Adaptation of Canine Saphenous Veins to Grafting
Correlation of Contractility and Contractile Protein Content

Charles L. Seidel, Robert M. Lewis, Rebecca Bowers, Richard D. Bukoski, Han-Seob Kim, Julius C. Allen, and Craig Hartley
From the Department of Medicine, Section of Cardiovascular Sciences, and the Department of Pathology, Baylor College of Medicine, and The Methodist Hospital, Houston, Texas

SUMMARY. Saphenous veins are used extensively to replace stenotic coronary arteries. However, the contractile and biochemical adaptations of grafted veins are unknown. The three purposes of this work were to characterize the contractile properties of grafted veins, to determine whether altered contractile characteristics were associated with quantitative changes in actin, myosin and collagen, and to determine which changes were associated with the surgical procedure and which with placement in the arterial circulation. Canine saphenous veins were removed and returned to their original location (venous autograft), while others were used to replace a segment of femoral artery (arterial graft). The grafts were removed 1, 4, and 8 weeks later and compared with the contralateral saphenous vein. Both graft types exhibited an increase in sensitivity to norepinephrine but not to potassium chloride. The venous autograft exhibited a reversible reduction in myosin content and in maximum contractile response (force/cross-sectional area) to potassium chloride and norepinephrine. In contrast, the arterial graft exhibited increased wall thickness and content of all measured proteins and decreased maximum contractile response. The latter occurred even though there was an increase in the net production of actin and myosin. Expressing the maximum contractile response in terms of the myosin content did not normalize the contractile response. These results suggest that, except for the elevated sensitivity to norepinephrine, the vein is capable of recovering from the effects of surgery within 8 weeks; however, placement of the vein in the arterial circulation delays this recovery and initiates a hypertrophic response that includes an attenuation of contractile function. (Circ Res 55: 102–109, 1984)

SAPHENOUS veins are used extensively to replace stenotic coronary arteries. Many histopathological studies have been performed on grafted human saphenous veins (Campeau et al., 1978; Grondin et al., 1979; Lawrie et al., 1976; Spray and Roberts, 1977; Unni et al., 1974), and these indicate the occurrence of muscle cell necrosis, wall thickening, and proliferation of connective tissue. A limitation of these studies is that only the final vascular changes associated with grafting can be determined. To elucidate the sequence of changes, vascular grafts have been performed in experimental animals (Brody et al., 1972a, 1972b; Fonkalsrud et al., 1978; Karayannacos et al., 1978) using venous tissue from different locations. It was concluded from these studies that the necrosis may result from hypoxia associated with disruption of the vasa vasorum, while the increase in wall thickness may be due to the elevated transmural pressure associated with placing the vein in the arterial circulation.

To date, no studies have been reported which describe the contractile function of grafted veins. The smooth muscle cell is responsible for the synthesis of both connective tissue and contractile proteins (Ross and Klebanoff, 1971). This synthetic activity is sensitive to chemical (Ross and Glomset, 1976), physical (Berry and Greenwald, 1976; Leung et al., 1977; Wolinsky, 1970), and neural (Bevan and Tsuru, 1981) stimuli which may be altered following grafting, producing quantitative changes in protein composition. Such quantitative changes may result in altered contractile function.

In this study, canine saphenous veins were grafted into the femoral artery and compared to controls in order to characterize the contractile properties of grafted veins, to determine whether observed alterations in contractile characteristics were associated with quantitative changes in actin, myosin and collagen, and to determine which of the observed contractile or biochemical changes was due to the surgical procedure or to placement in the arterial (femoral) circulation.

Methods

Experimental Design

Two types of saphenous vein grafts were performed on dogs. To determine the effect of the surgical procedure, a segment of saphenous vein was removed and returned to its original location (venous autograft). To determine the effect of the surgical procedure and placement in the arterial system, a segment of saphenous vein was removed and used to replace a segment of the contralateral femoral...
artery (arterial graft). At the end of 1, 4, and 8 weeks, the
grafted saphenous vein (autograft or arterial graft) and
the contralateral unoperated saphenous vein (control)
were removed. Pharmacological and biochemical charac-
terization of these samples was performed. Analysis of
variance was used to determine whether there were be-
tween-group differences, and the modified t-test (Wallen-
stein et al., 1980) was used to test for significance between
particular means. A P value of 0.05 or less was used as
the criterion for significance.

Animal Surgery and Tissue Preparation

Thirty-six mongrel dogs of either sex weighing 20 kg or
more were anesthetized with Na-pentobarbital (30 mg/
kg, iv). By routine sterile procedures, the left saphenous
vein was exposed and all branches ligated. A length (6–8
cm) of vein was removed, gently flushed with 0.9% NaCl
(saline) to remove residual blood, and allowed to remain
at room temperature in saline for 20–30 minutes. The
saline solution and the time period mimicked conditions
used at Baylor during human aortocoronary grafting. At
the end of this time period, the vessel segment was either
returned to its in situ location (venous autograft) by end-
to-end anastomosis, or was used to replace an equal length
of femoral artery (arterial graft) in the contralateral leg.

After the surgical procedures had been completed, the
animal was given 10,000 U of heparin-Na, iv, as a bolus
injection, and 5,000 U of penicillin, subcutaneously.

After surgery, the animals were housed in pens which
allowed free movement at all times. In addition, each day
during the first week after surgery, the animals were taken
out of their pens and encouraged to walk within the
confines of the animal quarters. This mild exercise pro-
gram and the single injection of heparin enhanced graft
patency.

One, 4, or 8 weeks after grafting, the animal was
anesthetized and the patency of the graft determined by
severing the vessel beyond the downstream anastomosis.
Only patent vessels were used (36 out of 40 vessels). The
entire length of the grafted vein and an equal length of
the contralateral, unoperated vein were removed. The
veins were preserved in 4°C physiological saline solution
(PSI) of the following composition in mM: NaCl, 132;
KCl, 4.7; MgSO4·7H2O, 1.2; NaHCO3, 18; CaCl2, 2; glu-
cose, 5. While in cold PSS, excess adhering tissue was
removed and the vein cut into segments to be used for
contractile and biochemical determinations.

Contractile Characterization

Duplicate segments of control and grafted saphenous
veins (4–6 mm long) were mounted in the same muscle
bath by threading two 23-gauge stainless steel rods
through the lumen of each segment. The lower rod was
attached to an immovable support, whereas the upper rod
was attached to a Grass FT03 isometric force transducer.
The transducer in turn was attached to a calibrated, mov-
able stage by which segment length (i.e., the distance
between the upper and lower rods) could be determined.
On either side of the suspended segment were two parallel
platinum plates (10 mm X 5 mm) 1 cm apart by which the
segments could be electrically stimulated. The seg-
ments were bathed in PSS at 37°C which was continu-
ously gassed with 95% O2 and 5% CO2. Immediately after
the segments were mounted in the muscle bath, their
unstretched length was determined. This was defined as
the distance between the upper and lower rods at which
resistance to an increase in segment length was first de-
tected by the force transducer. The length then was in-
creased so that tension was 1.5 g. After 30 minutes of
equilibration (during which time, most of the tension
decayed), the segments were stretched to 10 g and then
released to 0 g of tension within a 1-minute period. These
slow changes in length were repeated until both the length
at 10 g and the length at 0 g became consistent. Segment
length at 0 g tension was defined as the length at which
further increases in length resulted in a detectable tension
(as with initial length determination). The segments then
were equilibrated at 0 g of tension for another 30 minutes
before their responsiveness to agonists was determined.

Such cyclical length changes have been used by other
investigators to obtain preparations with stable passive
elastic properties and does not result in the deterioration
of contractile responses (Cox, 1977). After these stretches,
the segment length at 0 g tension was 2.4 times the initial
length, and was the same in all groups. In preliminary
experiments on control veins, it was observed that the
optimal segment length for active tension development
was the length at 0 g tension after the stretching proce-
dure.

After equilibration, the segments were stretched at
least three times with 30 mm KCl (added to the bathing
solution) to ensure that consistent contractile responses
were obtained. Cumulative concentration-response curves
then were generated, according to the methods of Van
Rossum and Van den Brink (1963), using norepinephrine
(NE) and potassium chloride (KCl) added to the PSS. To
eliminate a possible interactive effect of prior exposure to
a given agonist, NE and KCl concentration-responses
curves were done in different orders in the pairs of strips.
To determine the contractile response to nerve stimu-
lation, the segments were electrically stimulated at 2
pulses/sec with a pulse duration of 0.2 msec over a voltage
range of 0–10 V. These parameters were selected because
they have been shown to stimulate only nerves (Vanhouette
et al., 1967) and in the present study, the contractile
responses to electrical stimulation were completely
blocked by 10–6 M phenoxybenzamine.

After determination of the contractile characteristics,
the wet weight of each segment was determined and the
cross-sectional area calculated (area = wt/(lxD)) from the
relationship between weight (wt), length (l), and density
[D, 1.05 g/cc (Gordon and Siegman, 1971)]. From the
concentration-response curve, the maximum response was
determined and the ED50 value calculated by probit anal-
ysis. The maximum contractile response to NE and KCl was
normalized to tissue cross-sectional area at the length at
which contraction was determined (i.e., 0 g passive ten-
sion), as well as to the amount of heavy chain myosin in
the segment as calculated from the myosin content of an
adjacent portion of the vessel (see Biochemical Character-
zation, below). Values from pairs of segments from the
same vessel were averaged to obtain a single value for
that animal. For statistical comparison of between-group
ED50 values, the average log ED50 was used as suggested
by Fleming et al. (1972).

Biochemical Characterization

Segments (at least 20 mm long) adjacent to those used
for contractile characterization were used for collagen
and contractile protein determination. They were opened
length-wise, and their length (longitudinal axis) and width
(equal to unstretched internal circumference) were deter-
mined to the nearest millimeter with a microscope and calibrated eye piece (accuracy, 0.1 mm). Prior to the processing, the wet weight of each segment was determined and the cross-sectional area calculated as described above, using the segment length in the longitudinal direction. This area represents the wall area of a section cut perpendicular to the long axis of the vessel. The wall thickness was calculated from the ratio, area:width.

The actin and heavy chain myosin content of saphenous veins was determined as described previously (Seidel and Murphy, 1979). At least 50 mg of vein was homogenized in a glass-on-glass homogenizer containing 0.45 ml of a 25 mM sodium phosphate, 1% β-mercaptoethanol solution (4°C). After homogenization, the solution was made 1% (w/v) in sodium dodecylsulfate (SDS) and heated at 37°C for 15 minutes. The homogenate was centrifuged at 2500 g for 10 minutes at room temperature and the supernatant removed. The remaining tissue pellet was extracted two more times with 0.5 ml of a solution containing 25 mM sodium phosphate, 1% β-mercaptoethanol, and 1% SDS. The protein content of each supernatant sample was determined by the micro-Kjeldahl method. The tissue pellet remaining after the third extraction was dissolved in 1 M sodium hydroxide, and the protein content determined as above. Total protein content of the tissue was the sum of the protein contents of the three supernatants, plus the protein remaining in the tissue pellet.

A 200-μl aliquot from each of the three tissue supernatants was combined with 40-μl of a glycerol-pyronin-Y (0.1% pyronin-Y in glycerol) solution and applied to a 10% T polyacrylamide-SDS gel at 15–70 μg of supernatant protein per well. On the same gel, known amounts (0.8–9.4 μg) of purified skeletal muscle actin and myosin (Sigma Chemical Co.) were also applied to form a standard curve (Fig. 1). After Coomassie blue staining, the amount of actin and heavy chain myosin in each of the three tissue supernatants was determined from densitometric scans of the purified proteins and tissue samples as described previously (Seidel and Murphy, 1979). The actin and heavy chain myosin content of the three extracts of the tissue segment were added together to give the amount of contractile protein in the segment. Approximately 90% of the extractable actin and heavy chain myosin was removed in the first two extracts (see Results). The quantity, "actomyosin," is defined as the sum of the amounts of actin and heavy chain myosin.

Collagen was extracted as described by Wolinsky (1970) from dried fat-free tissue, which was prepared by placing minced tissue pieces in acetone for 6 hours, in ether for 24 hours, and drying to constant weight. The hydroxyproline content of the extract was determined by the method of Bergman and Loxley (1963), and the amount of collagen in the tissue samples was calculated by the correction factor: 7.46 mg collagen/mg hydroxyproline (Neuman and Logan, 1950).

The various tissue protein values were normalized to length and tissue wet weight. Normalization to tissue length provides information about the absolute amount of protein present whereas normalization to tissue wet weight reflects the amount present relative to all tissue components (Wolinsky, 1970).

Results

Figure 1 shows a typical Coomassie blue-stained polyacrylamide-SDS gel containing purified skeletal actin and heavy chain myosin (standards) and an aliquot from each of the three successive extractions of a control saphenous vein segment (C1, C2, C3) and of a 4-week venous autograft (G1, G2, G3). Standards ranged from 0.8 to 9.4 μg of protein per lane (myosin increasing to the right, actin increasing to the left). Tissue samples ranged from 18 to 66 μg of applied total supernatant protein per lane. Heavy chain myosin and actin in tissue samples were identified by their mobility relative to the standards.

actin and heavy chain myosin (standards) and aliquots from a successively extracted control saphenous vein and a 4-week venous autograft. The two bands in the tissue samples with the same mobilities as the purified protein standards were assumed to represent the actin and heavy chain myosin in the tissue samples.

Since it is possible that protease activity and extractability of actin and heavy chain myosin may differ in control and grafted veins, the percentage of total extractable actin or heavy chain myosin in each of the three extractions from a given vessel segment were compared between the various groups. The percent of actin or heavy chain myosin in a given extract was taken as the ratio of the amount in the extract to the amount in the sum of the three extracts times 100. In control veins, the percentage of the total extractable actin in the first and second extracts was 64 ± 3% and 25 ± 2%, respectively, whereas, for heavy chain myosin, it was 58 ± 3% and 27 ± 2%, respectively (n = 35). For any given extract (i.e., first, second, or third), the percentage of the actin and heavy chain myosin extracted was not different, indicating that actin and heavy chain myosin were extracted in a constant ratio. The constancy of this ratio argues against the degradation of actin or myosin by nonspecific tissue
proteases during the three extractions of the tissue segment. Comparison of these percentages between control and grafted tissue indicated that, in 1-week venous autografts and arterial grafts, there was a significant ($P < 0.05$) increase in the percentage of actin appearing in the first extract (80 ± 6% and 88 ± 6%, for venous autograft and arterial graft, respectively); however, there were no differences in the heavy chain myosin percentages or in the actin or myosin percentages in the second and third extracts (data not shown). At all other graft durations, the percentage of actin and heavy chain myosin in the various extracts was not different from control (data not shown). These data indicate that, except for actin in the first extract at 1 week, the extraction of contractile proteins from grafted tissue is similar to its extraction from control tissue, suggesting that protease activity and contractile protein extractability do not vary between control and experimental groups.

Figure 2 describes the dimensional characteristics of the vessels. No significant change in circumference or wall cross-sectional area was detected in venous autografts at any time. Only at 8 weeks was a significant increase in thickness noted. In contrast, the arterial grafts exhibited a significant increase in circumference and wall cross-sectional area at all periods and a significant increase in wall thickness at 4 and 8 weeks after grafting. The significant increase in cross-sectional area at 1 week implies that wall thickening had begun at this time, even though a significant increase in thickness was not detected. The increase in area and wall thickness correlated with graft duration ($r = 0.524$, $P = 0.026$; $r = 0.553$, $P = 0.017$, respectively).

The total protein content of venous autografts (Fig. 3) was unaltered; however, in arterial grafts, protein per unit length was increased significantly at all periods. This increase in total protein was correlated with graft duration ($r = 0.605$, $P = 0.008$). At 1 and 4 weeks, protein per wet weight was reduced in arterial grafts, suggesting accumulation of water.

The actomyosin content of venous and arterial grafts (Fig. 3) was altered only at 1 week where the amount per tissue wet weight was significantly decreased. When actin and heavy chain myosin were considered separately (Fig. 4), a significant change in weight ratio of actin to heavy chain myosin was observed only in 1-week venous autografts, where the ratio was elevated. This increase was due primarily to a reduction in the amount of heavy chain myosin, and had returned to control value by 4 weeks. In arterial grafts, unlike venous autografts, the amount of actin and heavy chain myosin per length continued to increase with graft duration, becoming significantly elevated at 8 weeks. Like total protein, the amounts of actin and heavy chain myosin per length were correlated with graft duration ($r = 0.602$, $P = 0.008$).

The collagen content of venous autografts (Fig. 5) was altered only at 8 weeks, at which time the amount per protein and tissue wet weight was reduced. In contrast, arterial grafts demonstrated a decrease in collagen per tissue weight only at 1 week, and the amount per length increased with the duration of grafting ($r = 0.772$, $P = 0.002$).

None of the grafted tissue demonstrated a change in contractile sensitivity (Fig. 6) to KCl. However, at 4 and 8 weeks, both graft types exhibited an increase in sensitivity to NE. At no time did any of the grafted tissue respond to electrical stimulation using parameters that initiated contraction via nerve stimulation (data not included).
Figure 3. Total protein and actomyosin contents of and grafted saphenous veins. All values are means with the number of observations indicated within each leftmost panel. Control n = 36. *P < 0.05 relative to control; †P < 0.05 relative to venous graft.

Figure 4 illustrates the maximum contractile response of the various groups expressed in three ways: (1) the absolute maximum contractile response to KCl and NE, (2) the maximum contractile response to KCl expressed relative to vessel wall cross-sectional area, and (3) the maximum contractile response to KCl expressed relative to heavy chain myosin content. Similar relationships between control and experimental groups were obtained if the responses to NE were normalized as in Figure 7, B and C (data not shown). Venous autografts did not demonstrate a significant change in either the absolute maximum contractile response or the maximum force relative to heavy chain myosin content; however, when maximum force was expressed relative to vessel cross-sectional area, a significant decrease was observed at 1 and 4 weeks, returning to control values at 8 weeks. In contrast, arterial grafts exhibited a significant reduction in absolute maximum response at 1 and 4 weeks, returning to control values at 8 weeks. Normalization of maximum force to cross-sectional area or heavy chain myosin content did not return these values to control. At 8 weeks, when absolute maximum force (Fig. 7A) had returned to control values, force that was normalized to area (Fig. 7B) or heavy chain myosin content (Fig. 7C, one-tailed modified t-test) was still reduced.

Discussion

In an aortocoronary bypass graft, the grafted saphenous vein is subjected to the effects of (1) the surgical procedure which may include endothelial damage, denervation, hypoxia, and temperature reduction, and (2) placement in the arterial circulation, which includes in addition, increased transmural pressure, shear stress, and oxygen tension. The experimental design of the present experiments permitted separation of these two general effects and
indicated that the effects of the surgical procedure were essentially reversible, whereas placement in the arterial circulation resulted in the establishment of vessel characteristics that remained markedly different from those of control vessels.

The surgical procedure, as reflected in venous autografts, resulted in a sustained loss of responsiveness to electrical stimulation, suggesting functional denervation and a slowly developing increase in sensitivity to NE but not KCl (Fig. 6). The increase in NE sensitivity may be due in part to the apparent denervation (absence of electrical responsiveness); however, an increase in sensitivity to KCl is also usually associated with denervation-induced supersensitivity (Westfall, 1981). We have no explanation for the specificity of the observed sensitivity increase. The maximum contractile response relative to wall cross-sectional area was reduced through the first 4 weeks after grafting, but returned to control values by the 8th week (Fig. 7B). This reduction could be accounted for in part by dilution of contractile protein with noncontractile material, since the amount of actomyosin per wet weight was reduced (Fig. 3), and absolute maximum force (Fig. 7A) and force normalized to myosin content (Fig. 7C) were unchanged. In summary, these observations suggest that the surgical procedure produces minimal changes in the protein composition and contractile characteristics of the vein. The necrosis observed by Brody et al. (1972a, 1972b) in femoral vein grafts, if present in saphenous vein grafts, does not appear to produce a sustained impairment of contractile function.

Placement of saphenous veins in the arterial circulation resulted in the development of characteristics that were distinct from those seen in venous autografts. Arterial grafts exhibited wall hypertrophy, as indicated by increased wall thickness (Fig. 2) and protein contents [total, actin, myosin, and collagen relative to vessel length (Figs. 3-5)]. These parameters increased in correlation with the duration of grafting, suggesting that, even at 8 weeks, the changes associated with hypertrophy may not have stabilized.

It is important to note that the ratios of actomyosin and collagen to total protein remain constant during this hypertrophic response (Figs. 3 and 5), suggesting that: (1) the net production by the smooth muscle cell of contractile and connective tissue proteins

**FIGURE 5.** Collagen content of control and grafted saphenous veins. All values mean ± SEM, with the number of observations indicated within each bar in the leftmost panel. Contr n = 36. *P < 0.05 relative to control; †P < 0.05 relative venous graft.

**FIGURE 6.** *EDSO* values for control and grafted saphenous veins. All values are means ± SEM, with the number of observations indicated within each bar in the leftmost panel. *P < 0.05 relative to control.
increases in parallel, (2) the adaptation of the muscle cell to placement in the arterial circulation is not simply the formation of a protective connective tissue matrix, and (3) the production of actomyosin and collagen may be sensitive to similar stimuli.

The similarity in protein changes observed in arterial grafts and arteries from animals with chronic systemic hypertension (Berry and Greenwald, 1976; Seidel, 1979; Wolinsky, 1970) suggests that the changes in arterial grafts may be due in part to the elevated transmural pressure to which the graft is exposed. However, the graft is also exposed to an elevated partial pressure of oxygen and an increase in shear stress which may also be important stimuli (Faulkner et al., 1975). The experimental design does not permit the differentiation between these or other possible stimuli acting on the vessel wall to produce the observed changes.

As with venous autografts, arterial grafts were unresponsive to electrical stimulation and developed an increase in sensitivity to NE alone (Fig. 6); however, unlike venous autografts, arterial grafts exhibited a sustained reduction in maximum contractile response per vessel wall cross-sectional area (Fig. 7B) or heavy chain myosin content (Fig. 7C). The present data do not permit an identification of the cause of this force reduction, but it could result from structural reorganization of contractile filaments and muscle cells within the vessel wall, as well as alterations in the chemomechanical transduction process.

In summary, the surgical procedure associated with grafting of saphenous veins results in a transient reduction in force generated per wall cross-sectional area and a sustained increase in sensitivity to NE. In contrast, when the vein is grafted into the arterial circulation, there is vessel wall hypertrophy and a sustained reduction in force per wall cross-sectional area. The latter occurs even though there is an increase in the net production of actin and myosin, suggesting that it may result from structural reorganization within the vessel wall, a change in the chemomechanical transduction process, or both.

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Dr. Kim is affiliated with the Department of Pathology.

Address for reprints: Dr. Charles L. Seidel, Department of Medicine, Section of Cardiovascular Sciences, Baylor College of Medicine, One Baylor Plaza, Houston, Texas 77030.

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FIGURE 7. Panel A: absolute maximum contractile response to KCl (120 mm) and NE (10^(-5) M) for the various vessels. Panel B: maximum contractile response to KCl relative to vessel wall cross-sectional area (stress). Panel C: maximum contractile response to KCl expressed relative to vessel wall cross-sectional area and a sustained increase in sensitivity to NE alone (Fig. 6); however, unlike venous autografts, arterial grafts exhibited a sustained reduction in maximum contractile response per vessel wall cross-sectional area (Fig. 7B) or heavy chain myosin content (Fig. 7C). The present data do not permit an identification of the cause of this force reduction, but it could result from structural reorganization of contractile filaments and muscle cells within the vessel wall, as well as alterations in the chemomechanical transduction process. In summary, the surgical procedure associated with grafting of saphenous veins results in a transient reduction in force generated per wall cross-sectional area and a sustained increase in sensitivity to NE. In contrast, when the vein is grafted into the arterial circulation, there is vessel wall hypertrophy and a sustained reduction in force per wall cross-sectional area. The latter occurs even though there is an increase in the net production of actin and myosin, suggesting that it may result from structural reorganization within the vessel wall, a change in the chemomechanical transduction process, or both.
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