significant (p<0.05). Furthermore, individual EF decreased in 18 patients (from 0.25 ± 0.09 to 0.21 ± 0.09; p<0.0005), remained unchanged in 2, and increased in only 6 patients (Fig 4). No clinical or laboratory
parameters were predictive of the direction of change in EF.

Mean LV end-diastolic and end-systolic volume equivalents both tended to decrease (by 6.5 and 4.2 percent, respectively) during occlusion; this trend did not reach statistical significance (Table 1). Likewise, there was no significant change in LV dimensions or shortening fraction evidenced with echocardiography (Table 2).

There was no correlation between individual increments in peripheral radionuclide counts and the magnitude or direction of change in EF and LV volume equivalents of the 18 patients in whom EF decreased (Fig 5). Both LV volume equivalents decreased simultaneously during cuff inflation in six patients, both increased in five while only the end-systolic volume increased in another four. Thus, no clear pattern of change in volume could be discerned in the patients in whom EF decreased with tourniquets. Five of the six patients with increased EF during occlusion exhibited a decrease in both end-diastolic and end-systolic volume equivalents.

Mean stroke volume equivalent for the entire group decreased from 0.28 ± 0.14 to 0.24 ± 0.12 units (percent = -14 ± 7 percent; p<0.0005 [Fig 6]). Stroke volume equivalents decreased in 17 patients, remained unchanged in 5, and increased in 4. Since heart rate did not increase (95 ± 7 vs 94 ± 8 beats per minute), mean cardiac output equivalent also decreased significantly (p<0.0005) while blood pressure did not change (Table 1), and the calculated systemic arterial resistance increased significantly (p<0.005 [Fig 6, Table 1]). After release of venous occlusion, the values of LV volume equivalents and EF all returned to control values.

DISCUSSION

The Tourniquet Controversy

Tourniquets were once extensively used in acute

Table 2—Echocardiographic and Nuclide Measurements in 11 Patients Before and During Venous Occlusion

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Occlusion</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Echo diastolic dimension, mm</td>
<td>64.9±8.9</td>
<td>65.3±8.1</td>
<td>NS</td>
</tr>
<tr>
<td>Echo systolic dimension, mm</td>
<td>53.3±10.7</td>
<td>52.6±9.2</td>
<td>NS</td>
</tr>
<tr>
<td>Echo shortening fraction, %</td>
<td>18.0±7.0</td>
<td>19.5±5.5</td>
<td>NS</td>
</tr>
<tr>
<td>Nuclide EF</td>
<td>0.22±0.08</td>
<td>0.20±0.08</td>
<td>&lt;0.005</td>
</tr>
</tbody>
</table>

Effect of Venous Occlusion With Tourniquets (Klein et al)
pulmonary edema, and reports of cardiocirculatory collapse after their application suggest that they may profoundly affect hemodynamics. Early observations that central venous and pulmonary arterial pressures were reduced by tourniquets in congestive heart failure also are consonant with this theory. The observation that tourniquet application is followed by reduced cardiac output and decreased diuresis and sodium excretion also suggests that tourniquets reduce effective blood volume. Diastolic mitral flow velocity patterns also have been shown to be altered during tourniquet application.

However, the findings of Brams and Golden and of more recent investigators have cast doubt on the effectiveness of tourniquets in heart failure. Samet et al estimated that tourniquets decreased the effective blood volume by only about 5 percent. Habak et al demonstrated that right atrial pressure decreased significantly less in patients with congestive heart failure than in normal subjects and that pulmonary wedge pressure was significantly reduced only if the constricting cuffs were inflated to 80 mm Hg. Increases in limb volume measured by strain-gauge plethysmography also were significantly smaller in patients with congestive heart failure than in normal subjects. Bertel and Steiner found no difference in mean pulmonary arterial and wedge pressures or in right atrial pressure after venous occlusion of the legs at 50 mm Hg. Roth et al similarly failed to observe any change in pulmonary pressure with leg occlusion at 60 mm Hg.

**Determination of Peripheral Venous Pooling**

All previous studies that dealt with blood volume changes in the limbs with tourniquet application are methodologically problematic because they relied on techniques which either warranted unproven assumptions or introduced interventions that could themselves alter the results obtained. For example, plethysmography used by Habak et al involved elevating the limbs 15 cm above the heart to collapse the veins, which is certainly not the position required for treating acute pulmonary edema.

The study of Samet et al required 2 h of supine rest and at least one more hour for tourniquet application, again bearing no resemblance to the clinical situation. Furthermore, their results were obtained by indirect calculations of circulating red cell mass and plasma volume and not by direct data on blood actually pooled in the occluded extremities. Finally, both studies involved very heterogeneous patients: three had coronary artery disease and only one was studied shortly after an acute myocardial infarction.

In the present study, only patients with EF of less than 0.42, all recovering from myocardial infarction and suffering from acute or subacute LV failure, were studied. In addition, the radionuclide technique permits study of the lower limb positioned below or at the level of the heart and allows quick determination of rapid changes in local blood volume. The technique also compares favorably with fluid-displacement plethysmography and is sensitive enough to detect intravascular volume changes during tourniquet application at pressures as low as 15 mm Hg.

**Tourniquets and Trapping of Peripheral Blood**

This study shows that almost all post-infarction patients with recent or new CHF respond satisfactorily to venous occlusion at 60 mm Hg by a mean increase of 46 ± 26 percent in the peripheral blood pool. This increment even exceeds that obtained in similar patients with morphine. No benefit seems to accrue from further cuff inflation to 80 mm Hg. On the other hand, occlusion at 40 mm Hg, as traditionally performed in the past, is significantly less effective in pooling blood (Fig 3).

If we use the data of Vismara et al, the 46 ± 26 percent increase in radionuclide counts produced by 60 mm Hg, occluding tourniquets represents an approximate trapping of 200 ± 80 ml of blood. According to data of Ebert and Stead who suggested that 900 ml of blood is contained at rest in three limbs, the same increment in counts could represent even more (as much as a total of 360 to 400 ± 160 ml of blood). Thus, trapping of a considerable quantity of blood in the periphery is indeed achieved by tourniquets despite the increased sympathetic tone.

**Left Ventricular Function Response to Peripheral Blood Pooling**

The drastic increment in peripheral blood pool achieved by tourniquets accordingly should be reflected by an observable and consistent improvement in LV size and function. Therefore, it is disappointing to see that LV function responds little (and if so, it may be in a negative way) to the substantial peripheral blood pooling produced by rotating tourniquets: in fact, EF decreased, and certainly did not improve, in most of the patients of this study, and stroke volume (and cardiac output) equivalent decreased whereas peripheral vascular resistance increased. Left ventricular equivalents did not universally decrease. Echocardiography likewise failed to show any substantial change in LV diameters and shortening fraction, thus corroborating the radionuclide findings. In a recent echocardiographic study in which tourniquets were applied to the four limbs of normal subjects, the end-diastolic volume decreased but the end-systolic volume did not, so that the shortening-fraction actually decreased. These findings are therefore not inconsistent with those of our study.

It could be argued that a decrease in mean EF from
0.23 to 0.21 is within the range of error of the method. However, duplicate control radionuclide counts were almost identical, the decrease in EF, although modest, was observed in the vast majority of patients (18 of 26), and the EF values returned to the control value after release of tourniquets. Certainly a decrease, even if small, must be biologically significant if it is repeatedly observed in a majority of subjects. Conceptually, then, the decline of EF values and the deterioration of other important parameters observed in this study fail to support the hypothesis that preload reduction by tourniquets leads to significant LV hemodynamic improvement.

The theoretical basis for the hoped-for effect of tourniquets is simple: preload reduction reduces diastolic LV volume and pressure, and shifts the heart leftward and down on the ventricular diastolic pressure-volume curve, thus decreasing pulmonary congestion and myocardial oxygen consumption. However, it appears that the hemodynamic effect of tourniquets is not so simple. First, reduction in preload may in itself reduce stroke volume. Second, pooling blood in the limbs by tourniquets introduces another variable: direct pressure by the tourniquets and the increase in extravascular tissue tumor lead to greater resistance to arterial flow. Therefore, there is afterload increases! Thus, deterioration in the already precarious function of the impaired LV may result from the combination of decreased preload and increased afterload. Thus, our findings explain why previous studies have failed to demonstrate reduced pulmonary artery pressures, despite the satisfying increments in the peripheral blood pool.

Erythrocyte count and isoactivity may be affected by increased sympathetic tone and this could theoretically introduce an artifactual error in LV volume calculations. However, the whole blood radionuclide counts were not substantially changed in our patients by tourniquet application, and thus, the changes in LV volumes are not related to changes in erythrocyte count.

A limitation of this study is that the patients were not in actual acute pulmonary edema. Ethical and technical considerations do not at present permit withholding urgent therapy from patients in acute pulmonary edema, and, theoretically, tourniquets could conceivably improve LV function during the acute stage of pulmonary edema and no longer make any contribution in the recovery phase. However, this seems highly unlikely in view of the demonstrable increase in peripheral vascular resistance and the decrease in LV function in the presence of adequate venous pooling. Therefore, since this study and other data fail to show any improvement in the function of the failing heart, the benefit of tourniquets should still be considered as conjectural and lacking a convincing data base.

ACKNOWLEDGMENTS. We wish to express our appreciation to Hadar Leshem and Osnath Sharoni for the echocardiographic studies.

REFERENCES