Urol Res (1998) 26:215–221

ORIGINAL PAPER

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The latissimus dorsi bladder myoplasty to assist detrusor function

Received: 14 March 1997 / Accepted: 12 January 1998

Abstract The objective of this study was to evaluate whether an innervated skeletal muscle might augment detrusor function. In four dogs we performed the latissimus dorsi myoplasty, a transfer of the latissimus muscle as an innervated free flap wrapped around the bladder. Stimulation of the latissimus dorsi free flap initially achieved an average bladder pressure of 45.8 ± 8.41 cm H_2O, sufficient for partial evacuation. After 4 months the muscle generated a maximal pressure of 82 cm H_2O, resulting in an evacuation of 27.7%. For selected patients, the latissimus dorsi bladder myoplasty may provide an alternative to intermittent catheterization in the future.

Key words Latissimus · Detrusor · Myoplasty · Bladder · Atony

Introduction

To restore bladder contractility in the patient with a hypotonic bladder, we explored the possibility of using the latissimus dorsi muscle to assist detrusor function. In our previous study, the latissimus, which was left in situ, generated sufficient pressure during thoracodorsal nerve stimulation to evacuate 48.3 ± 6.7% of a bladder-like reservoir 8 months after transsection and repair of the thoracodorsal nerve [20]. Because the proposed model showed encouraging nerve and muscle regeneration in situ, the transplantation of the latissimus dorsi muscle as a functional free flap, wrapped around the existing bladder, was investigated next.

The transfer of skeletal muscle to replace or augment the function of another muscle is well accepted in clinical practice. The pedicled latissimus dorsi muscle flap has been used as a biceps muscle substitute [11] or to augment myocardial contractility, a procedure termed the latissimus cardiomyoplasty [2, 10]. In this, the latissimus is raised as a pedicled flap and wrapped around the heart. Muscle stimulation via the thoracodorsal nerve compresses the heart, thereby improving left ventricular ejection. In the present study we examined the feasibility of transferring the latissimus as a free flap to the pelvis, wrapping it around the bladder, and reattaching the thoracodorsal nerve and vessels supplying it microsurgically. With electrostimulation, the resultant bladder compression should augment detrusor function.

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Materials and methods

Four male beagle dogs, aged 22–23 months and weighing 11–13.2 kg, were used in this study. The dogs were sedated with 0.1 mg/kg acepromazine, intubated and maintained with halothane 0.8% and oxygen after intubation. An 8-French feeding tube was used to catheterize and decompress the urinary bladder. Through a 15-cm incision running from the axilla along the medial border of the latissimus, the muscle was dissected free until it remained attached only by the thoracodorsal vessels and nerve. Next, a lower abdominal midline incision from the umbilicus to the symphysis pubis exposed the urinary bladder. Distally, the incision curved laterally to allow access to the iliac vessels. Their exposure was facilitated by division of the right rectus abdominis muscle at its insertion on the pubic ramus. The external iliac vessels were used as recipient vessels in dog 1; in dogs 2–4 the inferior epigastric vessels were chosen. The recipient vessels (Fig. 1) were divided and

prepared for anastomosis with the thoracodorsal vessels. The obturator nerve was transsected as it entered the obturator foramen. Next, the latissimus pedicle was divided in the axilla and the triangularly shaped muscle transferred down to the pelvis where it was placed around the filled bladder and fixed at the corners. It was secured with interrupted 4-0 absorbable sutures (full-thickness sutures of the latissimus, with only the seromuscular layers of the detrusor). The latissimus fully enveloped the bladder (Fig. 2), preventing development of bladder diverticula. An epineural anastomosis of the thoracodorsal to the obturator nerve was performed with interrupted 9-0 sutures under 16-power magnification. Next, the thoracodorsal vein and artery were anastomosed end-to-side to the recipient vessels (insert in Fig. 2). Total warm ischemia time for the latissimus ranged from 80 to 100 min. Finally, a custom-made monopolar electrode (platinum coil surrounded by a silicone sheet) was placed around the thoracodorsal nerve without compressing it.

In dogs 1 and 2 we used an Itrel II stimulator (Itrel II, 7424 Multiprogrammable Neurological Pulse Generator with four channels, Medtronic, Minneapolis, Minn.). Three of the four channels stimulated the latissimus, while the fourth channel was not used: the first channel was used for indirect stimulation and connected to the electrode around the thoracodorsal-obturator nerve. The other two channels were used for direct muscle stimulation and connected to two surface electrodes on the transplanted muscle. (We used flat platinum surface electrodes embedded in silicone with a contact window of 1×3 mm.). For dogs 3 and 4, Itrel I stimulators, supplied with only one channel were available to us (Itrel I, 7421 Multiprogrammable Neurological Pulse Generator). This channel was connected to the electrode around the thoracodorsal-obturator nerve for indirect muscle stimulation. All stimulators were placed subcutaneously in the groin and fixed to the abdominal fascia. Before skin closure both wounds were irrigated with a cephalosporin solution. Upon completion of surgery, which lasted 6–6.5 h, the urinary catheter was removed. All animals received oral cephalosporin for 10 days.

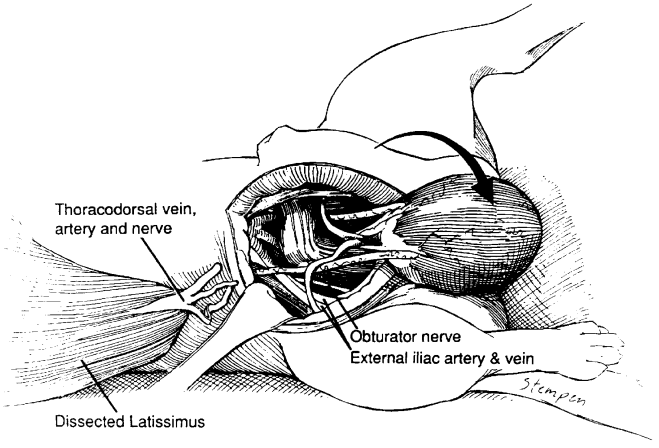


Fig. 1 The urinary bladder is exposed and mobilized, showing the ureters and both vasa. Note the external iliac vessels and the obturator nerve, which will be connected to the pedicle of the latissimus free flap visible at the left

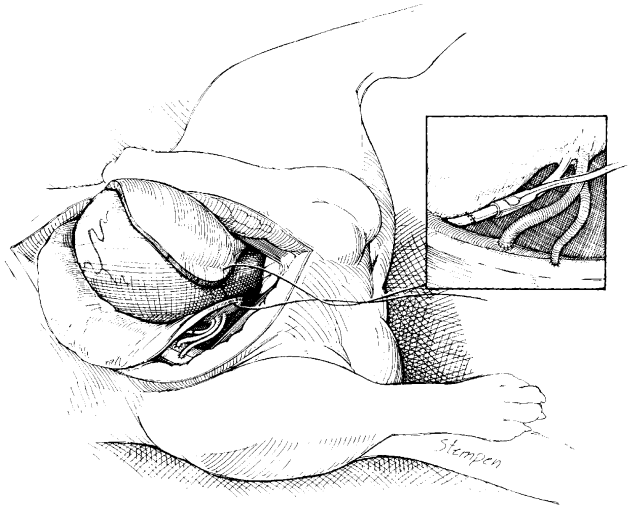


Fig. 2 Envelopment of the urinary bladder with the latissimus dorsi muscle. The thoracodorsal artery and vein are anastomosed to the external iliac vessels. The thoracodorsal nerve is connected with the proximal end of the obturator nerve. The stimulating electrode is placed around the nerve distal to the anastomosis, as the insert shows

Stimulation parameters

During the measurements, when a *tetanic* contraction was induced, we used a monophasic balance-charged rectangular pulse, a pulse width of 180 μs, a pulse rate of 20 pulses per second (pps) and an amplitude of 2.0 V. Stimulation time ranged from 5 to 25 s with a rest period of 2 min between measurements.

To speed neural regeneration, we induced *twitch* contractions continuously for the first 4 weeks after surgery. For this purpose, the following stimulation parameters were used: pulse width, 180 μs; pulse rate, 10 pps (continuous mode). To preserve the battery power of the stimulators, the stimulation time was reduced to 90 min over a period of 24 h from week 5 on (“on” time, 2 min; rest period, 30 min) (cycling mode).

Pressure measurements

Pressure measurements were obtained by a urethral catheter connected to a Gould pressure recorder (Recorder 2400, Gould Instruments Systems Division, Cleveland, Ohio). The bladder was filled to capacity at a rate of 5 ml/min and filling was stopped once leakage occurred around the catheter. After filling the bladder, we waited 2 min until the pressure at bladder capacity stabilized. Next, the thoracodorsal nerve was stimulated (20 pps, 2.0 V) and the bladder pressure recorded. Baseline measurements were recorded during the initial surgery, immediately after the transplant; measurements were repeated at monthly intervals thereafter.

The anesthetized dogs were catheterized and the bladders slowly filled until the “leak point” volume was reached and noted. Next, the relationship of generated pressure to volume was established (Fig. 3). As the latissimus muscle transplant was deprived of its baseline tension the contractile efficiency depended upon the volume in the bladder before stimulation. With increasing volume the baseline or resting tension (before stimulation) and the generated pressure with stimulation were expected to increase. A Babcock clamp was placed around the base of the catheterized penis to prevent leakage. We began with a bladder volume of 10 ml followed by 10-ml increments. With each stepwise increase (once a stable baseline pressure was reached), the latissimus was stimulated and the resultant pressure recorded. We recorded the total bladder pressure (i.e., the sum of baseline pressure before stimulation plus the pressure increase caused by the contracted latissimus muscle during stimulation). We arbitrarily stopped the bladder filling once a resting pressure of 80 cm H_2O was reached. Measurements at a baseline pressure above 80 cm H_2O were not done as the leak point pressure in the anaesthetized dogs ranged from 25 to 30 cm H_2O (see below). Therefore in the awake dog baseline pressures above 80 cm H_2O were not likely to occur. Additionally, at resting pressures above 80 cm H_2O, renal damage might develop.

To assess bladder evacuation, the bladder was slowly filled until leakage occurred. Next, the catheter was removed and the latissimus stimulated; the voided and residual volumes were determined.

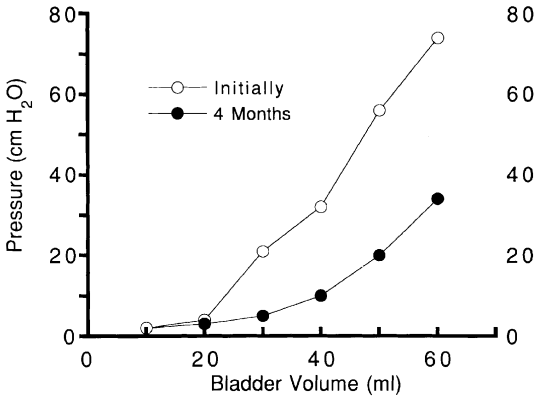


Fig. 3 Bladder pressure generated at 20 pulses per second (pps) and 2.0 V vs bladder volume recorded initially, i.e., at the end of initial surgery, (*open circles*) and 4 months after latissimus transplant, (*filled circles*). The total bladder pressure, i.e., the sum of baseline pressure before stimulation plus the pressure increase caused by the contracted latissimus muscle during stimulation, is given

To avoid injury to the vascular anastomoses, which were subjected to tension with each contraction, we limited ourselves to one pressure measurement at bladder capacity during the initial procedure.

Tissue collection

Biopsies of the latissimus muscle stained for collagen and muscle (Masson's trichrome) were obtained at the time of initial muscle flap elevation. In addition, a second biopsy was obtained when the dogs were killed 3–4 months later. Biopsies of the thoracodorsal nerve were taken at the initial surgery and at the time of death. Sections of the nerve were fixed in 3% glutaraldehyde, 2% paraformaldehyde and 0.05 mol phosphate buffer, then embedded in EM bed-812 (Electron Microscopy Sciences, Ft Washington Pa.). One-micron sections were stained with 1% toluidine blue. The comparison of the cross-sections harvested initially and 3–4 months after transplant permitted a qualitative assessment of nerve regeneration.

Radiographic studies

Imaging studies were performed of the upper and lower urinary tract at the conclusion of the study. Each of the three dogs completing the study underwent an intravenous pyelogram (Hypaque meglumine 60%, 2 ml/kg i.v.; Winthrop Pharmaceuticals, New York, N.Y.). Four months after the transplant a voiding cystourethrogram during latissimus stimulation was obtained in dog 1. All animal experiments were approved by the animal care committee of the University of California, San Francisco (UCSF) and followed the 'Principles of Laboratory Animal Care' of the NIH.

Results

After transfer to the pelvis, all latissimus dorsi muscles were successfully revascularized initially, as evidenced by a pulse within the thoracodorsal artery, the muscle's appearance, and bleeding from the cut edges. Stimulation during the initial surgery induced voiding from and alongside the urinary catheter in all dogs. The maximal pressures during latissimus stimulation at the initial surgery ranged from 30 to 66 cm H_2O (mean ± SEM = 45.8 ± 8.41), representing an increase of 20–66 cm H_2O (mean 37.5 ± 9.94).

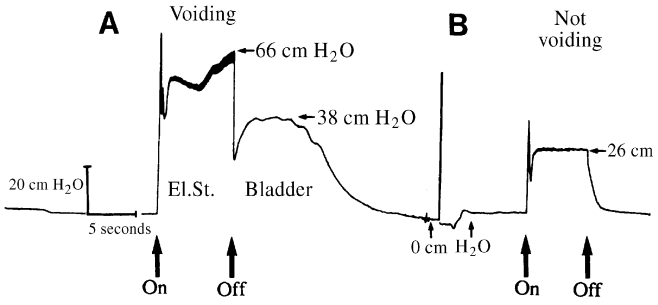


Fig. 4A,B Bladder pressure recorded at the end of surgery during latissimus stimulation (20 pps, 2.0 V). **A** The individual contributions of the latissimus and the detrusor to bladder pressure become apparent once the stimulation is turned off and the detrusor contraction alone is reflected by the pressure tracing. **B** No detrusor contraction occurs. A sharp pressure increase, a subsequent plateau and sharp decline (expected with contraction of skeletal muscle) are seen. The traces were obtained from dog 3, which was supplied with an Itrel I stimulator

To determine the bladder's contribution (if any) to the total generated pressure, the stimulation was stopped during the voiding process (Fig. 4A). Contraction of the latissimus resulted in an initial steep pressure rise to 60 cm H_2O; however, within seconds a slower pressure rise – induced by detrusor contraction – was seen. Once the electrical stimulation was turned off, the contribution of the detrusor, with a maximal pressure of 38 cm H_2O, was evident. This detrusor contribution was only seen initially, not during subsequent measurement. Fig. 4B demonstrates latissimus stimulation without voiding: the bladder shows a pressure plateau typical of a tetanic contraction in a skeletal muscle caused by the latissimus contraction alone. (The traces in Fig. 4 were obtained from dog 3, which was provided with an Itrel I stimulator.)

Postoperatively, no wound or intra-abdominal complications were noted. The unilateral division of the obturator nerve caused a slight, transient impairment in gait. Initially the affected hindleg could not be prevented from sliding outward owing to a paralysis of the denervated adductor muscles, but within a week the animals adapted to the deficit and ambulated normally.

The batteries of the Itrel I pulse generators (placed in two dogs) were exhausted during the first weeks of training stimulation. One dog was killed at 6 weeks because of this stimulator failure; the second remained in the study so that long-term measurements of bladder capacity could be obtained. The Itrel II stimulators (dogs 1 and 2) both remained functional until the conclusion of the study. In one of these dogs, however, no significant bladder pressure increase was seen with stimulation, and fatty degeneration of the latissimus muscle was found when the dog was killed.

In one dog (dog 1) the revascularized and reinnervated latissimus free flap equipped with a functioning stimulating unit enabled us to collect data over the entire 4 months. Six weeks after implantation, we measured the

lead–electrode–tissue impedance and the contribution of each channel of the Itrel II pulse generator to latissimus contraction: the neural electrode had an impedance of 330 ohms, the surface electrodes 838 and 1089 ohms. The battery current flow was measured at 13 μamps. The stimulation voltage was 2.0 V during indirect latissimus stimulation via the thoracodorsal nerve. Activation of the surface electrodes produced a pressure rise of 6 cm H_2O, as opposed to an increase of 24 cm H_2O with activation of the neural electrode. Based on these measurements, the surface electrodes accounted for 25% of the latissimus contraction, the neural electrode 75%.

The urodynamic results of the dog with a functioning stimulator and viable latissimus free flap are shown in Table 1. The pressure increase induced by the stimulated latissimus ranged over time from 30 to 44 cm H_2O. The peak pressures during stimulation were 62–98 cm H_2O, reflecting the continuing ability of the muscle to generate significant bladder pressures for the entire study period. The leak point pressure was 25–30 cm H_2O. Bladder capacity with nonprovocative filling decreased slightly – from 70 ml initially to 65 ml (93%) at completion of the study. To demonstrate the ability of the latissimus to generate pressure with a fully distended bladder, the bladder was filled with volumes 5–30 ml beyond its capacity (with the urethra occluded) until a resting pressure of 80 cm H_2O was reached. The volume when filling was stopped ranged from 55 to 70 ml.

The preoperative bladder capacities in dogs 1–4 were 70, 130, 150 and 100 ml, respectively. After 6 weeks the capacity was reduced to 35 ml (50%) in dog 1 (in which the latissimus muscle remained viable for 4 months) and to 35 ml (27%), 30 ml (20%) and 45 ml (45%) in dogs 2–4, respectively. Late occlusion of the vessel anastomoses in dogs 2–4 caused the latissimus to degenerate, and we attribute their reduction in bladder capacity to accompanying shrinkage. At completion of the study the bladder capacity in dog 1 was 65 ml (93%) (Table 1). The leak point volume and the maximal volume were not reduced after week 6 and regained their initial values at completion of the study (Table 1). However, no other dogs were available to sustain the finding of preserved bladder capacity. The bladder compliance was markedly reduced after latissimus dorsi bladder myoplasty at completion of the study in dog 1 (Fig. 5).

Table 1 Urodynamic data after latissimus bladder myoplasty

	Postoperative weeks				
	0	6	10	13	16
ΔP_{stim} (cm H_2O)	30	22	20	34	44
P_{stim} at max. volume (cm H_2O)	62	72	80	74	98
Leak point pressure (cm H_2O)	25	30	27	30	30
Leak point volume (ml)	70	35	30	35	65
Maximal volume (ml)	70	55	60	55	70

ΔP_{stim}, pressure increase induced by latissimus
P_{stim} at max. volume, bladder pressure after maximal filling with latissimus stimulation

Figure 6A demonstrates a sharp pressure rise caused by latissimus contraction, followed by a rapid pressure decrease indicating voiding. Before the latissimus muscle was stimulated, the bladder was filled to capacity (65 ml) and demonstrated a stable pressure of 42 cm H_2O. Pressure after voiding was 24 cm H_2O. The initial fast pressure drop during voiding and the drop of the baseline pressure thereafter characterize skeletal muscle de-

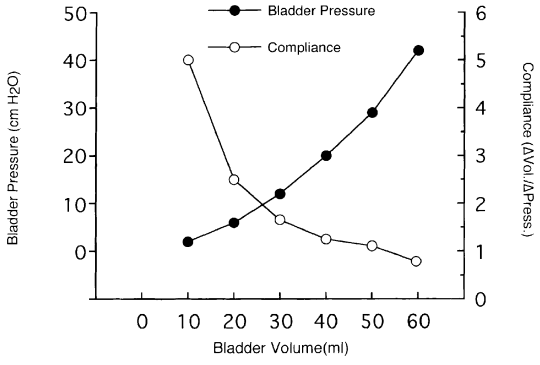
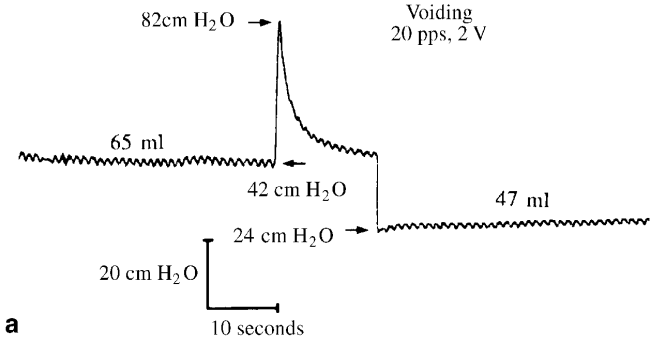


Fig. 5 Bladder compliance after 4 months



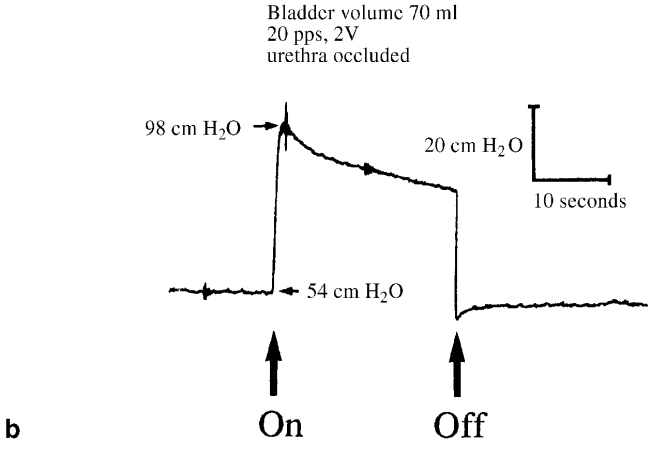


Fig. 6 Bladder pressure 4 months after latissimus transplantation recorded **a** during voiding and **b** with the urethra occluded induced by stimulation (20 pps, 2.0 V)

prived of its resting tension. Isovolumetric pressure measurements performed with the urethra occluded are shown in Fig. 6B. Instead of the rapid pressure drop seen during voiding, a slower and smaller decline in pressure occurred, reflecting muscle fatigue. As no volume was expelled, the pressure before and after stimulation did not differ significantly. The sharp increase and decrease of pressure marking the stimulation period is typical for skeletal muscle (Fig. 6B).

Four months after surgery, the voided volume with latissimus stimulation was 18 ml from a capacity of 65 ml (27.7%). Flow occurred during the initial pressure rise, as seen in Fig. 6A. The urinary catheter was removed, but the sphincter was intact during this evacuation study.

Intravenous pyelograms were performed in all three surviving dogs at the end of the study (after 12 weeks in two dogs and after 16 weeks in one dog; mean 13.3 weeks). In all, the upper urinary tracts were normal and without dilatation.

Histologically, cross-sections of the thoracodorsal nerve showed thinner myelin of newly regenerated nerve fibers and fiber clustering (groups of fibers in one axon sheath) in the segment distal to the anastomosis in all dogs. Both features characterize nerve regeneration. The histologic evaluation of the latissimus bladder myoplasty showed normal detrusor muscle surrounded by striated latissimus muscle fibers in dog 1; in dogs 2–4 the latissimus muscle tissue was replaced by fat.

Discussion

Transfer of the latissimus muscle to the pelvis is a complicated procedure requiring reinnervation of the muscle and microsurgical reattachment of the thoracodorsal vessels. However, no local muscles were found to be suitable for bladder myoplasty. Our first choice was the rectus abdominis, avoiding the risks inherent in a free flap procedure. In anatomical studies in dogs and pigs the rectus was found unsuitable because of its segmental innervation by four or five intercostal nerves. To avoid muscle atrophy and to permit later muscle stimulation, the divided intercostal nerves would have to be reanastomosed to three or four motor nerves near the bladder. Unfortunately, there are not enough suitable motor nerves within the pelvis that can be used for these anastomoses. The geometry of the comparatively large and thin latissimus with a single pedicle (thoracodorsal nerve, artery and vein) supplying the entire muscle prompted us to use this muscle.

We previously addressed the question of reinnervation and muscle regeneration when the latissimus muscle is used as a detrusor substitute [20]. In that study the pedicled latissimus was elevated and wrapped around a silicone reservoir, designed to simulate the denervated bladder and allow pressure measurements. Initially the neurovascular pedicle was left intact and the pressure generated inside the silicone reservoir was measured during stimulation. Next, the thoracodorsal nerve was divided and immediately reanastomosed microsurgically. Eight months later, with thoracodorsal nerve stimulation, the reservoir pressure reached 79.3 cm H_2O (72.4% of the initial value), resulting in an evacuation of 48.3% of the reservoir volume. Nerve regeneration distal to the anastomosis was seen in all dogs [20].

In the present study, although all four latissimus dorsi free flaps were successfully revascularized at the initial procedure, only one muscle remained viable at 4 months. The remaining muscles underwent fatty degeneration, which was attributed to thoracodorsal vessel thrombosis, most likely owing to an inability to prevent leg motion and subsequent vessel kinking during the immediate postoperative period in the dog. Postoperative rest to allow healing before mobilization and the use of the deeper and less mobile iliac vessels and of Itrel II stimulators appear to be factors likely to improve the success rate in humans. As the techniques of microsurgical free flaps have established success rates of 95% [6–8], we would expect a similar rate for the latissimus bladder myoplasty. Despite the loss of the free latissimus flap in three cases, the histologic studies performed at completion of the study demonstrated nerve sprouting and regeneration within the thoracodorsal nerve distal to the anastomosis in all animals, indicating the reliability of this neural repair.

An ideal animal model, able to exclude a detrusor contribution to generated pressure, requires a decentralized bladder, i.e., a spinal injury. For chronic study, we felt that this would cause excessive morbidity. Still, the contribution of the latissimus myoplasty and that of the detrusor muscle to bladder pressure were separable (Fig. 4). We can only speculate why we found a detrusor contraction with stimulation at the initial surgery but not in later measurements. Possibly current leakage to the detrusor occurred initially and not in later measurements. A higher tissue impedance might have blocked the current spread later. Also the reduced bladder capacity after the myoplasty might have kept the detrusor from reaching its normal distension, thus preventing reflex bladder contraction. As a result we were able to measure the effect of the latissimus contraction without a contribution from the detrusor. As shown in Fig. 6, the pressure tracings exhibited the rapid increase with stimulation and decrease at cessation characteristic of skeletal muscle alone. The ability of the transplanted latissimus to generate sufficient bladder pressure to allow partial evacuation could be demonstrated repeatedly over 4 months and is encouraging.

While initial pressures were excellent, the ability of the latissimus to maintain sufficient pressure during the entire voiding process seems limited. In our previous study the in situ muscle evacuated 48.6% of a silicone reservoir 8 months after thoracodorsal nerve repair [20], while this study demonstrated an evacuation of 27.7% of the native bladder at 4 months by the free flap. At 4 months nerve regeneration is incomplete, and further improvement in bladder evacuation must be expected

for up to two years [9, 18]. In addition, we did not perform a sphincterotomy (to avoid the resulting incontinence and urinary tract infection). A lower residual volume is anticipated with a relaxed sphincter.

Bladder evacuation results in a decrease of resting tension in the latissimus muscle, which in turn reduces its contractile efficiency [16]. In contrast, the detrusor is able to maintain pressure while it is shortening. With refined stimulation techniques it might be possible to alter the contraction of the latissimus, thus improving bladder evacuation. In the latissimus cardiomyoplasty, altering the stimulation has been reported to change the twitch characteristics of skeletal muscle in animals [14] and humans [15]. When skeletal muscle is used to augment cardiac function, modulating the stimulating frequency will prolong contraction: inefficient twitches will become sustained contractions, increasing cardiac output [4, 5]. Studies of the electrical activity of the detrusor showed a distinct pattern that could be correlated with the voiding cycle and degree of bladder filling [13]. Imitating these stimulation patterns in the latissimus bladder myoplasty may cause the muscle to adapt adequately to allow more efficient bladder evacuation, similar to the success achieved with cardiac myoplasties. Compared with the latter, the latissimus bladder myoplasty has the advantage that muscle fatigue is less of a problem, as one contraction every 4–6 hours is sufficient for bladder evacuation.

The reduction in bladder capacity in the first weeks after the free flap procedure is probably caused by skeletal muscle adaptation [1] and scarring around the bladder. A skeletal muscle placed around the bladder will shorten to a length that restores its former resting tension. To overcome the anticipated capacity reduction, we distended the enveloped bladder but avoided pressures above 80 cm H_2O to spare the upper urinary tract. With this procedure the bladder capacity was preserved (92%) at 4 months in one dog, but no other dogs were available to sustain the finding of preserved capacity. As the degeneration of noninnervated latissimus muscle results in fatty muscle replacement rather than scarring, no permanent bladder shrinkage is anticipated [A. Stenzl, pers. comm., 1996].

The reduction in bladder compliance with increasing volume occurs because, unlike the detrusor, skeletal muscle does not distend in response to stretch. However, these changes in compliance and capacity did not visibly damage the upper urinary tract. Prompt excretion of contrast without evidence of dilatation was seen in the pyelograms performed at the study's conclusion.

The low voltage (2.0 V) and current flow (13 μamps) used to achieve contraction underline the integrity of the regenerated nerve-muscle unit. Indirect muscle stimulation via the obturator/thoracodorsal nerve was used effectively and has a number of advantages over direct detrusor stimulation. The latter would have required voltages of 20–40 V and is fraught with pain, dyssynergic sphincteric contraction and involuntary contractions of surrounding muscles induced by current spread [3, 12, 19] – all of which were avoided with the latissimus bladder myoplasty.

In conclusion, the latissimus dorsi bladder myoplasty is a technically feasible surgical procedure that can partly augment lost detrusor function. The reinnervated and revascularized latissimus free flap generated bladder pressures sufficient for partial evacuation throughout the 4-month study period. This operation makes use of microsurgical techniques and stimulating equipment already established in human clinical practice. Minimal morbidity is expected, and success rates for patients undergoing this procedure would expected to be acceptable.

At present it seems premature to define the clinical value of the latissimus dorsi myoplasty, as the residual volume predicted by these animal experiments would still necessitate the use of intermittent catheterization. These experiments demonstrate the advantages and limits of skeletal muscle used to augment lost detrusor function. A recent study in which the latissimus dorsi myoplasty was performed with partial bladder removal showed a sufficient bladder pressure rise in 3 of 12 dogs, supporting our approach [17]. For the many patients now performing intermittent catheterization the latissimus bladder myoplasty may eventually provide a treatment alternative.

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