Fasciotomy, Chronic Venous Insufficiency, and the Calf Muscle Pump

Kenneth Bermudez, MD; M. Margaret Knudson, MD; Diane Morabito, RN, MPH; Olivia Kessel, BS

Objective: To test the hypothesis that fasciotomy may impair the function of the calf muscle pump, which in turn could result in the development of chronic venous insufficiency.

Design: A cohort study of patients with a history of lower extremity fasciotomy.

Setting: An urban trauma center.

Patients: Seventeen of the 83 patients identified through trauma, vascular, and/or orthopedic registries consented to participation in this study.

Interventions: Participating patients completed a study questionnaire, and then underwent a complete vascular examination, including air plethysmographic (APG) assessment. Patients with a history of venous injuries were also studied with color flow duplex venous imaging.

Main Outcome Measures: Function of the calf muscle pump as measured by APG, and evidence of chronic venous insufficiency as measured by APG, findings on clinical examination, and by venous ultrasonography.

Results: Seventeen patients completed the study, including 8 with a history of vascular injuries, 6 with old fractures, and 3 who had undergone fasciotomy for soft tissue infections. The time from injury to examination ranged from 5 months to 20 years. Eight patients had signs or symptoms of venous insufficiency, the severity of which appeared to be time dependent. The APG data showed significant mean differences between fasciotomy and control extremities in ejection fraction (P < .001) and residual volume fraction (P < .001), both measures of calf muscle pump function. There were no significant changes in venous filling index, a measure of venous reflux, or in outflow fraction, which correlates with venous obstruction. There were no differences in APG variables between patients with vascular injuries vs those with orthopedic or soft tissue injuries.

Conclusions: Lower extremity fasciotomy impairs long-term calf muscle pump function, as measured by APG, in patients with and without vascular injuries. These patients are at risk for the long-term development of chronic venous insufficiency following lower extremity trauma.

Arch Surg. 1998;133:1356-1361

The beneficial role of fasciotomy in preserving lower limb function in patients with postinjury compartment syndrome is well established.1-3 Recently, however, it has been suggested4-5 that an intact fascia is a crucial component of the calf muscle pump, and that dysfunction of this pump could lead to venous hypertension. Clinical studies on this potential complication of fasciotomy have yielded controversial results, and few include long-term follow-up.6-7 In a previous investigation,8 it was noted that fasciotomy was an independent risk factor for the development of chronic venous insufficiency (CVI) in patients with venous injuries. The purpose of the present study was to investigate the long-term effects of fasciotomy on the calf muscle pump. We hypothesized that fasciotomy would lead to physiologic abnormalities of the lower extremity venous system, and, ultimately, to venous insufficiency.

RESULTS

Of the 83 patients identified through our trauma registry, 16 responded and came to our noninvasive vascular laboratory to be evaluated. One additional patient was recruited from the nursing staff, whose injury was 20 years prior. The average age was 37 years (range, 19-67 years), and there was a preponderance of men (n = 14; 82%). Eight patients had sustained vascular injuries, primarily as the result of penetrating trauma, 6 had a history of a lower extremity fracture, and the 3 re-
PATIENTS AND METHODS

Patients who had been admitted to our trauma center during a 9-year period (1988-1996) with vascular, orthopedic, or soft tissue injuries requiring fasciotomy were identified through the trauma registry. These patients were initially contacted by formatted letters sent to their last known addresses, and they were asked to send reply cards if they were willing to participate in the study that we described. The study protocol and subject letters were reviewed and approved by the Committee on Human Research at the University of California, San Francisco. All fasciotomies that had been performed in these patients included both medial and lateral fascial releases without fibulectomy, as described by Mubarak and Owen.4 We excluded patients who were younger than 18 years old, were currently pregnant, had bilateral fasciotomies, had a history of venous disease or elective venous surgery of the lower extremities, and those with pelvic fractures or tumor. The remaining patients were encouraged to come to the trauma center for an outpatient evaluation. After obtaining informed consent from these eligible patients and reviewing their medical record, the study investigators interviewed participating patients regarding any symptoms of venous insufficiency (ie, pain, edema, limb heaviness, skin changes) and about their physical functions with reference to the time of injury. A physical examination of the lower extremities noted any evidence of varicose veins, ankle or leg edema, skin irritation, pigment changes, and/or ulceration. Patients with a history of venous injury underwent color flow duplex ultrasound imaging (CFD) (model 128XP, Acuson Inc, Mountain View, Calif) performed by a registered vascular technologist. Color flow duplex ultrasound examinations of both legs included the external iliac, long saphenous, femoral (common, deep and superficial), popliteal, tibioperoneal, and calf perforating veins. These veins were evaluated for patency, flow characteristics during respirations (phasicity), and evidence of chronic venous obstruction. Finally, air plethysmography (APG) was conducted on both lower extremities. Air plethysmography is a method of measuring absolute limb blood volume changes, and consists of an air chamber that, when inflated around the leg, can separately measure venous reflux, calf muscle function, venous obstruction, and venous hypertension (Figure 1).10,11 Venous volume is the total venous volume of the measured limb and is calculated while the patient is standing. Outflow fraction is the fractional part of the total venous volume that is expelled 1 second after elevating the limb. During the standing phase, the venous filling index is calculated as the average refilling rate to 90% of venous volume. A venous filling index greater than 2 mL indicates venous reflux.12,13 Ejection fraction is the percentage of blood expelled from the calf after 1 toe-up exercise and is a measure of calf muscle pump function. Residual volume fraction is the fractional part of the venous volume after 10 toe-up exercises, and is proportional to ambulatory venous pressure.10

A confidential data sheet was kept on every patient and included mechanism, type, and date of injury, time from injury to operation, postoperative course, findings on clinical examination (edema, pain, ability to ambulate, lipodermatosclerosis, ulcers), mode of wound closure, and APG measurements. We classified clinical CVI according to the recommendations of the Ad Hoc Committee for Reporting Standards of the Society for Vascular Surgery and the North American chapter of the International Cardiovascular Society (Table 1).14

Bivariate analysis using nonparametric tests for significance (Mann-Whitney U, Wilcoxon rank sum, Kruskal-Wallis) were used to compare APG measurements between patients grouped by various factors (eg, mechanism of injury, presence of symptoms). Pearson correlations were used to study the effect of age on APG data. The McNemar test for 2 related variables was used to compare CFD measurements in patients with venous injuries.
and a residual volume fraction as high as 73.7% compared with 41.2% and 35.3%, respectively, in the normal limb. He had been unemployed since the time of his injury more than 2 years prior.

Figure 2 shows the association between the class of CVI and the time in weeks from injury to the vascular evaluation. Patients were grouped into those with symptoms and those without symptoms, and compared by time from injury to examination. There was a significant association between the 2 groups in the presence of symptoms and the amount of elapsed time from the original injury (P = .04).

The APG data on these patients is given in Table 2. We compared the ejection fraction, residual volume fraction, outflow fraction, venous volume, and venous filling index of the injured leg vs the control leg. There were significant differences in ejection fraction and residual volume fraction (which measure venous pump function) but not in outflow fraction (a measure of venous obstruction) (Figure 3). We also compared the APG data from the affected leg in blunt vs penetrating injuries; presence vs absence of muscle herniation; method of closure (delayed primary repair vs skin graft); male vs female patients; and the effect of age on measurements. There were no statistically significant differences in any of these comparisons. In addition, we grouped our patients into those with vascular injuries vs those with blunt or infectious causes. Again, the APG data did not distinguish between these 2 groups, ie, the APG data were similar in all patients who had undergone fasciotomy.

Venous duplex data on the 7 patients with venous injuries are shown in Table 3. Among these patients, 3 had femoral vein injuries (2 were ligated and 1 was repaired), and the other 4 had popliteal injuries (all were symptomatic and those without symptoms, and compared by time from injury to examination. There was a significant association between the 2 groups in the presence of symptoms and the amount of elapsed time from the original injury (P = .04).

The APG data on these patients is given in Table 2. We compared the ejection fraction, residual volume fraction, outflow fraction, venous volume, and venous filling index of the injured leg vs the control leg. There were significant differences in ejection fraction and residual volume fraction (which measure venous pump function) but not in outflow fraction (a measure of venous obstruction) (Figure 3). We also compared the APG data from the affected leg in blunt vs penetrating injuries; presence vs absence of muscle herniation; method of closure (delayed primary repair vs skin graft); male vs female patients; and the effect of age on measurements. There were no statistically significant differences in any of these comparisons. In addition, we grouped our patients into those with vascular injuries vs those with blunt or infectious causes. Again, the APG data did not distinguish between these 2 groups, ie, the APG data were similar in all patients who had undergone fasciotomy.

Venous duplex data on the 7 patients with venous injuries are shown in Table 3. Among these patients, 3 had femoral vein injuries (2 were ligated and 1 was repaired), and the other 4 had popliteal injuries (all were symptomatic and those without symptoms, and compared by time from injury to examination. There was a significant association between the 2 groups in the presence of symptoms and the amount of elapsed time from the original injury (P = .04).

The APG data on these patients is given in Table 2. We compared the ejection fraction, residual volume fraction, outflow fraction, venous volume, and venous filling index of the injured leg vs the control leg. There were significant differences in ejection fraction and residual volume fraction (which measure venous pump function) but not in outflow fraction (a measure of venous obstruction) (Figure 3). We also compared the APG data from the affected leg in blunt vs penetrating injuries; presence vs absence of muscle herniation; method of closure (delayed primary repair vs skin graft); male vs female patients; and the effect of age on measurements. There were no statistically significant differences in any of these comparisons. In addition, we grouped our patients into those with vascular injuries vs those with blunt or infectious causes. Again, the APG data did not distinguish between these 2 groups, ie, the APG data were similar in all patients who had undergone fasciotomy.

Venous duplex data on the 7 patients with venous injuries are shown in Table 3. Among these patients, 3 had femoral vein injuries (2 were ligated and 1 was repaired), and the other 4 had popliteal injuries (all were symptomatic and those without symptoms, and compared by time from injury to examination. There was a significant association between the 2 groups in the presence of symptoms and the amount of elapsed time from the original injury (P = .04).
repaired). There were no significant differences in the presence of collateral flow channels, the presence of deep vein thrombosis, or the disappearance of phasicity between injured vs uninjured extremities. However, 6 of the 7 injured limbs demonstrated reflux by CFD.

**COMMENT**

Compartment syndrome, a recognized complication of acute lower extremity ischemia or severe soft tissue trauma, results in edematous muscle tamponade of the nutrient capillary bed.13 If unrelieved, progressive tissue expansion in the closed osteofascial compartments causes myonecrosis and permanent neurologic damage, a syndrome first described by Von Volkmann.16 So devastating are the sequelae of untreated compartment syndrome that many trauma surgeons advocate prophylactic fasciotomy in patients considered to be at high risk for development of this complication.13 In the urban hospital, an increasing number of fasciotomies are being performed to treat the complications of severe necrotizing soft tissue infections.

Skeletal muscles have an important role in forcing blood out of veins and back toward the heart. During exercise, the muscle pumping mechanism provides more than 30% of the energy required for blood circulation.6 In particular, the calf is recognized as a highly effective venous pump, capable of generating pressures of greater than 200 mm Hg.17 Components of the calf muscle pump include competent venous valves, the pressure generated by the gastrocnemius and soleus muscles, and the ensheathing fascia and skin surrounding the calf muscles.6 Disruption of any of these elements may lead to calf muscle pump dysfunction, a factor that will increase ambulatory venous pressure and, may, in turn, lead to CVI.

The clinical diagnosis of CVI includes edema, pain, hyperpigmentation, varicose veins, and ulcers. Lipodermatosclerosis occurs when localized venous hypertension promotes capillary endothelial breakdown, causing brown discoloration of the skin from hemosiderin deposits.18 More precise information on the patency and physiologic function of the venous system can be obtained in the noninvasive laboratory with the use of CFD, venous photoplethysmography, and APG. Color flow duplex ultrasonography is an excellent method of defining the anatomy of the venous system, locating collateral channels, detecting acute or chronic deep venous thrombosis, determining venous velocity and flow direction, and detecting reflux. It is a completely noninvasive, painless, and reproducible test that can be used for serial examinations. In a previous and related study,

**Table 2. Air Plethysmography Measurements in Injured and Control Limbs**

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Injured Limbs</th>
<th>Control Limbs</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>EF, %</td>
<td>32.1 ± 12.1</td>
<td>60.8 ± 16.8</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>RVF, %</td>
<td>58.9 ± 19.3</td>
<td>22.2 ± 10.8</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>OF, %</td>
<td>45.2 ± 5.9</td>
<td>49.2 ± 6.9</td>
<td>.09</td>
</tr>
<tr>
<td>VV, mL</td>
<td>119.7 ± 40.0</td>
<td>123.9 ± 41.2</td>
<td>.27</td>
</tr>
<tr>
<td>VFI, mL/s</td>
<td>2.0 ± 1.4</td>
<td>1.8 ± 1.8</td>
<td>.11</td>
</tr>
</tbody>
</table>

* Data are given as mean ± SD. EF indicates ejection fraction; RVF, residual volume fraction; OF, outflow fraction; VV, venous volume; and VFI, venous filling index.

**Table 3. Color Flow Doppler Measurements of Patients With Venous Injury: Injured vs Control Limb**

<table>
<thead>
<tr>
<th>No. With Positive Finding</th>
<th>Injured Limb (n = 7)</th>
<th>Control Limb (n = 7)</th>
<th>P (McNemar Test)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Collateral flow</td>
<td>4</td>
<td>0</td>
<td>.13</td>
</tr>
<tr>
<td>Deep vein thrombosis</td>
<td>0</td>
<td>0</td>
<td>Not applicable</td>
</tr>
<tr>
<td>Reflux</td>
<td>6</td>
<td>1</td>
<td>.06</td>
</tr>
<tr>
<td>Phasicity</td>
<td>3</td>
<td>0</td>
<td>.25</td>
</tr>
</tbody>
</table>

**Figure 2.** The degree of chronic venous insufficiency symptoms (classes 0-3) over time. Note that all 9 patients without symptoms were examined relatively early in respect to their injury, whereas patients with moderate or severe symptoms (n = 4) were examined more than 300 weeks after their initial injuries.

**Figure 3.** Air plethysmographic data on all patients comparing the ejection fraction (EF), residual volume fraction (RVF), and outflow fraction (OF) in the normal (control) vs the injured (fasciotomy) leg.
we performed CFD on 21 patients with a history of venous injuries treated with either repair or ligation.\(^8\) We found no patients with chronic deep vein thrombosis and all repaired veins were patent. However, as shown in Table 3, patients with venous injuries had evidence of reflux by CFD, although these values did not reach statistical significance in this small sample. However, in patients with venous injuries but without fasciotomy, none had evidence of calf muscle pump dysfunction by APG.\(^8\)

VENOUS photoplethysmography is a method that uses a light-sensitive diode placed on the skin of the ankle or foot to detect changes in skin perfusion in response to calf muscle exercise.\(^9\) With photoplethysmography, the venous refilling time can be determined, which is the time it takes to return to baseline after 5 compressive maneuvers (such as tiptoe exercises in the standing position). If the venous refilling time is greater than 20 seconds, a diagnosis of CVI is excluded.\(^10\) Photoplethysmography is not as sensitive as CFD in defining reflux, and calibration of the instrument is not possible. Moreover, the refilling time method of detecting CVI is subject to a number of variables, including the volume of blood displaced, the skin thickness, skin color, and initial blood volume.\(^11\)

Air plethysmography is the only noninvasive method that can evaluate the calf muscle pump function. As described by Christopoulos et al.\(^12\), APG measures changes in the venous volume of the whole leg as a result of postural changes and exercise. By measuring venous reflux and ejection fraction, APG is capable of quantitating both the degree of CVI and the predominant hemodynamic factor (ie, ejection fraction vs reflux) contributing to the symptoms in an individual patient.\(^12,13,19\) The clinical implications in terms of surgical treatment of the venous disease with this type of testing are obvious.\(^20\)

Not all authors agree on the importance of an intact fascia in maintaining competence of the calf muscle pump. Ris et al.\(^7\) studied 21 patients between 32 and 56 months after fasciotomy with physical examination, ambulatory strain-gauge plethysmography, and CFD. None of the patients showed signs of CVI, and ambulatory strain-gauge plethysmography revealed no significant differences in refilling volume or recovery time when comparing limbs with fasciotomy and those without. All veins were patent by CFD and no reflux was seen. While the accuracy of strain-gauge plethysmography compared with APG can be questioned, the real limitation of this study is that Ris et al excluded from evaluation patients of most interest, ie, those with venous hypertension, thus introducing a significant bias into the study.

Aita and colleagues\(^6\) studied 47 patients with a history of 4-compartment fasciotomy, with physical examination, photoplethysmography, and CFD, and found that 92% of patients had unchanged muscle pump function at 19 weeks after fasciotomy. All patients studied had suffered vascular trauma. Although these patients were examined in a prospective fashion, one explanation for their findings may be that they were studied in relatively close proximity to the time of injury. It seems that a longer period may be needed for the development of CVI, as suggested by our present study.

In contrast, Garfin and coworkers\(^21\) found that muscle force and pressure both decreased significantly after fasciotomy and that this decrease could not be attributed to muscle fatigue. Similarly, Rosfors et al,\(^4\) who investigated 21 patients who underwent fasciotomy with photoplethysmography, strain-gauge plethysmography, foot volumetry, and Doppler ultrasonography, found that the affected legs had shorter venous refilling times and reduced volumes of expelled blood in the foot. These investigators concluded that an intact muscle fascia was important for venous return and venous pump function.

The present study clearly shows that patients who have undergone fasciotomy with or without a lower extremity vascular injury consistently have abnormalities in 2 important variables measured by APG—ejection fraction and residual volume fraction. These 2 variables measure the function of the calf muscle pump. Indeed, CVI was evident in 8 of 17 patients. It is probable that fasciotomy results in permanent subclinical malfunction of the calf muscle pump, which may, over time, lead to clinical symptoms. Another important finding in this study is the association between the severity of vascular symptoms and the elapsed time after fasciotomy. Because all of the patients without CVI were evaluated relatively earlier after injury compared with those with symptoms, one might expect asymptomatic patients to develop clinical evidence of CVI on serial examinations.

One of the limitations of the present study is the potential for bias introduced by evaluating only 17 of 83 patients who had undergone fasciotomy. Given the transient nature of trauma patients, it is often difficult to get them to return for scheduled clinic visits, let alone for the research studies, even with financial incentives. It is possible that only patients with what they thought were “poor results” returned for evaluation. Similarly, the need to rely on the patients’ evaluation of their symptoms over a period of time may have influenced our results. Finally, it is possible that the nature of the injury requiring a fasciotomy had more to do with the development of CVI than the fasciotomy alone. These questions are currently being addressed in a large, multicenter prospective study.

We are not questioning the importance of fasciotomy in preserving limb viability and function in patients with compartment syndrome. However, just as Renz and Feliciano\(^22\) have demonstrated that “negative” laparotomy for trauma can no longer be considered a “benign” procedure, our data suggest that fasciotomy is associated with identifiable consequences. The criteria for performing fasciotomy in the absence of clinically apparent compartment syndrome must be rigorously studied, in light of the findings of this study. Alternatively, continuous compartment pressure monitoring for patients at risk may be warranted. We have no experience with reclosing fascia after the resolution of symptoms, and to date, there has been no study of the restoration of calf muscle pump function by closing fascia. Because most trauma patients are relatively young,
and the development of CVI symptoms after fasciotomy are often delayed, this entity may represent an increasingly important surgical problem in the future. One might speculate that compression stockings or other mechanical devices to support the calf muscle pump might prevent or delay symptoms in patients with subclinical dysfunction. Clearly, our study has identified clinically significant and long-term consequences of lower extremity fasciotomy, and opens the door for further investigations into this important posttraumatic problem.

This work was supported by grant R49CCR903697 from the Centers for Disease Control and Prevention, Atlanta, Ga. ACI Medical, Inc, Irvine, Calif, provided training and equipment.

Presented at the 27th Annual Meeting of the Western Trauma Association, Snowbird, Utah, March 5, 1997.

Reprints: M. Margaret Knudson, MD, Department of Surgery, Ward 3A, San Francisco General Hospital, 1001 Potrero Ave, San Francisco, CA 94110 (e-mail: pknudson@sfghsurg.ucsf.edu).

REFERENCES