The Effect of Tension on Patency of Rat Femoral Artery Anastomoses

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**Objective:** To determine experimentally the role of tension in the failure of microarterial anastomoses.

**Methods:** Sixteen microarterial anastomoses were performed in femoral arteries from adult Sprague-Dawley male rats after resecting 0, 1, 2, or 3 mm. The percentage of vessel excised was calculated. The vessel was then examined in approximately 1 week for patency. Next, 5 femoral arteries were excised and length-tension curves generated.

**Results:** All vessels were patent in which less than 20% of the length was resected. Clinically, only vessels under extreme tension failed. Length-tension curves suggested 2 zones of vessel extensibility with a breakpoint between them. Extension of the vessel beyond this breakpoint required extreme tension.

**Conclusions:** Moderate tension at a microarterial anastomosis does not lead to anastomotic failure. Failure occurs after the elastic reserve of the vessel is exceeded.

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Although longitudinal tension is widely assumed to be a cause of vascular anastomotic failure, this has not been well documented in prospective studies. Especially in revision of microvascular anastomoses, it is sometimes necessary to choose between performing a primary anastomosis under tension or using an interposition graft. This study was undertaken to elucidate the role of tension on microarterial anastomotic patency.

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**METHODS**

**PHASE 1**

Adult male Sprague-Dawley rats were anesthetized with intraperitoneal xylazine hydrochloride and ketamine hydrochloride. Institutional guidelines regarding animal experimentation were followed. All surgical procedures were performed by one of the investigators (M.E.D.).

Following anesthesia the rat’s groin was opened and femoral vessels exposed from the inguinal ligament to the epigastric vessels. The muscular branch between these 2 points was ligated proximally, cauterized distally, and cut. With the leg in neutral position, the distance between the inguinal ligament and epigastric artery was measured with calipers and recorded. Next, a distance of 0, 1, 2, or 3 mm was marked on the vessel with a marking pen as measured by the calipers. The order of amount excised was rotated (0 mm on one anastomosis, followed by 1 mm on the next, and so on). After placing the marked area in double microclamps, the appropriate length of artery was resected. End-to-end anastomosis was performed with 8 to 9 interrupted stitches of 10-0 nylon suture on a 100-µm needle. The groin wound was closed in 2 layers with 3-0 polyglactin 910 (Vicryl) suture. On postoperative day 6 or 7 the wound was explored and the patency of the vessel was determined using the double-clamp empty-and-refill test.

**PHASE 2**

Average tension for each length of arterial excision was determined in another series of rats. After exposing the femoral artery, the animal was killed by anesthetic overdose. The vessel was clamped distally (at the takeoff of the epigastric vessel), including underlying tissue to prevent vessel retraction. It was then divided proximal to the clamp. Using a stitch to oversew the proximal end (leaving a loop to engage the tensiometer), a tensiometer (Geneva-Gage Inc, Albany, Ore) was used to measure tension required to return the vessel end to its original location. This tension approximated the tension at the anastomosis when no tissue was excised.

The vessel was then excised at the inguinal ligament, and the vessel removed from the animal. After clamping the proximal end of
the excised vessel to a ruler, the distal end was engaged with the tensiometer. The vessel was stretched until the tensiometer read 0.8 cN. This number was selected because it represents the low end of the vessel tension range. The vessel was then extended in 0.5-mm increments and the tension recorded. These measurements represent the approximate tension required to hold vessel ends together at an anastomosis when similar segments of artery are excised.

Eighteen anastomoses were performed in phase 1. Two rats died too early for evaluation, one from intraoperative anesthetic overdose and one at 5 days postoperatively without obvious cause. All anastomoses were patent immediately after surgery.

In the 16 surviving rats, there were 4 anastomoses in each group (0-, 1-, 2-, 3-mm resection groups). All of the anastomoses in the 0-, 1-, and 2-mm groups were patent. Three of the 4 anastomoses in the 3-mm resection group were not patent; the one patent anastomosis in this group had obvious pseudoaneurysms. The average femoral artery length from inguinal ligament to epigastric artery measured 10.8 mm (SD, 1.8 mm).

In phase 2, 5 vessels were tested for baseline tension and then excised for length-tension analysis. The baseline tension was 1.3 cN (SD, 0.3 cN). Because the excised vessel length varied, measured lengths were normalized by dividing the measured length by the length at which the measured tension was 0.8 cN (Figure 1).

During microvascular surgery the surgeon may be faced with a choice: accept a small amount of tension across the anastomosis or perform an interposition graft, requiring additional time for graft harvest, increasing the ischemia time, and doubling the number of anastomoses at risk for thrombosis. Although widely cited as a cause of anastomotic failure, the role of tension as a cause of anastomotic failure has not been well defined. Russell studied tension across anastomoses of the rabbit femoral arteries and found that anastomoses under mild and moderate tension remained patent. He concluded that arteries could be repaired successfully after resecting up to 25% of their mobilized length. Chow et al, however, found that as much as 50% of a mobilized femoral artery segment could be resected without decreasing patency substantially. Katz and Parsa, on the other hand, concluded that even minimal tension on the rat femoral vessels has an adverse effect on the patency rate.

In phase 1 of this study, we resected varying lengths of artery and anastomosed the remaining artery. It has been found that the first postoperative week is a reliable time to evaluate vessel patency after microvascular anastomosis. The vessels that were anastomosed after excision of 1 mm of artery appeared to be under a clinically acceptable amount of tension. After 2-mm excisions, there were slight longitudinal striations visible along the vessels, extending outward from the sutures, in a manner that most would be hesitant to accept in a clinical situation. After 3 mm of arterial resection, the anastomosed vessels appeared to be under extreme tension, with micro-rips at the suture sites. This amount of tension would be unacceptable in a clinical situation.

For phase 1, we calculated the resected length as a percentage of mobilized vessel length. The results depicting vessel patency as a function of the percentage resected are shown in Figure 2.

In phase 2, we wished to measure the actual tension in the vessel. Our initial plan was to measure tension in the vessels after excision of a segment, prior to anastomosis. To prevent exsanguination after arterial division, however, one must place clamps on the vessels, and in the rat’s small femoral vessels, placement of even the smallest clamp markedly changed the vessel extensibility. Therefore, we decided to make tension measurements in freshly killed animals, to permit these measurements without other modifying factors. The in vitro characteristics of the freshly sacrificed artery walls should parallel in vivo performance. Since the amount of vessel excised was variable (within a small range), we normalized measurements by setting tension at 0.8 cN and recording the length. Subsequent lengths for each vessel were divided by this baseline measurement to equalize comparisons. This established a determination of change in vessel length independent of the amount excised. The resulting length-tension curves for 5 vessels are shown in Figure 1.

The same trend was observed for all 5 vessels. Near baseline tension the vessel could be extended easily with small changes in tension. With further extension, the elas-

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**Figure 1.** Tension vs length for 5 rat femoral vessels.

**Figure 2.** Patency as a function of percentage of vessel resected. Each bar represents 1 anastomosis.
tic reserve of the vessel was exhausted and large tension increases were needed to effect small length changes. Although the 5 curves are slightly offset from one another, their shape is similar, suggesting mild frame-shifting due to the limits of experimental accuracy. Although the normalized vessel tension used (0.8 cN) is slightly below average baseline tension, the “breakpoint” consistently occurred about 20% above the starting tension. This is consistent with the results of phase 1, in which no vessel thrombosed if less than 20% of the mobilized vessel length was resected.

Our findings suggest that the inherent elasticity of the arterial wall causes it to behave somewhat like a spring. At rest, the arterial wall has baseline tension related to an inherent stretch. If divided, it recoils about 30%.12 When stretched beyond baseline, inherent elasticity allows moderate accommodation with little increase in vessel tension. When the limit of elastic accommodation is reached, any further extension causes vessel wall damage, exposing thrombogenic proteins to the circulation, resulting in anastomotic failure.

This study addressed only microarterial circulation. Given the anatomic difference in vessel wall structure, these results cannot be extrapolated to microvenous circulation. We hypothesize that the arterial circulation, a high-flow system with thicker vessel walls, is much more tension tolerant than the venous system. In addition, the dynamic situation in clinical microvascular surgery is more complex than the controllable laboratory environment. Individual patient factors and anatomic geometry must be weighed in clinical decision making.

CONCLUSIONS

Tension has long been assigned a causal role in microvascular anastomotic failure. Results of our study show that in the microarterial realm the anastomosis can withstand a surprising amount of tension. We calculate 20% to be a safe upper limit of arterial extensibility. We also provide data on actual vessel tensions, which may guide future investigation.

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