Complications of Percutaneous Radial-artery Cannulation: An Objective Prospective Study in Man

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One hundred and five percutaneous radial-artery cannulations were prospectively studied in 100 patients. The patients were examined daily for complications, utilizing physical examination and Doppler flow measurements. Forty of the 105 cannulations (38 per cent) resulted in radial-artery thrombosis. Arterial occlusion occurred in 25 per cent of 43 vessels cannulated for less than 20 hours, in 50 per cent of 40 cannulations lasting 20 to 40 hours, and in 41 per cent of 22 cannulations lasting 40 to 144 hours. Radial arterial pulses, produced by retrograde ulnar-artery flow, were palpable distal to 73 per cent of the radial-artery thrombi, and Doppler flow signals were audible distal to 90 per cent of the thrombi.

Arterial occlusion was found at the time of decannulation in only 42.5 per cent of those vessels which eventually thrombosed. An additional 30 per cent of the total number of thrombi developed within 24 hours of decannulation. However, 27.5 per cent first occurred later than one day after decannulation. All of the 20 thrombosed vessels which were followed during return visits recanalized. The longest time taken for recanalization was 75 days. No major ischemic complication was observed in any patient, despite the high incidence of temporary thrombosis. Histologic specimens from three cannulated radial arteries were obtained at subsequent postmortem examinations; photomicrographs of these specimens showed a pathologic picture consistent with the clinical impression. (Key words: Percutaneous radial-artery cannulation; Thrombosis; Retrograde ulnar-artery flow; Recanalization.)

Several studies have shown the increasingly common procedure of percutaneous radial-artery cannulation to be relatively free of major complications. Thrombosis of the radial artery has been reported to have a 0–60 per cent incidence,1–4 and necrosis of the skin over the tip of the cannula occurs in 0.45 per cent of the cases.5 Hematoma formation and ecchymosis follow approximately 50 per cent of radial-artery cannulations.4 Isolated complications, such as peripheral embolization,6 Osler node formation,7 and discomfort in the hand related to positioning,8 have also been reported.

This prospective study is the first to examine radial-artery function daily for at least ten days following prolonged percutaneous cannulation, and to utilize both physical examination and Doppler flow measurements. Perhaps for these reasons, we observed an incidence of radial-artery thrombosis considerably higher than those reported in most previous studies.

Material and Methods

One hundred and five consecutive radial-artery cannulations were studied in 100 patients undergoing general anesthesia for cardiothoracic or major vascular surgery, or neurosurgery.

All patients were examined preoperatively for patency of the ulnar artery by a modification of Allen’s test for arterial patency.9 While the patient made a tight fist, both radial and ulnar arteries were occluded with the tips of the examiner’s fingers. The patient then opened his hand while the examiner released the pressure over the ulnar artery. Ulnar-artery patency was indicated by the immediate appearance of an erythematous blush over the entire palmar surface of the hand and fingers. Ulnar-artery filling was judged slow when 7 to 15 seconds passed before the blush was apparent. Six radial arteries were cannulated despite slow ulnar-artery filling. Since none of these

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patients developed radial-artery thrombosis following decannulation, no data concerning the safety of radial-artery puncture in the presence of an equivocal Allen's test were obtained. When the hand remained blanched for longer than 15 seconds, ulnar flow was judged inadequate and radial cannulation was not done in that extremity.

Percutaneous radial-artery cannulation was performed with the wrist dorsiflexed at a 60-degree angle. The supinated hand and forearm were affixed to an arm board and the wrist dorsiflexed over a roll of tape or paper towels. The radial artery was palpated for a 3-inch distance proximal to the base of the thumb and a line drawn over the artery with a skin-marking pen. Following preparation of the skin with iodine and alcohol, a cutaneous wheal of 1 per cent lidocaine was made. The cannula was inserted through the wheal and advanced subcutaneously along the course of the radial artery at a 10–15 degree angle to the surface of the skin. A free spurt of bright red blood indicated entry into the vessel. With the stylet held fixed, the cannula was advanced up the arterial lumen for approximately 4 cm. The stylet was then withdrawn and the cannula firmly connected to flexible tubing leading to a strain gauge.

Occasionally the cannula and stylet would pierce the posterior wall of the vessel. When this was suspected, the stylet was removed and the cannula withdrawn until free flow of arterial blood was seen. The cannula was then advanced without replacing the stylet. Piercing the posterior wall of the vessel was scored as 1+ trauma. Two or more arterial punctures with inability to advance the cannula successfully were tabulated as 2+, 3+, or 4+ trauma, depending on whether two, three, or four attempts were necessary.

The skin over the volar surface of the wrist and the arterial cannula itself were painted with tincture of benzoin. A dab of Neosporin bacitracin–neomycin ointment was applied to the puncture site, and the entire area was covered with a small steri-drape. The angle of wrist dorsiflexion was then reduced to 30–45 degrees for the duration of the cannulation.

All arterial cannulae were flushed with physiologic saline solution containing 2 units of heparin per ml. Intermittent 5-ml injections of flush solution were made whenever damping of the arterial trace was noted. In some cases, a continuous slow infusion of 4 ml of flush solution per hour from a Sage pump was used to keep the cannula patent. Total volumes of heparin solution infused were recorded.

While the cannulae were in place, the arteries were examined for the presence of pulse and adequate distal perfusion, evidence of ecchymosis or erythema at the site of puncture or at the cannula tip, local discomfort, and signs of peripheral embolization.

After decannulation, radial-artery patency was evaluated both by the modified Allen's

18 or 20 Argyle Polypropylene catheters.
test,* as described above, and with a Doppler Model 303 flowmeter (Parks Electronics), using a flat probe with a 15-degree sensor angle. Radial-artery occlusion was considered present by Allen’s test when the patient’s hand remained blanched longer than 15 seconds after release of pressure over the radial artery. In each case, this was confirmed by absence of the Doppler flow signal along part of the course of the radial artery.

No subsequent radial-artery puncture was performed in any cannulated vessel. All radial arteries were inspected at least five days a week for the duration of the patient’s hospitalization, and 20 of the 40 patients who developed radial-artery thrombi were followed on an outpatient basis until radial-artery flow was re-established.

Three patients who had radial-artery cannulation subsequently were examined post mortem, and of these, two had clinical evidence of thrombosis precisely where indicated by Doppler examination. Histologic specimens from these radial arteries were examined.

Results

Temporary thrombosis was demonstrated in 40 radial arteries, one each in 40 of the 100 patients, or in 38.1 per cent of the cannulations in this study. Figure 1 shows a postmortem specimen of a radial-artery thrombus which developed following cannulation. Note that the thrombus lies within the vessel at the site previously occupied by the cannula.

A Doppler signal was audible distal to the area of occlusion in 92.5 per cent of the 40 thrombosed vessels, and a distal pulse was palpable in 72.5 per cent of the occluded vessels. The pulse distal to the thrombus represented collateral circulation from the ulnar artery via the palmar arterial arches. Such a pulse disappeared when the ulnar artery was occluded. Figure 2 demonstrates this phenomenon and shows the areas of radial-artery thrombosis outlined with the Doppler meter.

The incidence of thrombosis was related to the duration of cannulation. Figure 3 shows the cumulative incidence of thrombosis as a
function of time. Cannulae removed in the recovery room (less than 20 hours of cannulation) induced a 25 per cent incidence of thrombosis, while cannulations lasting into the first postoperative day in the I.C.U. (20 to 40 hours of cannulation) resulted in a 50 per cent incidence of occlusion. Cannulae left in place longer than 40 hours induced a 43 per cent thrombosis rate.

In addition to total arterial occlusion, six cannulations resulted in narrowing of the artery, as evidenced by decreased intensity of the Doppler flow signal and slow radial filling (7 to 15 seconds versus less than 5 seconds pre-cannulation). In all such cases, these findings cleared within seven days, resulting in normal Doppler flow signals and brisk radial-artery filling demonstrable by Allen’s test.

No difference in incidence of thrombosis could be related to the patients’ ages, catheter size, degree of trauma during cannulation, volume of flush solution infused, or type of flush mechanism employed.

An unexpected finding was that many radial arteries did not thrombose until several days following decannulation (see fig. 4). Seventeen of the 40 post-cannulation thrombi were present immediately after decannulation, and 12 more thrombi developed during the first 24 hours. However, an additional 11 thrombi developed more than one day after decannulation. All but three of the thrombi became apparent within seven days of decannulation.

Eight radial arteries developed spasm at the time of decannulation (absent Doppler flow signal and no radial-artery filling), but were found to have normal filling and strong Doppler flow signals the following day. These eight were not counted as arteries that had thrombosed.

Most patients with radial-artery thrombi left the hospital before recanalization occurred, and half of these were lost to follow up. The remaining 20 were seen on subsequent outpatient visits until radial-artery flow was re-established. Figure 5 shows the time courses of recanalization in the 20 vessels in these patients, the longest period being 75 days. Diminution in thrombus size was followed with the Doppler meter. Allen’s test always showed intact radial-artery flow two to four days after Doppler examination showed apparent recanalization.

Of the 40 patients who developed radial-artery occlusion, four had some distal vascular insufficiency in the form of a pale, cold thenar eminence. This occurred despite the demonstration of brisk ulnar-artery flow by Allen’s test prior to cannulation. In each of these cases there was no Doppler signal distal to the site of cannulation, indicating that the radial artery was an end-artery to the thumb and thenar area. Two other patients each had a cold, purple, tender distal index finger, indicating peripheral embolization. Both types of findings in each instance cleared within seven
days, and in each case increasing ulnar collateral circulation demonstrable by Doppler measurements over the digital arteries and palmar arterial arches accompanied the clinical improvement. No patient whose radial artery was patent by Doppler examination developed clinical signs of vascular insufficiency.

In one case, necrosis of the skin measuring 2 × 4 cm developed over the tip of a cannula that had been in place for 144 hours. This necrosis developed after decannulation and despite the fact that there was no sign of cutaneous damage at the time of decannulation.

Complications are summarized in table 1. There were 14 instances of 1–2-cm nontender purpuric skin lesions which developed over the cannula tip several days after decannulation. We believe these represent early signs of infarction of the skin, but all resolved within three to five days. Eighteen arteries were tender after decannulation, usually for periods of one to three days. The incidences of tenderness in patent and occluded vessels were identical. Ecchymoses and minor hematomata occurred after 88 of the 105 cannulations. These became apparent one to two days following decannulation and persisted for seven to 14 days. None was symptomatic. There was no instance of numbness of the hand after cannulation, and no puncture site became infected.

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**Fig. 4.** Times when arterial occlusion was first noticed. The bar at day 0 indicates those patients with occlusion at the time of decannulation. Solid bars represent patients who had intact radial artery flow at the time of hospital discharge, but were found to have occlusion on subsequent outpatient visits.

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**Fig. 5.** Time courses of recanalization in 20 patients whose arteries had become thrombosed. The shortest duration of arterial occlusion was three days. Six patients recanalized thrombi between days 4 and 9 after occlusion occurred. All arteries were patent by day 75.
Figures 6, 7, and 8 are photomicrographs of cannulated vessels obtained at autopsy. Figure 6 shows residual changes 20 days after radial-artery cannulation lasting six hours. Even in the absence of clinical evidence of thrombus formation, intimal proliferation and medial scarring were present, with lymphocytic infiltrates and giant-cell formation. Figure 7 shows a totally occluded vessel containing an organizing thrombus which developed after 32 hours of cannulation. Figure 8 shows a 2-week-old recanalized thrombus which had originally caused total obstruction of flow for nine days after 24 hours of cannulation.

Discussion

Percutaneous radial-artery cannulation has been shown to have fewer vascular complications than the Seldinger technique for catheterization of the brachial and femoral arteries. We report an incidence of thrombosis following percutaneous radial-artery cannulation considerably higher than those found in most other studies. Hasse et al., using brachial-artery angiography, observed a 60 per cent incidence of radial-artery occlusion in 28 patients following percutaneous cannulation. Twenty-three of their patients were cannulated for longer than 24 hours, and thus were in the same category of patients in which we found the highest incidence of thrombosis. It is possible that they may have interpreted temporary post-decannulation arterial spasm as thrombosis, since repeated angiograms were not done, and we observed eight patients who had arterial occlusion at the time of decannulation but whose radial arteries were patent the following day.

Brown et al., also reported results similar to ours, i.e., a 20 per cent incidence of "diminished radial artery pulsation" following cannulation lasting less than 24 hours, compared with our 25 per cent incidence of thrombosis after cannulation lasting 20 hours or less. Their study was discontinued three days after removal of cannulae, while we have found that thrombi continue to form in some cases for longer than two weeks.

Although survey plethysmography has been reported to be of use in the evaluation of arterial occlusion following Seldinger catheterization, studies to our knowledge, no one has previously reported using both Doppler examination and Allen's test to verify arterial flow following cannulation. In most previous studies, radial-artery patency was tested only by palpating for a pulse distal to the site of cannulation. In our studies, 29 of the 40 thrombosed radial arteries had such a palpable pulse. Previous investigators may have misinterpreted the presence of such a pulse as indicating radial-artery patency, whereas in fact it reflects collateral flow from the ulnar artery. Hasse et al. document the collateral circulation to the hand, including angiographic evidence of retrograde filling of the radial artery distal to an area of thrombosis.

The arterial thrombosis appears to be caused by changes in the integrity of the vessel wall related to the presence of the cannula. Cannulae in place for short periods and removed in the recovery room induced significantly fewer thrombi than cannulae which remained in place for a day or longer. Cannulations lasting an intermediate length of time (20 to 40 hours) were used primarily in patients undergoing open-heart surgery, many of whom were awake and active on the first postoperative day; these patients may well have induced more arterial trauma than those patients whose cannulae were removed on the day of surgery. The longest cannulations (longer than 40

<table>
<thead>
<tr>
<th>Number of Vessels Cannulated</th>
<th>0-20 Hours</th>
<th>20-40 Hours</th>
<th>40-144 Hours</th>
<th>All Cannulae</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vessels thrombosed (per cent)</td>
<td>25</td>
<td>50</td>
<td>41</td>
<td>38.1</td>
</tr>
<tr>
<td>Ischemia of the hand (per cent)</td>
<td>4</td>
<td>2</td>
<td>4.5</td>
<td>3.8</td>
</tr>
<tr>
<td>Emboli to fingers (per cent)</td>
<td>4.7</td>
<td>0</td>
<td>0</td>
<td>1.9</td>
</tr>
<tr>
<td>Purpura at the cannula tip (per cent)</td>
<td>14</td>
<td>12.9</td>
<td>8.7</td>
<td>13.3</td>
</tr>
<tr>
<td>Hematoma or ecchymoses (per cent)</td>
<td>76.8</td>
<td>95</td>
<td>77.3</td>
<td>83.8</td>
</tr>
<tr>
<td>Tenderness at cannulation site (per cent)</td>
<td>18.6</td>
<td>20</td>
<td>9</td>
<td>17.1</td>
</tr>
</tbody>
</table>
hours) were mainly in critically ill and less active patients, perhaps explaining a somewhat lower incidence (41 per cent) of thrombosis, compared with cannulations lasting 20 to 40 hours (50 per cent).

Hasse et al.² reported that the incidence of thrombosis in nine of their patients less than 40 years of age was 33½ per cent, whereas in 19 patients more than 40 years old the incidence was 75 per cent. They did not correlate these findings with the duration of cannulation. Our own results showed a 39 per cent incidence of occlusion in 18 patients less than 40 years old and a 38 per cent incidence of occlusion in 87 patients more than 40 years old. We feel, therefore, that the age of the patient is not a contributing factor in the overall incidence of post-cannulation arterial thrombosis.

Microscopic sections of cannulated vessels showed intimal damage and proliferation. Marked vessel-wall scarring was found after
only six hours of cannulation. The denuded vascular lining, subsequent proliferated endothelium, and arterial constriction at the time of decannulation all probably combine to contribute to thrombus formation at the site of the cannula. While Brown et al.4 found no difference between the incidences of decreased radial pulsation in arteries cannulated with polyvinylchloride cannulae and those cannulated with teflon cannulae, a prospective, controlled study of this issue remains to be undertaken. Likewise, no controlled, prospective study of 18- vs. 20-gauge cannulae has yet been undertaken.

Despite the considerable incidence of radial-artery thrombosis, cannulation resulted in remarkably few vascular complications because of abundant collateral flow to the hand. Isolated reports of peripheral embolic phenomena and ischemia may well result from dislodgment and distal passage of a portion of the thrombus to an end-artery, or inadequate ulnar collateral flow in cases where ulnar patency is not tested prior to cannulation. When ulnar patency is being observed by Allen’s test, particular attention should be directed toward demonstrating circulation to the thenar area.

Angiographic studies by Hasse et al.2 demonstrate that the radial artery is frequently an end-artery to the thenar area. In our four patients with evidence of ischemia of the thenar eminence, the absence of Doppler flow signal distal to the area of occlusion indicated that this was the case. As the signs and symptoms of ischemia cleared, collateral ulnar filling resulted in the appearance of Doppler signals distal to the radial-artery thrombus. Two of these patients were each followed until recanalization of the thrombus was complete. They took the longest periods for this to occur: 68 and 75 days, respectively.

In summary, using Doppler flow measurements and physical examination following radial-artery cannulation, we have found a considerable incidence of temporary radial-artery thrombosis. This incidence increased with increasing duration of cannulation. The onset of thrombosis was often delayed for some days following decannulation. Despite demonstrated ulnar flow, there was a 10 per cent incidence of temporary minor vascular insufficiency in those patients who developed radial-artery thrombi. In these 105 percutaneous arterial cannulations, there was no major vascular complication. All cases of occlusion which were followed eventually showed recanalization.

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References