The Effects of Transfemoral Catheterization on Blood Flow in the Extremities


SUMMARY

The peripheral hemodynamics of 20 patients were studied 24 hours before and 24 hours following completion of Seldinger transfemoral cardiac catheterization. All patients were catheterized in the right femoral artery and vein, while in 13 an arterial needle was placed in the left femoral artery.

In the catheterized leg, mean calf blood flow and venous capacitance fell while mean calf vascular resistance was increased. The placement of an indwelling arterial needle caused mean calf blood flow to fall with an increase in calf vascular resistance. Venous capacitance was unchanged. The above changes while present 24 hours after catheterization had returned to normal one week later. While no symptoms or signs of limb ischemia occurred, oscillometry showed deterioration in the lower limb pulsation amplitudes in 13 of the 20 patients. No significant changes in peripheral hemodynamics were seen in the limbs, the arteries of which had not been catheterized.

Thus, although all patients were symptomless and free of signs suggestive of ischemia clinically, arterial and venous catheterization and/or the placement of an arterial needle causes significant changes which last at least 24 hours distal to the invasion of the vessels.

Additional Indexing Words:

Arteriography, Calf blood flow, Oscillometry, Seldinger technique, Venous capacitance

With the widespread and increasing use of central cardiac catheterization, often in critically ill patients, vascular changes caused by such interventions have become increasingly recognized. Some studies have suggested that the pulsations of patients are somewhat diminished after catheterization, but generally there is a deficiency of data on the effects of arterial and venous catheterization on peripheral hemodynamics. Other studies have placed the incidence of thrombosis of the vessels of the leg that has been used in central cardiac catheterization in the magnitude of 1 to 8%.

While embolic or thrombotic episodes are likely to cause, at least partially, the diminished peripheral pulses often encountered clinically, it would appear that vasomotor or vasospastic changes due to iatrogenic alterations in limb arterial resistance and venous tone may also play some part. Unfortunately, most studies are retrospective and document only serious symptomatic complications. Thromboembolic accidents are usually detected only by the presence of gross pulse deficits. The recognized fallibility of pulse detection by various observers makes palpation of the pulses an unreliable criterion for assessment of peripheral circulation.

With this problem in mind, we elected to study the peripheral vasculature of 20 patients before and after cardiac catheterization in an attempt to ascertain if changes in arterial resistance and venous tone were produced by cardiac catheterization, how long such changes, if any, lasted, and if the development of new or exacerbation of old stenotic processes as measured by oscillometry could account for all changes in the peripheral hemodynamics following catheterization.

Methods

Twenty consecutive patients without symptoms or signs of peripheral vascular disease underwent peripheral vascular
examination, 16 to 24 hours before cardiac catheterization and again 16 to 24 hours following the completion of catheterization. The protocol and purposes of the investigation were explained to each patient and informed consent was obtained. In seven patients, further determinations of calf blood flow and venous capacitance were made one week after catheterization. Following premedication with secobarbital, 100 mg, P.O. and local anesthesia provided by infiltration of the surrounding tissue with 2 ml of 1% lidocaine, all patients were catheterized in the right femoral artery and vein using the Seldinger technique. Number seven French teflon catheters (Cook and Co., Inc.) were used for pressure measurements and ventriculograms catheterization and number 7.2 French polyethylene catheters were used for coronary arteriography. In 13 patients an arterial needle was placed in the left femoral artery after lidocaine infiltration and remained indwelling throughout the length of the procedure. In seven patients there was no intervention in the left groin. Pressure was applied over puncture sites for 10 min at which time all bleeding had usually stopped, following which a dry dressing was applied. In several patients, pressure was required for a further 20 min following which a dry dressing was applied.

Peripheral vascular studies on the lower limbs were performed supine in a quiet room at a temperature of 68°F using a four limb pneumatic plethysmograph. The patients were in a postictal state.

After resting for 20 min, oscillometry was performed above the elbow, at the wrist, upper thigh, above and below the knee, and at the ankle using the pneumatic plethysmograph. The accepted reading was the greatest pulsation amplitude in a given area following 10 mm Hg decrements of arterial blood pressure determined by a sphygmomanometer.

After resting for a further 20 min, determinations of calf blood flow and venous capacitance were undertaken to insure a relatively steady state. When a steady state was considered to be present, three successive measurements of calf blood flow and venous capacitance were undertaken, averaged, and utilized as the measurement.

A venous occlusion plethysmographic technique was used for the measurement of calf blood flow and venous capacitance. The plethysmographic cuff was placed around the midcalf to measure calf blood flow. A second cuff connected to a cylinder of compressed air was placed around the leg just above the knee which allowed an immediate pressure increase by sudden inflation to 30 mm Hg. Calf blood flow was calculated from the change in limb circumference during venous occlusion and expressed in ml per 100 cc tissue per minute. During the recording, blood flow to the feet was occluded by the use of a third cuff inflated to suprasystolic levels of pressure.

Venous capacitance was determined by the equilibration method as described elsewhere. The venous occlusion cuff was suddenly inflated to 30 mm Hg and venous pressure and limb circumference were permitted to equilibrate for 2 min. The time of the capacitance results were calculated from the increments, if any, in volume and pressure that had taken place during the full 2 min period. During measurements the legs were elevated so that the leg was above the level of the heart.

Vascular resistance was defined as the mean arterial blood pressure measured in the arm by the auscultatory method divided by the calf blood flow. Mean arterial blood pressure was calculated by using the formula: Mean BP = Diastolic BP + 1/3 (Systolic BP - Diastolic BP).

The patients studied were of mean age 49 years; they were predominantly male and the duration of the catheterization study varied from 34 to 176 minutes (average, 93 minutes). Coronary artery disease was the most common diagnosis and occurred in 11 of the 20 patients (table 1).

Statistical comparison of the data was obtained by analysis of the change from the control level by Student’s t-test for paired replicates.

Results

During the catheterization no marked differences in the temperatures of the patients’ feet were seen. The postcatheterization course of all 20 patients was without incident or complications. In particular, no patient described any symptoms of or developed any signs of peripheral ischemia and/or thrombosis or embolism in his legs.

Oscillometry

Central cardiac catheterization caused a marked deterioration of oscillometric readings in the lower limbs. Twenty-four hours after catheterization, five patients appeared to have developed possible new stenotic processes in their arterial trees — the most common site being in the popliteal area. Five additional patients showed a definite worsening of lesions already present while three patients showed both the development of probable new stenotic processes and the worsening of lesions already present. In the remaining seven patients there were no changes in the lower limbs’ pulsation amplitudes. In the upper limbs, which had remained uninvaded, oscillometric examinations were essentially unaltered.

Catheterized Leg

The effect of arterial and venous catheterization in the right leg of all 20 patients was to reduce the mean calf blood flow from 1.15 to 0.82 ml/100cc/min (P < 0.0005) (fig. 1). Conversely mean calf vascular resistance increased from 103 to 172 mm Hg/ml/100 cc/min (P < 0.0025) (fig. 2). Mean calf venous

Table 1

<table>
<thead>
<tr>
<th>Diagnosis Obtained Upon Catheterization of Twenty Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coronary artery disease</td>
</tr>
<tr>
<td>Aortic regurgitation</td>
</tr>
<tr>
<td>Mitral stenosis</td>
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<tr>
<td>Mitral stenosis and regurgitation</td>
</tr>
<tr>
<td>Aortic stenosis</td>
</tr>
<tr>
<td>Atrial septal defect</td>
</tr>
<tr>
<td>Hyperventilation syndrome</td>
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<tr>
<td>Total</td>
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Uninvaded Limb

In seven patients the left groin remained intact, being uninvaded by either catheterization or by the placement of an arterial needle. In these limbs, mean calf blood flow, mean calf vascular resistance, and mean calf venous capacitance remained unchanged (fig. 5).

Follow-Up Study

In seven patients, studies were undertaken both one day before catheterization and one day and one week after catheterization. In these patients mean calf blood flow, vascular resistance, and venous capacitance which had undergone changes as described above returned to levels that were not significantly different from the precatheterization control levels (fig. 6). This return to normal levels was seen in both the limbs which had been catheterized and those invaded by the placement of an arterial needle.

Discussion

The results of this study demonstrated that cardiac catheterization causes marked changes in peripheral hemodynamics up to 24 hours following completion of central catheterization in patients, all of whom remained clinically free of vascular ischemia.

Thus, arterial and venous catheterization caused mean calf blood flow and venous capacitance to fall while mean vascular resistance in the limb was increased 24 hours following completion of the procedure. The placement of an indwelling arterial needle also reduced mean calf blood flow and caused mean calf vascular resistance to increase at 24 hours. In those limbs which were not invaded no significant differences were seen in any of these measurements. Since no significant differences were apparent in the
intact limbs, this suggests the changes must be the result of the invasion of the limb rather than the mental anguish or some other factor associated with the procedure.

However, psychological factors attendant upon cardiac catheterization may be operative, at least in part. The precatheterization control measurements of peripheral hemodynamics are lower than what one would normally expect. This probably results from a number of factors including 1) the low temperature of the area in which the studies were performed (68°F); 2) the serious nature of the underlying coronary artery disease, valvular heart disease, low cardiac output, and sometimes attendant heart failure which is known to increase venous tone and diminish peripheral blood flow; 3) the presence of peripheral vascular disease which was shown by oscillometry to be present in over one-half of the patients; and 4) the presence of possible vasospasm which may result, in part, from the mental stress of knowing invasion of one's heart is imminent.

Oscillometric study shows that in 13 of the 20 patients there was deterioration of their peripheral pulses severe enough to warrant the assumption that 1) either a new stenotic lesion had developed; 2) that a stenotic lesion already present had gotten worse, or 3) both. Since the main area for the development of new lesions was in the popliteal area, it suggests the possibility that at least in some patients a stenotic process possibly due to thrombosis or embolism had been carried downstream from the femoral area and lodged in this area.

Although mean calf blood flow and venous capacitance diminished in 16 patients while mean calf vascular resistance was found to increase in 18 of the 20 patients, seven patients showed no change occurred by oscillometry. Thus factors other than embolism or thrombosis must be operating.

In previous investigations emphasis has been placed upon puncture technique, length of the catheter, and the volume of contrast medium used, the duration of the examination, the degree of arterial compression after examination, the age of the patient, the degree of atherosclerosis and heart disease present, and the presence of arterial spasm as the cause of changes in peripheral arteries after catheterization. While most of these investigators have suggested thromboembolic events as the major mechanism in diminished limb blood flow following catheterization, the present study suggests that because of the disparity of the diminished blood flow and increased venous tone seen in some patients with a normal oscillometric examination vasospasm may be a causative mechanism as well.

Since the follow-up studies in seven patients showed that mean calf blood flow, vascular resistance, and venous capacitance returned to near normal precatheterization levels within one week of catheterization (fig. 6), it appears that the changes induced by catheterization causing alterations of venous and arterial tone are probably in the main short-lived.

Previously, vascular spasm has not been regarded as an important cause of diminished peripheral
hemodynamics following catheterization since it was thought to occur mainly among young individuals. A central question raised by this study of older patients which could bear on future therapeutic measures, is whether or not the described changes are of a mechanical or neurogenic origin. If there is a neurogenic cause, pharmacologic interventions in the catheterized limb might abolish the reaction. These same intervention studies could assess the part played by local autonomic imbalance, disruption of sympathetic fibers in the vascular sheath, the reflex responses to embolic phenomenon, and the effect of trauma upon its local blood vessels could be ascertained.

In opposition to the possible neurogenic changes in the postcatheterization patient, recent studies have indicated mechanisms by which thromboembolic phenomenon can occur in such patients. Formanek, Frech, and Amplatz have shown deposition of thrombotic material on catheters in over half of 93 patients subjected to diagnostic catheterization. These workers suggested that postcatheterization thrombosis occurs in many of the catheterized vessels due to fibrin collection on the outside of the catheters. These conclusions are supported by similar work of Jacobsson and his colleagues. As the catheter is withdrawn, the adherent material is stripped from the shaft at the arterial puncture site, piles up, and may occlude the arterial lumen. Thus although the catheter emerges free of thrombi, the thrombus may remain attached at the puncture site, a fact that accounts for the common subsequent finding of thrombus at the time of arterial cannulization for heart surgery, or for embolization to the peripheral vessels. In the present study no symptoms or signs of clinical peripheral embolism were seen. In the study of Formanek and his colleagues, a definite time relationship between the thrombus formation and the duration of the catheterization procedure was noted; the longer the catheterization, the greater the occurrence of thrombus formation. However, in the present study there appears to be no significant relationship between the reduction in calf blood flow in the catheterized limb and the length of time of the procedure. Since over half of the patients studied were suffering from peripheral vascular disease, it would seem probable that the invasion of these vessels could result also in the displacement of small emboli and hence changing of flow patterns in the limb. However, Jacobsson and Schlossman thought that femoral arteriosclerosis was probably less important a factor than one might expect.

Fogarty and Krippaehne have shown that in some instances an intimal tear and resultant flap can occur causing gradual occlusion of a vessel and change in limb blood flow.

Since the changes we found occurred frequently in asymptomatic patients following the completion of catheterization or the placement of an arterial needle, it provides an additional constraint on the subjection of patients to such studies. It also implies that if central catheterization is to be performed, it should be done in such a way that peripheral changes as well as central complications are minimized.

Efforts to minimize the incidence of such alterations should include: 1) proper preparations of needles, dilators, and catheter tips; 2) prevention of arteriospasm by adequate infiltration of lidocaine and by use of catheters with high surface lubricity such as those made of teflon; 3) careful flushing of catheters to minimize clotting; 4) the use of the smallest catheter size consistent with a satisfactory study; 5) avoidance of extensive manipulation or repeated catheter changes which might traumatize the vessels; 6) avoidance of excessive pressure in controlling bleeding; 7) the aggressive management of heart failure or dehydration in the precatheterization period; and 8) recognition that patients with valvular heart disease often suffer a special disposition to peripheral vascular complications. Finally, while an obvious solution to the problem of the stripping of thrombi from the catheter by the arterial wall may be a heparin-impregnated plastic which prevents initial deposition of thrombus material, unfortunately at the present time most of the described techniques used to achieve this end alter the consistency of the plastic material used and are unacceptable.

References


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