From the Southern Association for Vascular Surgery

First experience in human beings with a permanently implantable intrasac pressure transducer for monitoring endovascular repair of abdominal aortic aneurysms

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Objectives: **Endovascular stent graft repair of abdominal aortic aneurysms (AAAs) prevents rupture by excluding the aneurysm sac from systemic arterial pressure. Current surveillance protocols after endovascular aneurysm repair (EVAR) follow secondary markers of sac pressurization, namely, endoleak and sac enlargement. We report the first clinical experience with the use of a permanently implantable, ultrasound-activated remote pressure transducer to measure intrasac pressure after EVAR.**

Methods: **Over 7 months, 14 patients underwent EVAR of an infrarenal abdominal aortic aneurysm with implantation of an ultrasound-activated remote pressure transducer fixed to the outside of the stent graft and exposed to the excluded aortic sac. Twelve patients received modular bifurcated stent grafts, and 2 patients received aortouniiliac devices. Intrasac pressures were measured directly with an intravascular catheter and by the remote sensor at stent-graft deployment. Follow-up sac pressures were measured with a remote sensor and correlated with systemic arterial pressure at every follow-up visit. Mean follow-up was 2.6 ± 1.9 months.**

Results: **Excellent concordance was found between catheter-derived and transducer-derived intrasac pressssure intraoperatively. Pulsatile waveforms were seen in all functioning transducers at each evaluation interval. One implant ceased to function at 2 months of follow-up. In 1 patient a type I endoleak was diagnosed on 1-month computed tomography (CT) scans; 3 type II endoleaks were observed. Those patients with complete exclusion of the aneurysm on CT scans had a significant difference in systemic and sac systolic pressures initially ($P < .001$) and at 1 month ($P < .001$). Initial sac diastolic pressures were higher than systemic diastolic pressures ($P < .001$). The ratio of systemic to sac systolic pressure increased over time in those patients with complete aneurysm exclusion ($P < .001$). Four of 6 patients with no endoleak and greater than 1-month follow-up had diminution of sac systolic pressure to 40 mm Hg or less by 3 months.**

Conclusion: **This is the first report of a totally implantable chronic pressure transducer to monitor the results of EVAR in human beings. Aneurysm exclusion leads to gradual diminution of sac pressure over several months. Additional clinical follow-up will be necessary to determine whether aneurysm sac pressure monitoring can replace CT in the long-term surveillance of patients after EVAR. (J Vasc Surg 2004;40:405-12.)**

Since the initial report by Parodi et al[1] in 1991, endovascular stent graft repair of abdominal aortic aneurysms (AAAs) has emerged as a potential alternative to traditional open repair. Endovascular aneurysm repair (EVAR) has clear benefits in minimizing periprocedural morbidity and potentially reducing mortality.[2] Midterm results with second-generation and third-generation devices have been excellent.[3] However, complications unique to endovascular repair have been identified. Perhaps the most significant of these are endoleak and aneurysm sac expansion. Current surveillance protocols after EVAR, whether computed tomography (CT), magnetic resonance imaging, or duplex ultrasound scanning, are sensitive in enabling identification of these secondary markers of sac pressurization. Much work has been done, both in vitro and in animal models, to characterize the changes in aneurysm sac pressurization after EVAR.[4-12] As yet no prospective studies have been

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performed to analyze what happens to aneurysm sac pressure over time after EVAR in human beings.

We report the first clinical experience with the use of a permanently implantable, ultrasound-activated remote pressure transducer to measure intrasac pressure after EVAR.

METHODS

Since June 2003, 14 patients undergoing EVAR of an infrarenal AAA were enrolled in this protocol. Twelve patients received modular bifurcated devices, and 2 patients received aortouniiliac devices. Mean preoperative aneurysm size was 6.3 ± 0.9 cm (range, 5.0-8.0 cm). Mean follow-up was 2.6 ± 1.9 months. This study was performed in conjunction with our investigator-sponsored investigational device exemption examining the use of the Talent endovascular stent graft in the treatment of infrarenal AAAs in patients at high risk. The expanded experimental protocol and informed consent were approved by the US Food and Drug Administration and the Institutional Review Board of Mount Sinai School of Medicine. All subjects gave informed consent.

Eligibility criteria. In brief, the Talent protocol is an investigator-sponsored investigational device exemption examining the use of the Talent-LPS endovascular stent graft for repair of infrarenal AAAs in patients at high risk. Patients must be deemed as at prohibitively high risk for standard surgical therapy because of severe comorbid medical illnesses. Anatomic criteria include a normal proximal neck at least 1.5 cm long and a normal distal neck at least 1 cm long, with access vessels at least 8 mm in diameter.

Endovascular stent-graft description. The Talent-LPS endovascular stent graft has been described.[13] In brief, this self-expanding endoprosthesis is composed of woven polyester supported by nitinol Z-forms and a longitudinal bar. The prosthesis is available in either an aortouniiliac or modular bifurcated configuration. Deployment is by means of manual retraction of the outer sheath. The outer diameter ranges from 18F to 25F.

Device description. The Impressure AAA Sac Pressure Transducer (Remon Medical) measures 3 mm × 9 mm × 1.5 mm (Fig 1, *A*). It consists of a piezoelectric membrane, which when actuated by ultrasound waves from a hand-held probe charges a capacitor. Once charged, the transducer measures ambient pressure, then generates an ultrasound signal, which is relayed to the probe. The data can then be downloaded and exported as an Excel data file consisting of pressure measurements and the corresponding times at which the measurements were taken. The transducers were hand-sewn to the outside of the stent graft, then repackaged in the delivery sheath. In patients who received an aortouniiliac device, the transducer was sewn to the graft approximately 6 cm below the attachment system. No upsizing of the delivery system was necessary. In patients who received a bifurcated device, the device was sewn to the contralateral limb just below its exit from the main body gate, and repackaged in a 20F delivery sheath (Fig 1, *B*). This represents upsizing of the sheath from its original 18F dimension.

Deployment technique. The technique for stent-graft deployment has been described.[13] At stent-graft deployment an intrasac catheter (5F Berenstein; Boston Scientific) is placed in the excluded aneurysm. Simultaneous catheter and ultrasound transducer pressures are obtained in the operating room.

Follow-up. Follow-up, consisting of a physician office visit, plain abdominal radiography, and 3-phase contrast material–enhanced CT angiography was performed at 1, 6, and 12 months, and yearly thereafter. Intrasac pressures were obtained at every physician office visit (Fig 1, *D*). Both translumbar and transabdominal approaches were used to obtain pressure measurements. Systemic pressure was obtained with a pressure cuff applied to the upper extremity.

Statistical analysis. Concordance between simultaneous pressure measurements was determined with the Pearson correlation. Logistic regression analysis was used to calculate the line of best fit for the scatterplots. Continuous variables were compared with the paired Student *t* test. Significance was assumed at $P < .05$.

RESULTS

Clinical success

In 13 of the 14 patients (93%) the aneurysm was successfully excluded at the primary operation. In 1 patient a distal type I endoleak with outflow through a patent lumbar artery was diagnosed at 1-month follow-up, and was successfully treated with an extension cuff. In this patient a type II endoleak persisted. In 2 patients type II endoleaks were noted on the 1-month CT scans, 1 from a lumbar artery and 1 from a patent inferior mesenteric artery (IMA) and paired lumbar arteries. The IMA endoleak was persistent on the 6-month CT scan.

Technical success

We were unable to obtain simultaneous intraoperative pressures in 4 patients. In 1 patient, small calcified iliac arteries prevented passage of the iliac limb alongside the intrasac catheter, and it was necessary to remove the intrasac catheter before device implantation. Two devices demonstrated initial software problems, which were corrected, enabling continued device function. In the fourth patient we were unable to obtain intraoperative pressures through an anterior transabdominal approach, because of the patient's body habitus. We were subsequently able to obtain pressures through a translumbar approach once the sterile drapes were removed.

Postoperatively we were successful in obtaining pressures at every visit in all 14 patients. Interrogation of the sensor in the office typically took 3 to 15 minutes. One device ceased functioning at 2-month follow-up.

Initial concordance

The intraoperative concordance between transducer-derived sac pressures and catheter-derived sac pressures was excellent (Fig 2, *A to C*). The Pearson correlation coeffi-

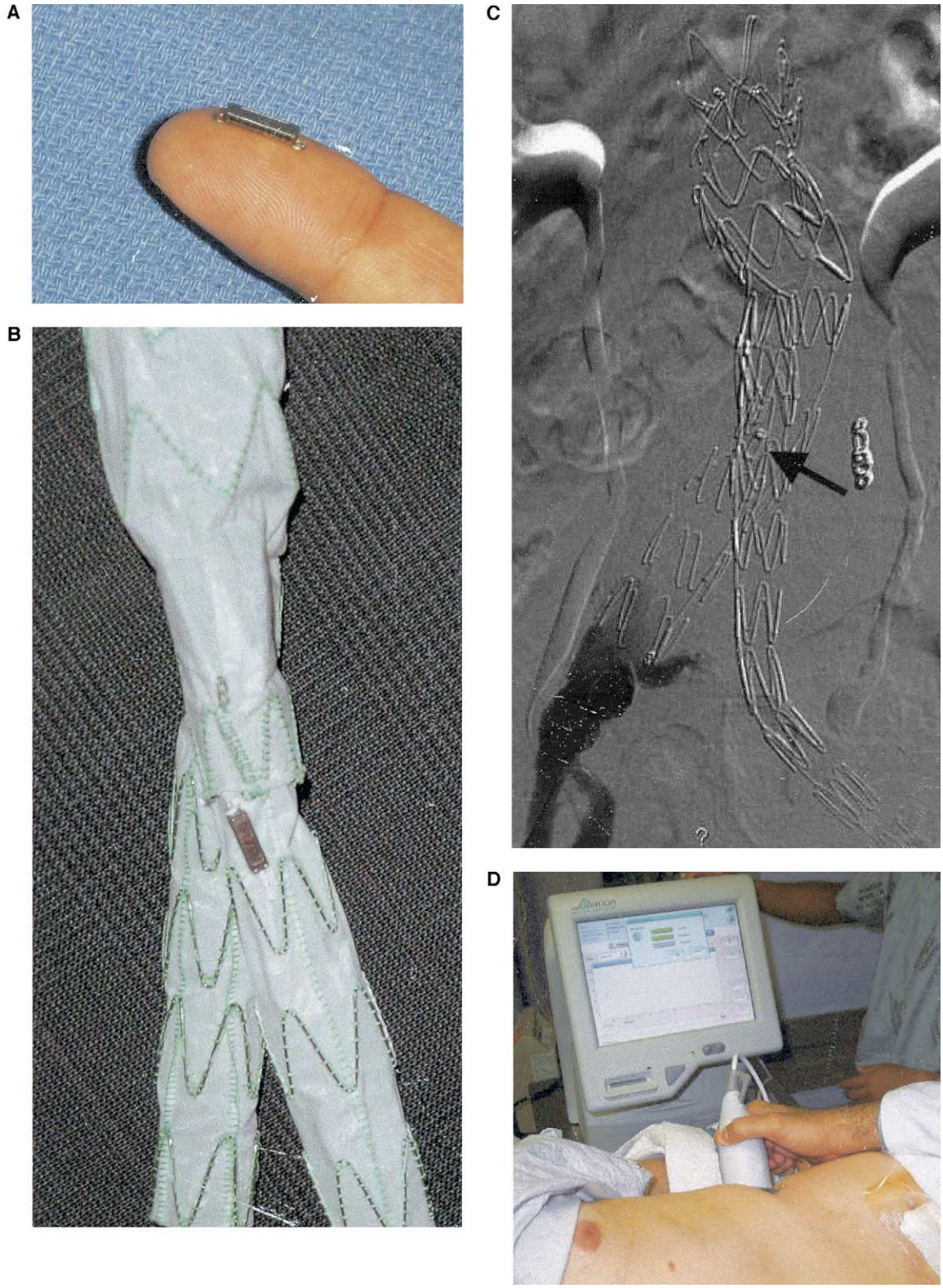

Fig 1. A, Impressure abdominal aortic aneurysm sac pressure transducer (Remon Medical). **B,** Pressure transducer sewn to contralateral limb of bifurcated Talent device. **C,** Digital subtraction angiogram shows distal type I endoleak (*arrow,* pressure transducer). **D,** Transducing intrasac pressures in recovery room after surgery.

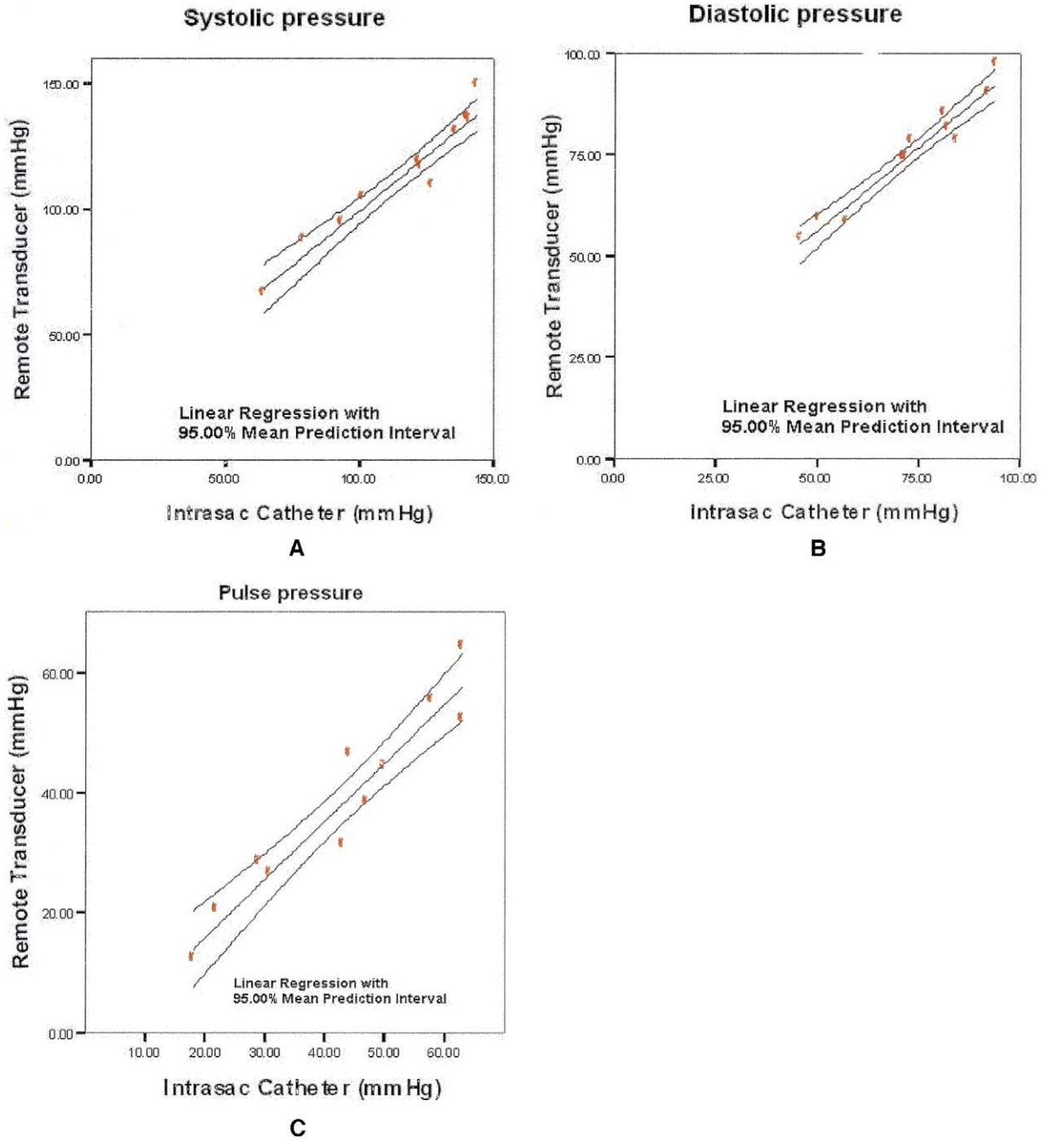

Fig 2. Scatterplots demonstrate concordance between intrasac catheter and ultrasound transducer measurements. **A,** Systolic pressure. **B,** Diastolic pressure. **C,** Pulse pressure. Simultaneous intraoperative measurements were not available for 4 patients.

cient for systolic, diastolic, and pulse pressures was 0.97, 0.97, and 0.96, respectively ($P < .001$).

Effect of increased abdominal pressure

Varying intra-abdominal pressure resulted in an expected variation in intrasac pressure measured with the remote transducer in each patient. This was achieved by having each patient perform the Valsalva maneuver during transduction. This consistently raised the sac pressures by approximately 15 mm Hg. Direct transcutaneous compression of the aneurysm sac during transduction raised the sac pressure by approximately 20 to 30 mm Hg. These observations were used to confirm transducer function in the postoperative period.

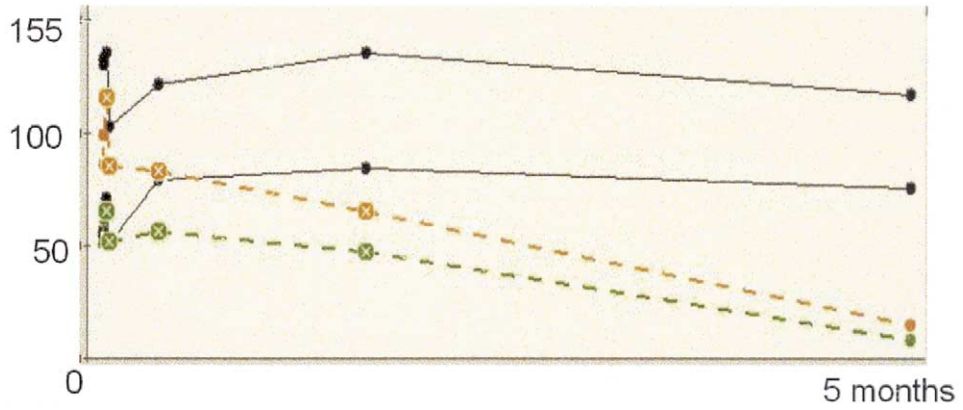

Fig 3. Pressure trends in a patient with completely excluded aneurysm. *Solid lines,* Systemic pressure (systolic and diastolic); *dashed lines,* sac pressure (systolic and diastolic).

Systemic and sac pressures, initially and at 1 and 3 months

		Initial		*1 Month*		*3 Months*	
Patient	*Endoleak*	*Systemic (mm Hg)*	*Sac (mm Hg)*	*Systemic (mm Hg)*	*Sac (mm Hg)*	*Systemic (mm Hg)*	*Sac (mm Hg)*
1	No	103/51	85/52	121/79	83/62	117/75	28/21
2	No	154/46	134/73	190/92	135/87		
3	No	179/68	188/86	178/70	167/82	168/86	24/11
4	No	121/59	106/71	110/75	87/67	118/72	40/35
5	No	171/71	144/95	137/85	113/74		
6	Type II	155/72	133/86	107/62	88/65	137/83	85/67
7	Type I	105/60	108/78	157/66	141/94		
	Type II*			150/65*	106/77*	159/78*	88/65*
8	No	174/81	120/96	120/76	86/75	136/89	18/16
9	No	151/70	145/105	144/77	131/103	135/78	117/96
10	No	155/66	140/82	133/76	111/79		
11	Type II	162/63	128/68	156/70	86/64		
12	No	126/72	89/57	137/89	121/74	137/80	†
13	No	127/52	93/71	135/68	89/59		
14	No	134/42	106/62	125/64	83/57	203/93	151/104

*After repair of type I endoleak.
†Implant no longer transmitting pressure after 1-month follow-up.

Sac pressure over time

Absence of type I endoleak. In patients with no type I endoleak the initial sac systolic pressure was significantly less than the systemic systolic pressure ($P < .001$). Of note, the initial sac diastolic pressure was greater than the systemic diastolic pressure ($P < .001$). The ratio of systemic to sac systolic pressure increased from the time of implantation to 1-month follow-up ($P < .001$). In all patients at 1 month follow-up the sac systolic pressure was higher than the systemic diastolic pressure (Table).

Of the 8 patients followed up for more than 1 month, 6 patients had no endoleak and 2 patients had type II endoleaks. In all 8 patients follow-up pressures were measured at 3 months. The sac systolic pressure decreased to less than the systemic diastolic pressure in 5 patients. Four of these 5 patients had no endoleak; in these 4 patients sac systolic pressure decreased to 40 mm Hg or less at 3 months (Fig 3). The sixth and seventh patients, who had no evidence of endoleak on CT scans, had systemic pressures of 135/78 mm Hg and 203/93 mm Hg, and sac pressures of 117/96 mm Hg and 151/104 mm Hg, respectively, at 3 months. At the present time we have not determined the cause of the increased sac pressures. An eighth patient, whose systemic pressure was 137/83 mm Hg and sac pressure was 85/67 mm Hg at 5 months, was noted to have a type II endoleak originating from a patent IMA and paired lumbar arteries (Fig 4).

Presence of type I endoleak. One patient with a bifurcated endograft had a distal type I endoleak on follow-up cine–magnetic resonance angiography at 1 month. Digital subtraction angiography was performed to better delineate the leak. Systemic pressure at the time was

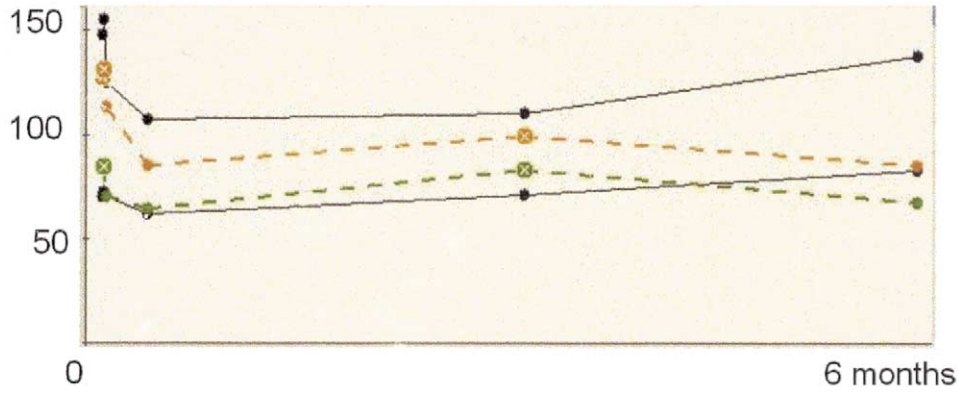

Fig 4. Pressure trends in a patient with type II endoleak. *Solid lines,* Systemic pressure (systolic and diastolic); *dashed lines,* sac pressure (systolic and diastolic).

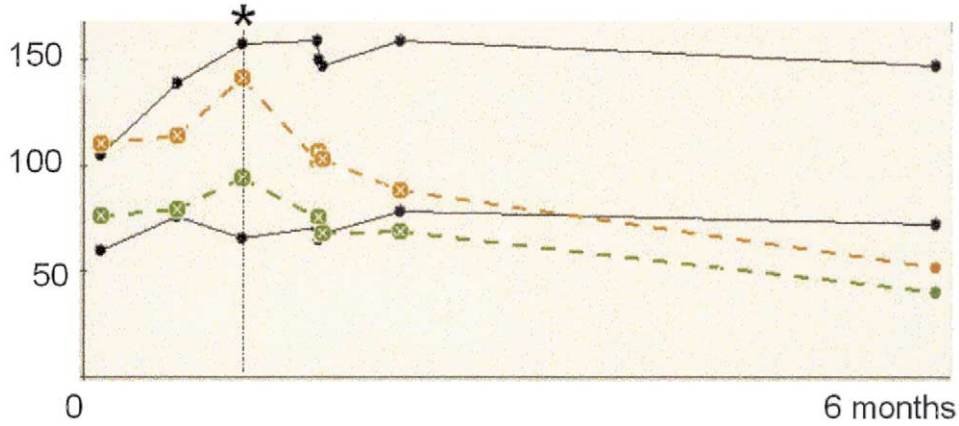

Fig 5. Pressure trends in a patient with type I endoleak that was corrected, now with type II leak. *Solid lines,* Systemic pressure (systolic and diastolic); *dashed lines,* sac pressure (systolic and diastolic). *Time of distal cuff placement.

157/66 mm Hg. The sac pressure measured directly with an intrasac catheter was 135/97 mm Hg, and the simultaneous ultrasound transducer–measured pressure was 141/94 mm Hg. This excellent correlation is also notable in that the catheter was wedged in the endoleak approximately 7 cm from the transducer (Fig 1, *C*). In this patient the type I endoleak was corrected with a distal cuff; however, there is a residual type II endoleak from a patent lumbar artery (Fig 5). Three months after correction of the type I endoleak systemic pressure is 147/72 mm Hg and sac pressure is 52/40 mm Hg.

DISCUSSION

The determinants of sac pressure after EVAR are multifactorial. Patient-related factors include the presence or absence of patent side branches, the nature of the aneurysm thrombus, and the aneurysm anatomy.[5-7] Device-related factors include graft porosity, compliance, and pulsatility.[4] A variety of experimental models have been used to examine the changes in sac pressure associated with EVAR. Early in vivo work with a canine aneurysm model with a chronic indwelling pressure transducer demonstrated that EVAR significantly reduces sac pressures.[4,5] Of note, the degree of sac pressure reduction varied with the degree of graft porosity. In vitro work with a canine AAA model demonstrated that transmission of pressure to the aneurysm wall depends on the nature of the sac contents.[6] Specifically, homogeneous thrombus appears to diminish transmission of pressure, compared with unclotted whole blood. This work also demonstrated that strain is not distributed uniformly to the wall of the aneurysm. This finding has been corroborated in vivo at open aneurysm repair, before aortic cross-clamping.[7]

The relationship of endoleak to sac pressure is less clear. The work by Chong et al,[8] who used a computer-generated 3-dimensional model of a real aneurysm cast in silicone, demonstrated the complex relationship between endoleak type, flow rates, and the resultant intrasac pressure. The presence of a type I endoleak in this model led to systemic

pressures in the sac. When a type II endoleak was present, sac pressure was a function of the inflow pressure. When a type I endoleak was present in conjunction with outflow from a patent side branch, increasing outflow lowered the sac pressure. Others, using synthetic aneurysm models connected to pulsatile pump systems, found that intrasac pressure may be higher than systemic pressure in the presence of endoleaks.[9,10]

This discrepancy in findings highlights the limitations of experimental aneurysm models in mimicking what happens in human beings after EVAR. There have been few studies in human beings that delineated long-term changes in aneurysm sac pressure after EVAR. Using direct translumbar puncture of the aneurysm sac, Sonesson et al[11] demonstrated that in patients with aneurysm exclusion and sac shrinkage mean intrasac pressure diminished to 20% (range, 13%-33%) of mean systemic pressure. Baum et al[12] reported their retrospective analysis of sac pressures in 21 patients. In 4 patients perioperative pressure measurements were obtained through a catheter left in place alongside the stent graft. In 17 patients the aneurysm sac was accessed at angiography to investigate an endoleak demonstrated on the 1-month CT scan. These authors noted elevated sac pressures in all of their patients. It should be kept in mind that these patients were examined early in the postoperative period, and our findings suggest that it takes 3 to 6 months for aneurysm sac pressure to diminish to the levels reported by Sonneson et al.[11]

Our results are consistent with much of what has been shown in experimental models. Initially after EVAR there is diminution in systolic pressure and a rise in diastolic pressure in the sac. The cause of early diastolic sac pressure elevation is uncertain. One potential explanation is that a pulsatile graft in a fluid-filled sac with little outflow will prevent decompression of the sac in diastole. What is clear, however, is that sac pressures evolve gradually, which suggests that thrombus maturation and changes in graft porosity may have a role in pressure diminution over time. The sac pressure in the 3 patients with type II endoleaks decreased over time, yet not so strikingly as in those patients with no endoleaks. In 4 of 6 patients with no endoleak and more than 1-month follow-up sac pressure was reduced to less than 30% of systemic pressure by 3 months. Further follow-up is necessary in the 2 patients without endoleaks who had elevated sac pressures at 3 months. Whether this was the result of endotension has yet to be determined.

Regarding the issue of sac pressure compartmentalization, an interesting finding was noted in the patient with the type I endoleak. Pressures obtained at the origin of the endoleak were equivalent to pressures obtained by the transducer fixed to the device, approximately 7 cm away. While there are limitations to the accuracy of a hydrostatic column pressure transducer (such as the end-hole catheter) wedged in thrombus, the similarity in pressure readings is striking. Experimental work has demonstrated that in the excluded aneurysm with thrombus sac pressure is greatest closest to the graft.[6] The design of our study, with only 1 transducer implanted in each patient, does not enable us to properly answer the question of sac compartmentalization. However, the fact that pressures measured immediately adjacent to the graft diminished suggests that a single transducer may be adequate in surveillance of sac pressures.

CONCLUSION

Chronic, noninvasive sac pressure transduction after EVAR is feasible. Complete aneurysm exclusion can be expected to result in considerable pressure reduction in the aneurysm sac. Further follow-up is needed to delineate the long-term sac pressures in patients with type II endoleaks. Noninvasive pressure transduction shows great promise in the future surveillance of patients after EVAR.

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DISCUSSION

Dr Samuel Money (New Orleans, La). The idea of developing a device that can sense intrasac pressure is one we all have been hoping for. This device is of a marvelous design. It's marvelous in that with its small size it can measure intrasac pressure after being activated by ultrasound. I have but 3 basic questions.

What do the numbers that it generates mean? I reviewed your paper and cannot tell, based on the intrasac pressure, what is going on with the aneurysm. Is the aneurysm expanding, is it stable, or is it contracting? Is it possible that it is too early to know what the individual numbers mean? You refer to sac systolic pressure to systemic diastolic pressure. Is that the number that seems to be the most appropriate to follow?

The second question is one that was thought up with a different device, that is, whether the device gets covered by thrombus or some intimal hyperplasia and stops functioning correctly. Another way to ask that is whether your long-term follow-up values are similar to the values you get in the beginning, or are they reflecting different states?

The third question is the easiest one to answer. How much do these devices cost, how much does it cost to get a pressure reading, and how much is the special equipment needed to use them?

I think this is a device that is truly going to affect what goes on in our treatment of aneurysms.

Dr Sharif H. Ellozy. I am going to answer your question with regard to the validity of the numbers over time. One of the phenomena that our physician's assistant noted, and I am grateful to him for this, is that when you increase intra-abdominal pressure by either having the patient perform the Valsalva maneuver or by directly compressing the aneurysm sac you could induce an elevation in the sac pressure. With compression you can increase it by about 20 to 30 mm, and with the Valsalva maneuver by about 15 mm. We use this as an internal control to make sure the devices are working, so when we measure pressure in these patients 6 months out we then do this. If the sac pressure is 10 mm Hg and we are able to elevate it to 30 mm Hg, we get the sense that this is a real pressure measurement and not simply one that is damped because the transducer is surrounded by thrombus. This is a phenomenon that we found reproducible in all of our patients.

With regard to what the numbers mean, they characterize the patients with no endoleak. As I mentioned, there were 4 of 5 patients whose sac pressures diminished to less than 40 mm Hg at 3-month follow-up. We had 1 patient who had persistent elevation of pressures, and I did not discuss this fully, but this is a sagittal reconstruction from the CT angiogram. You can see here that the patient had a preoperative left renal artery stent placed. The stent graft goes all the way up to the renal artery, so it is as proximal as possible; however, we see that there is some thrombus around the neck. We had originally sized it for the neck farther down, but I do not think it is fully deployed. It may be because it is constrained proximally, and my suspicion is that this patient, although he has no endoleak noted, has transmission of pressure through the thrombus. It will be interesting to see what happens with pressure over time, and I think that the patient is going to require either a large balloon-expandable stent to try to coapt this wall better, or open conversion.

With regard to your third question, it is a private company that produces this. I do not know what the ultimate cost will be. There is probably an up-front cost on the order of $1000 to $2000. However, if this does ultimately lead to better surveillance, you may forego the cost of yearly CT scans, and the surgeon can bill for insonating the device in the office. That is just something to keep in mind.

Dr Charles Sternbergh (New Orleans, La). Your final comments are a perfect segue to my question regarding long-term surveillance. In the abstract you hypothesize that perhaps intrasac pressure measurements could replace CT scanning for surveillance in the future. I have significant concerns about that conclusion. The difficulty with that concept is that you must wait until a late type I or III leak occurs before a potential intervention can be performed. All devices demonstrate some degree of migration. It may vary between devices, but all devices can migrate, and it is clear with some devices an existing endoleak does not predict subsequent rupture. However, so-called insecure fixation has been noted retrospectively to be apparent in many patients in whom the aneurysm has gone on to rupture. Are you really comfortable with the concept of just watching for changes in intrasac pressure? Don't you want to monitor for migration of the device?

Dr Ellozy. That's a very good question. One of the things we can do to look for migration is 4-view plain films of the abdomen. The other thing is, these are very preliminary data. We are still finding out what the pressure means, so by no means do I suggest that we are ready to do this yet. However, I think with longitudinal follow-up we may find that this is a useful tool in surveillance.

Dr Sternbergh. I do not think that many people here would think that plain KUBs, no matter how many views you take, are going to be useful in measuring anything but very, very large degrees of migration.

Dr Mitchell Goldman (Knoxville, Tenn). First, I congratulate you on confirming what we have noticed in the laboratory, which is essentially 3 principles. One, when you do completely exclude the aneurysm you do get diminution of sac pressure. Two, with possible endotension you get persistent rise in sac pressure. Three, when you have a type 1 endoleak you get systemic pressure. This is a phenomenal corroboration of what has been seen in the laboratory and taken to the clinical venue.

My question is, have you actually in that patient done an arteriogram and looked for any runoff, because if you postulate that you do have endotension there may be some indication that you have irregularity at the attachment site.

Dr Ellozy. At our institution, one of our radiologists has gotten a lot of experience with endoleaks and endotension. In those patients in whom we do not find endoleak on CT scans or angiograms, they do an MR sequence called a true FISP, and it is able to differentiate between liquid and organized thrombus. We have found that in our patients with endotension, enlarging sacs with no evidence of endoleak, they show up as liquid on this true FISP sequence. So this patient is actually going to be brought in for an MR with this sequence. If it is indeterminate, the patient is going to undergo angiography.