Scanning electron micrographic study of vascular lesions caused by microvascular needles and suture

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The authors studied the damage to blood vessels 1 to 2 mm in diameter caused by the most commonly used types of microvascular needle. Excluding variables introduced by anastomosis, the study focused specifically on lesions attributable only to the needle and suture. Scanning electron microscopy revealed four distinct types of lesion. A theory is proposed to explain the mechanisms whereby these lesions were produced, and a needle design is recommended that may aid in minimizing vascular trauma.

KEY WORDS • microsurgery • vascular needle • vascular lesion • electron microscopy • vascular suture

MICROSURGICAL techniques are used to reconstruct vessels, revascularize areas of the brain, and to tackle previously unmanageable surgical problems. These surgical improvements are in large measure related to technical advances that have allowed the development of finer operating instruments, needles, and suture material. With the increasing use of microvascular surgery in a number of clinical fields, there is justification for a continued effort to improve upon microinstrumentation and to search for the ideal needle and suture.

We undertook a study to demonstrate the intimal surgical damage to arteries 1 to 2 mm in diameter, analyzing the role of different needles and suture material in the formation of the lesions. Variables that contribute to the success or failure of microsurgical vascular repair, such as clip application, loose adventitia removal, transection of the artery, irritation of the arterial ends, and handling of the arterial walls, were eliminated from the study. Instead we concentrated upon the damage attributable only to the needle and suture.

Materials and Methods

The common carotid artery of the rat was chosen as the experimental model. One hundred Sprague-Dawley rats weighing 240 to 300 gm were anesthetized with intraperitoneal chloral hydrate solution, 0.75 mg/gm. The animals were then secured in the supine position, and a midline incision was made in the neck. A tracheostomy was performed, and both carotid arteries were exposed. The operating microscope and microsurgical instruments were used throughout the procedure, assuring that the vessel was handled as little as possible.

The needle was secured with No. 5 jeweler's forceps and passed through the vessel using the complete arc of the needle (Fig. 1). A loose tie was placed in the suture after a complete passage of the needle through the vessel. Each needle was used only once in order to avoid dulling of the tip. The blood vessel was never picked up or manipulated except by the single passage of the needle. The wound was closed in two layers, and the animal remained isolated until the time of sacrifice.

A period of 15 minutes to 24 hours elapsed between the placement of the last throw in the suture and the sacrifice of the animal. The animal was reanesthetized and fixation was achieved by perfusion with 3% glutaraldehyde solution, buffered in sodium cacodylate pH 7.4 at 38°C, via a direct cardiac cannula. In 25 animals, a needle was incompletely passed into the opposite carotid artery immediately before fixation, so that the actual passage of the needle could be studied.

After perfusion, the entire segment of artery was removed and placed in buffered fixative at 4°C for 1 to 4 days. At this point, the segment was dehydrated in alcohol, critical-point dried with liquid CO₂ as the immersion medium, coated with gold-palladium, and examined under the scanning electron microscope. Photographs were taken of all specimens at both × 200
Lesions produced by vascular needles

Fig. 1. Schematic representation of the surgical technique.

and × 500 for comparison. Specimens studied consisted of controls, blood vessels with the suture material in place, and those where the needle had perforated the vessel wall and remained in place. The needles used in this study are described in Fig. 2.

Results

The endothelial surface of the normal rat carotid artery demonstrated a clean and smooth contour, except for a few gentle undulating corrugations. The endothelial cell nuclear bulges were evenly spaced, and no endothelial deficits or platelet aggregations existed.\textsuperscript{12}

At the suture site, four distinct endothelial lesions were identified (Fig. 3): 1) a large intimal hole, attributed to the site of actual needle perforation; 2) a variable length of intimal tear continuous with the needle hole, but distinguishable from it; 3) in addition to the direct intimal damage, a variable number of patches of denuded subendothelium surrounding the needle hole in a satellite arrangement and extending

Fig. 2. Diameter and shape of the needles and size of the suture used in this study.

FIG. 1. Schematic representation of the surgical technique.

FIG. 3. Scanning electron micrographs of the intimal surface showing sites of needle entrance. Four distinct lesions were produced simply by passage of the needle: 1) the large intimal hole; 2) endothelial tear continuous with the needle hole; 3) associated satellite endothelial defects; and 4) variable amounts of platelet aggregation. \( \times 500 \).
FIG. 4. Scanning electron micrograph of the intimal surface showing the site of needle puncture immediately after needle passage. All four vascular lesions can be seen (see Fig. 3 for description). The primary hole created by the needle is approximately twice the needle diameter. × 500.

The findings were consistent if the animal had been sacrificed 15 minutes or 24 hours after the vascular insult.

The size of the hole in the vessel caused by the needle perforation was consistently approximately twice the diameter of the needle (Fig. 4). This was true regardless of the type or style of the needle. As expected, needles of larger diameter produced holes larger than the smaller needles. For instance, the 50-μm diameter needle produced a hole 50% to 60% the size of that produced by the 100-μm diameter needle.

The amount of satellite endothelial tears and the size of the areas denuded appeared to be quite variable, even between different specimens pierced by the same diameter needle. It was thought that no justifiable comparison could be made between the different needles. Large associated tears were seen with all needles studied. By scanning the intimal surface of the vessels, these tears were more commonly observed at the site of needle entrance into the lumen than at the exit site.

Similar to the intimal tears, the extent of platelet aggregation and adhesion of white blood cells was quite variable and usually extensive with all needles used. Another interesting observation was made with respect to the entrance versus the exit site (Fig. 5).

FIG. 5. Scanning electron micrograph of the intimal surface showing both the needle entrance site (a) and exit site (b). × 200.
Lesions produced by vascular needles

Consistently, gross and wide areas of platelet aggregation were seen at the entry site of the vessel, that is, the point at which the needle had passed from outside the vessel to the inside. Although the area of exit showed some platelet aggregation, this was clearly less than at the site of entry (Fig. 6).

We have attempted to theorize the pathogenesis of the endothelial lesions in the following manner. All needles currently used in microsurgery have the characteristic blunt and tapered point. This does not allow the needle to cut through the vessel, but dictates that the needle be pushed through the vessel (which is often a problem with the muscular superficial temporal artery). This dull, tapered needle indents and stretches the vessel wall and requires great force to achieve penetration. In the process of applying this concentrated force, a large area of intima is ruptured in association with small tears from overstretched sites, thus causing a wide area of exposed subendothelium (Fig. 7). This exposed subendothelium then initiates platelet aggregation. In addition to the hole itself, the exposed area is often five to six times the width of the needle. Taking into account the fact that endothelial damage is required for platelet aggregation, an estimate of this damage can be as much as 20 times the needle diameter.

It is not known why the exit site does not demonstrate a similarly extensive complex of associated lesions in comparison to that observed at the entry site. Perhaps as the needles exit from the vessel lumen, first the endothelium is perforated by direct contact with the needle tip and then the vessel is stretched as the needle is forced through the remainder of the vessel.

As expected, the larger sized needles created larger holes and wider areas of associated secondary intimal damage during perforation. In reviewing the many puncture sites, it was estimated that the hole created by the needle was approximately twice the diameter of the needle. In addition, these primary and secondary endothelial lesions produced widespread aggregation of platelets and adhesions of white blood cells. All these lesions were more marked at the site of entrance than at the site of exit.

Discussion

The patency rate for extracranial-intracranial anastomosis is estimated by some to be as high as 95%. However, it is not simply patency that is essential in this procedure, but rather an increased cerebral blood flow to a poorly perfused region of brain. Poiseuille's Law concerning hydrodynamics in rigid tubes suggests that a small change in luminal size could significantly alter flow through these 1- to 2-mm blood vessels.

Disruption of the normal endothelium initiates a complicated tissue response that involves platelets and smooth muscle, as well as the endothelium itself. Injury to the endothelium causes the platelets to adhere immediately to the subendothelial tissue and quickly lose their granules. These granules contain vasoactive chemicals, such as serotonin and lysosomal enzymes, that may alter flow in the already diseased vessels downstream from the anastomosis. In addi-

![Fig. 6. Scanning electron micrographic demonstration of the needle exit site showing significant diminution in pathological changes compared to Fig. 3. × 475.](image)

![Fig. 7. Schematic representation of the events that take place when the blunt-tipped needle is passed through the vessel wall, with subsequent formation of multiple intimal and endothelial lesions. A: Hole produced by actual needle perforation; B: intimal tear continuous with the needle hole; and C: satellite patches of denuded subendothelium. See text for details.](image)
tion, this endothelial damage causes a cellular proliferation of smooth muscle cells that increases the thickness of the muscular coat by five to 15 layers. Finally, the endothelium itself proliferates to as much as eight times its normal one-layer thickness.

Previous studies have focused on the histopathological changes that occur in anastomosed blood vessels and at the site of clamp application. In these studies, almost complete denuding of the intima was found around the anastomosis site. Acutely, it was coated with platelets, and this event was found not to be inhibited by heparin. In approximately 21 days, the specimens showed areas that were covered with eight layers of cells as opposed to the normal single layer. Also, many fibroblasts were present, indicating diffuse scarring. Medial necrosis of the vessel usually extended as much as 2 mm in either direction from the anastomosis site. Luminal compromise in this study was estimated at approximately 13% in the first 24 to 48 hours, and approximately 19% at 10 days, due to the factors outlined above.

Other studies have shown that patency does not guarantee an increase in regional cerebral blood flow (rCBF). In all cases, the objective is to increase rCBF maximally as soon as possible postoperatively. This can be especially important when superficial temporal-middle cerebral artery anastomosis is performed in conjunction with tumor removal or the trapping of a giant aneurysm. In order to maximally increase rCBF after extracranial-intracranial bypass, one should minimize the amount of luminal compromise by thrombus, scarring, spasm, and edema in the vessel wall.

In the present study, we attempted to eliminate the many variables present during an anastomosis and to limit our observations to the changes that take place in the vessel using the type of suture as the variable. This allowed us to observe the trauma caused by the suture alone, and thus make some comparison of the different types of suture available. It was found that the blunt-tipped needles presently in use create four distinct lesions, regardless of the needle used. These lesions increased in severity as the size of the needle increased. Subjectively, the flat-shaped needles were the most easily handled because they tended to roll less in the needle holder. Finally, from a close study of the lesions, it is our belief that a small cutting edge should be added to the needle. This would allow easy passage through the muscular vessel wall and perhaps decrease the severity of the lesions caused by the penetration of the blunt needle.

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References


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