◆ EXPERIMENTAL INVESTIGATION ◆

Modeling Endoleaks and Collateral Reperfusion Following Endovascular AAA Exclusion

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Purpose: To investigate the effect on intrasac pressure of stent-graft deployment within a life-size silicone rubber model of an abdominal aortic aneurysm (AAA) maintained under physiological conditions of pressure and flow.
Methods: A commercial bifurcated device with the polyester fabric preclotted with gelatin was deployed in the AAA model. A pump system generated physiological flow. Mean and pulse aortic and intrasac pressures were measured simultaneously using pressure transducers. To simulate a type I endoleak, plastic tubing was placed between the aortic wall and the stent-graft at the proximal anchoring site. Type II endoleak was simulated by means of side branches with set inflow and outflow pressures and perfusion rates. Type IV endoleak was replicated by removal of gelatin from the graft fabric.
Results: With no endoleak, the coated graft reduced the mean and pulse sac pressures to negligible values. When a type I endoleak was present, mean sac pressure reached a value similar to mean aortic pressure. When net flow through the sac due to a type II endoleak was present, mean sac pressure was a function of the inlet pressure, while pulse pressure in the sac was dependent on both inlet and outlet pressures. As perfusion rates increased, both mean and pulse sac pressures decreased. When there was no outflow, mean sac pressure was similar to mean aortic pressure. In the presence of both type I and type II endoleaks, mean sac pressure reached mean aortic pressure when the net perfusion rate was low.
Conclusions: In vitro studies are useful in gaining an understanding of the impact of different types of endoleaks, in isolation and in combination, on intrasac pressure after aortic stent-graft deployment.

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Key words: abdominal aortic aneurysm, in vitro model, sac pressure, stent-graft, endoleak

The primary purpose of endovascular repair of abdominal aortic aneurysm (AAA) is to prevent death from aneurysm rupture. As with conventional open repair, the aim of intervention is to isolate the aneurysm from the aortic or systemic pressure while maintaining flow to the lower limbs. In theory, the consequent fall in aneurysm sac pressure should result in a progressive reduction in aneurysm diameter and thus prevent rupture. However, in a significant proportion of patients, the aneurysm does not decrease in size but continues to expand following successful implantation of an aortic stent-graft.[1–4] In some patients, the apparent failure of the aneurysm sac to shrink may be explained by a persistent communication between the systemic circulation and the aneurysm sac—an endoleak. Less

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commonly, no endoleak is observed on contrast-enhanced computed tomography (CT) or angiography,[5,6] and yet the aneurysm continues to expand and rupture.[4,7] There are also reports of aneurysm sac shrinkage despite the presence of endoleaks.[8–10]

Several types of endoleaks have been classified as graft-related, specifically, leaks at the proximal or distal endograft attachment sites (type I), those arising from material failure or disjunction of the component parts of a modular graft (type III), and still another related to high graft porosity (type IV).[11,12] Type II, which results from backfilling of the aneurysm sac via patent lumbar (LA) or inferior mesenteric arteries (IMA), is a non-graft-related endoleak.[11,12] It has been suggested that type II endoleaks are of little clinical consequence because they are ''low pressure'' and/or ''low flow'' endoleaks. However, this assumption is controversial owing to several conflicting reports. Some studies have shown that the aneurysm sac is subjected to systemic pressure as a result of retrograde flow from the IMA,[13,14] while others have demonstrated that the size of the aneurysm does not increase or decrease in the presence of type II endoleaks.[15,16] Still other authors have reported that a significant number of patients with persistent side branch endoleaks show shrinking aneurysms.[8] Thus, it is difficult to draw any correlation, explanation, or conclusion from observing the progression or regression of the aneurysm sac based on the presence or absence of endoleaks.

Based on Laplace's law, it can be predicted that increasing intrasac pressure or aneurysm sac diameter should increase wall tension, thus inducing progressive aneurysm expansion until rupture. Therefore, an understanding of the determinants of aneurysm sac pressure after endovascular exclusion will allow us to better identify those parameters important to the behavior of the aneurysm, in particular, in relation to endoleaks. We therefore carried out an in vitro study to investigate the effect on intrasac pressure of stent-graft deployment within a life-size AAA model maintained under physiological conditions of pressure and flow. This type of model permitted accurate simulation of known clinical situations, with careful control of all relevant conditions and precise analysis of the pressure effects of various types of endoleaks, both individual and combined.

METHODS

AAA Model

The experiments were carried out using a silicone rubber model of a patient's AAA; details of its construction using a rapid prototyping technique have been described elsewhere.[17] Briefly, the geometrical data were obtained from preoperative axial CT scans and angiography of an 84-year-old man who was scheduled for endovascular repair. A 3-dimensional computer model of the arterial tree from the level of the celiac axis to the femoral arteries was produced using a CAD software package (DUCT 5.3; Delcam International, Birmingham, UK). A solid master model was then produced by laser stereolithography, from which a 2-part mould was fabricated. Injection of molten wax into the mould created a replica of the AAA, which was then retrieved and embedded in optically clear silicone rubber. Once the silicone rubber had cured, the wax was melted out, leaving a transparent flow-through model of the aneurysm. Fluid inlet and outlet connections to the suprarenal aorta and renal and femoral arteries were made via silicone rubber tubing embedded within the model. In addition, 2 segments of 6-mm silicone rubber tubing were inserted laterally to the aneurysm sac to represent patent IMA and LAs. Pressure ports in the suprarenal aorta and in the aneurysm sac were also provided.

Endovascular Device and AAA Exclusion

A commercially available bifurcated device (Vanguard Endovascular Aortic Graft System; Boston Scientific, Natick, MA, USA) was used in the experiments. This device, made from a self-expanding nitinol stent covered with a thin woven polyester fabric, consisted of a main stent-graft measuring 26×165 mm and a 12×105-mm contralateral extension limb. In the in vivo situation, thrombus forms on and within the walls of the porous graft, rendering it impervious to blood. To simulate this in the

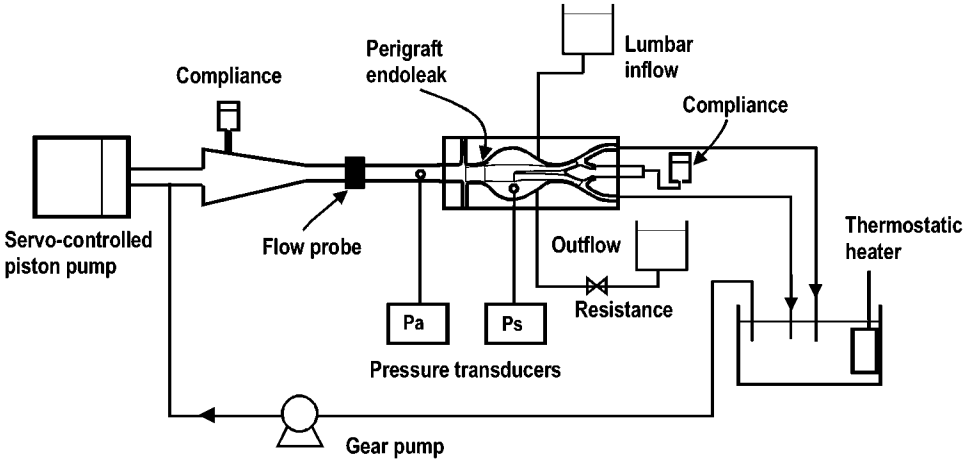

Figure 1 ◆ Diagram of the experimental model.

model and thereby isolate the aneurysm sac from arterial pressure, the device was preclotted with a 12% (w/w) aqueous gelatin solution, which was maintained at 40°C. This solution was injected under pressure into the lumen of the stent-graft with the outlets closed to allow it to permeate through the fabric. The stent-graft was then held vertically in ambient air to allow excess gelatin to drain off. After visual inspection to ensure complete coverage of the graft surface with a glistening layer of gelatin, the entire device was submerged in a 10% formaldehyde solution for about 25 minutes to cross-link and stabilize the gelatin. The stent-graft was then flushed under running tap water and stored in cold water until it was deployed in the AAA model.

After the stent-graft was in place in the model, perigraft endoleaks were simulated by placing 2 4-cm-long plastic tubing segments with internal diameters of 1.3 and 4 mm, respectively, between the aortic wall and the stent-graft at the proximal anchoring site. These tubes could be sealed off and opened as desired, providing communication channels between the systemic circulation and the aneurysm sac. The diameters of the tubes determined the degree of endoleak. Warm water was circulated through the stent-graft to allow it to expand and provide secure fixation at the proximal and distal ends. With the trunk and contralateral limb deployed, the lumen of the complete device was flushed with warm gelatin solution, followed by cross-linking with formaldehyde. Finally, with the communication channel blocked and the aneurysm sac opened to the atmosphere, the system was pressure tested to ensure that there was no endoleak.

The Perfusion System

A pulsatile flow generator was used to produce physiological flow and pressure waveforms in the AAA model. Details of the flow circuit (Fig. 1) can be found in Chong et al.[17] The circulating fluid was an aqueous 40% (w/w) glycerol solution. To minimize the erosion of gelatin from the luminal surfaces of the stent-graft, this fluid was maintained at 20°C, which gave a dynamic viscosity of 3.7×10^{-2} Pa·s, which is similar to that of blood. All measurements were carried out under resting condition at a heart rate of 60 beats/minute and a mean aortic flow rate of 1.6 L/min. The peripheral resistances and compliances of the flow circuit were adjusted to give physiologically relevant peripheral arterial flow distributions and a pressure of 125/75 $\pm$5 mmHg.

Pressure Measurement

Aortic (Pa) and intrasac (Ps) pressures were measured simultaneously using physiological pressure transducers (LUER; Gaeltec Ltd., Scotland) placed into the respective pressure port via 3-way valves. The pressure signals, which were amplified via a dual-channel am-

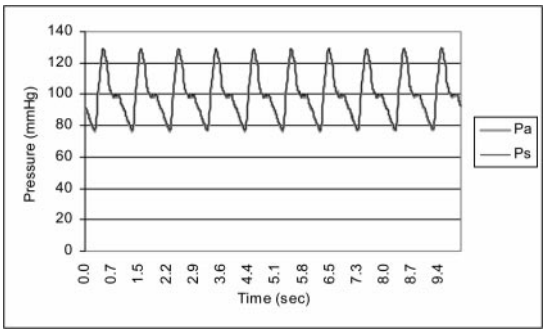

Figure 2 ◆ The pressure waveforms recorded in the aorta (Pa) and aneurysm sac (Ps) before the deployment of a stent-graft.

plifier (S7B/2, Gaeltec Ltd.) and acquired using an analogue-to-digital converter (K575, Keithley Instruments Inc., Cleveland, OH, USA), where stored in a microcomputer. Pressures were recorded over 10-second periods at a rate of 200 samples per second. Three consecutive data samples were obtained at intervals of about 1 minute. Before the deployment of stent-graft, the pressure recording system was tested by acquiring Pa and Ps. As expected, these pressures were essentially the same (Fig. 2), confirming the reliability of the measurement system.

Model I

This model was designed to investigate the effect on Ps when the aneurysm sac was totally or partially excluded by the stent-graft. Pa and Ps were first assessed after the deployment of the stent-graft as received, i.e., without gelatin coating and subsequently with the deployment of the gelatin-coated graft for the following conditions: (1) complete AAA exclusion, i.e., no endoleak and (2) partial exclusion with different endoleaks created by localized removal of gelatin from the graft fabric in the trunk. These endoleak rates were determined by time-collection of the perfusate exuding from the sac through the pressure port when it was opened to atmosphere. For each experiment, the pressure port was closed just before the measurement of Ps, and pressure signals were recorded until the pressure had stabilized.

Model II

This model investigated the change in Ps in response to different perfusion rates into the aneurysm sac through 2 aortic side branches. In this case, there was no communication between the aneurysm sac and the aortic flow. Each of these branches was connected to an inflow and outflow vascular bed maintained at different pressures by constant-head reservoirs. Adjusting the distal resistance controlled the flow rates in the outflow branch.

Model III

This model was set up to examine the change in Ps when both perigraft endoleak and a patent aortic side branch were present. To examine the effect of backpressure, the outflow pressure was set to 45 or 70 mmHg by means of the constant-head reservoir. Altering the resistance of the outflow branch set the perfusion rate.

RESULTS

Effect of Partial and Total Aneurysm Exclusion

With the deployment of the uncoated stent-graft, there was very little difference between the mean sac pressure (MPs) and mean aortic pressure (MPa) (Fig. 3). When a coated graft was deployed and no endoleak was present, MPs was reduced to 2.5 mmHg. In the presence of an endoleak, MPs increased gradually upon closure of the pressure port from the atmosphere, eventually reaching a value similar to MPa; the greater the endoleak, the more rapid the increase in MPs. The pulse pressure in the sac (PPs) was reduced slightly after deployment of the uncoated stent-graft. With a gelatin-coated graft in place, a greater reduction in PPs was observed, reaching a value of 5 mmHg when the sac was completely excluded.

Effect of Patent Aortic Side Branches

As expected, when there was only a patent inlet to the aneurysm sac, MPs was equal to the inlet pressure (Fig. 4A). When a communicating branch was present, resulting in a net

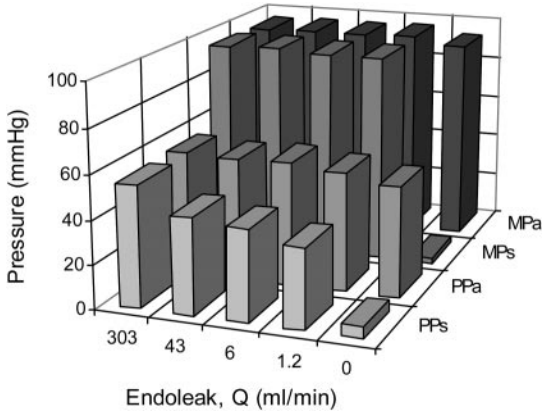

Figure 3 ◆ The change in mean (MPs) and pulse (PPs) pressures in the aneurysm sac compared to the mean (MPa) and pulse (PPa) aortic pressures as a function of endoleak through the graft by the removal of "thrombus." (Q=303 mL/min is the endoleak rate through the uncoated graft.)

flow through the sac, MPs decreased as the perfusion rates increased. The reduction in MPs was independent of inflow and outflow pressures (Table 1). These results also show that MPs is dependent only on the inlet pressure. Pulse pressure also decreased as the perfusion rate increased, and the actual PPs was dependent on both the inlet and outlet pressures.

Effect of Perigraft Endoleak and a Patent Aortic Side Branch

When there was no outflow, MPs was similar to MPa irrespective of the endoleak size. As the outflow increased, MPs was reduced (Fig. 4B). MPs reduction was dependent on the size of perigraft endoleak and independent of the outlet pressure. A larger perigraft

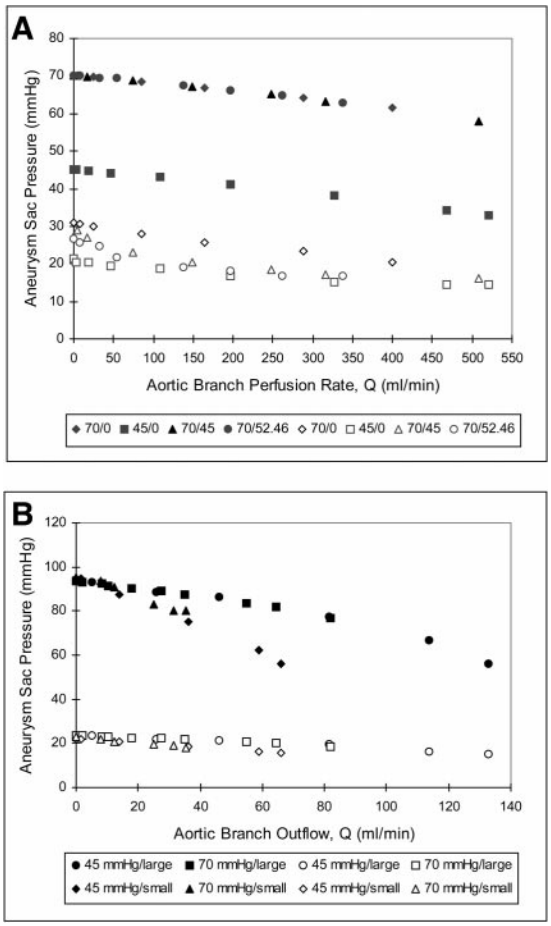

Figure 4 ◆ (**A**) Mean (closed symbols) and pulse (open symbols) pressures in the aneurysm sac as a function of aortic branch perfusion with the inlet and outlet at different pressures. (**B**) The mean (closed symbols) and pulse (open symbols) pressures in the aneurysm sac as a function of aortic branch outflow with distal pressure of 45 and 70 mmHg and in the presence of a small or large perigraft endoleak.

TABLE 1
Ratio of Change in Aneurysm Sac Pressure to Change in Flow (ΔMPs/ΔQ) at Different Inflow and Outflow Pressures

	Inflow/Outflow Pressure, mmHg			
	70/0	70/45	70/52.5	45/0
ΔP, mmHg	70	25	17.5	45
ΔP/ΔQ, mmHg/mL/min	−0.026	−0.021	−0.024	−0.025
R^2	0.991	0.996	0.992	0.992
Constant, C	70.8	70.2	70.5	45.5

TABLE 2
Ratio of Change in Aneurysm Sac Pressure to Change in Flow (ΔMPs/ΔQ) at Different Outflow Pressures and Perigraft Endoleak Sizes

	Small Leak		Large Leak	
P, outflow, mmHg	45	70	45	70
ΔP/ΔQ, mmHg/mL/min	−0.61	−0.54	−0.31	−0.21
R^2	0.996	0.977	0.964	0.983
Constant, C	95.9	96.9	97.1	94.1

endoleak resulted in higher MPs. Assuming a linear pressure flow relationship, the reduction in MPs with a large perigraft endoleak was about half that of a small perigraft endoleak at the same outflow pressure (Table 2). A similar trend was observed in PPs, albeit at a much lower rate.

DISCUSSION

Currently, postoperative assessment following endovascular AAA exclusion is mainly restricted to the detection of endoleaks and changes in AAA morphology, which thus far have proved unreliable in preventing aneurysm rupture. While pressure measurements are increasingly being recognized as the more accurate indication of AAA exclusion, the lack of a suitable noninvasive technique for measuring pressure reliably in the long term, together with incomplete knowledge about the transmission and distribution of pressure within the sac, makes this approach impractical at present. So far, the difficulties associated with short-term in vivo studies of intrasac pressure have led to conflicting results from both clinical and animal experiments. In light of this situation, we have adopted an in vitro approach with a system that allowed us to carefully control specific conditions and make accurate measurements.

We first assessed type IV endoleak related to the stent-graft. The results from Model I show that mean sac pressure remained comparable to mean aortic pressure when this type of endoleak was present, indicating failure of the deployed stent-graft to protect the aneurysm. On the other hand, MPs was reduced to 2.5 mmHg when the sac was completely excluded. It has been reported that AAAs with persistent endoleaks could either rupture[18] or expand at a rate similar to those of untreated aneurysms of comparable size.[19] Our results are also in accord with the observation made by Sanchez et al.[20] of high sac pressure in a canine model when a highly porous polytetrafluoroethylene graft was used. High porosity, in this case, is due to incomplete coverage of thrombus on the graft or a too diffuse covering, allowing transmission of intraluminal pressure into the sac, which may explain the AAA rupture in a patient given anticoagulation therapy.[21] Furthermore, thrombus itself may not prevent the transmission of pressure in the presence of multiple suture holes in the fabric of some stent-grafts.[4,22] In these circumstances, continued expansion and aneurysm rupture without documented endoleaks may be related to endoleaks that are too small to be detected by the currently available techniques (microendoleaks).[13,23] Apparent continued or recurrent pressurization of the sac after endovascular repair led to the definition of "endotension."[2,4] Reduction in MPs after complete AAA exclusion is likely to lead to gradual sac regression.[24]

Damping of PPs after stent-graft deployment, where the gelatin coating closely simulated the effect produced by thrombus formation, was also observed in our study and others.[20,25] This is also in agreement with report of Paty et al.,[26] who studied exclusion and bypass of aneurysms.

Although it is generally recognized that the presence of type I endoleak is a technical failure of endovascular AAA repair, the clinical significance of patent aortic side branches on intrasac pressure has not been adequately defined. These branches can act as afferent vessels feeding into the sac or as efferent vessels, permitting outflow from perigraft or graft-related endoleaks or from other afferent

aortic side branches. Some of these endoleaks may seal spontaneously, while others may require secondary intervention, including conversion to open surgical repair. In many of these endoleaks, outflow through one or more aortic branches may be involved. In vivo, a small perigraft channel may be expected to thrombose and seal off the aneurysm sac, but this may not occur with large endoleaks, especially if there is also an outflow channel.[27] Model II shows that when the aneurysm sac was perfused by patent aortic side branches, mean sac pressure was dependent on the pressure in the inflow vessel, while pulse pressure in the sac was dependent on both this pressure and the pressure in the outflow vessel. In vivo, mean and pulse sac pressures are likely to be influenced by the pressure in the larger aortic branches, e.g., the IMA, which has a stump pressure of about 66 mmHg.[28,29] The results indicate that the perfusion rate is a secondary factor in determining mean sac pressure, as its drop is only about 5 mmHg for a flow rate of 250 mL/min, which is unlikely in vivo.

Malina et al.[30] observed that aneurysms perfused from patent aortic side branches, one from the IMA and the other from LA, expanded postoperatively; once these branches were closed, the sac size reduced. In another report, Gorich et al.[31] demonstrated an increase in the aneurysm sac diameter associated with the lumbar endoleaks. In fact, the aneurysm sac pressure as a result of retrograde IMA flow could be as high as the systemic pressure.[13,14] However, it should be noted that it is not clear if such high pressure is influenced by undetected type II endoleaks from sources other than the IMA and LAs. Aneurysm rupture has been reported as a result of patent LAs and accessory renal arteries[18] or even from a patent accessory renal artery alone.[10] Furthermore, there are cases in which type II endoleaks from collateral arteries do not have significant effect on the size of the aneurysm,[15,16] while in others, shrinkage may occur.[1]

The type I endoleak simulated in Model III is recognized as the most dangerous. Our results indicate that mean sac pressure is similar to mean aortic pressure when there is no outflow. These endoleaks will continue to pressurize the sac in a manner similar to a pseudoaneurysm. When both type I and type II endoleaks are present, the sac pressure is dependent on the size of the type I endoleak as well as the perfusion rate due to the type II endoleak. The larger the type I endoleak, the higher the mean and pulse sac pressures and the slower the drop in pressure as the perfusion rate increases. However, the pressure in the outflow vessel determines the minimum value of the mean sac pressure. Englund et al.[28] and Shigematsu et al.[29] found that the flow rate in the IMA was about 37.5 ± 8.7 mL/min. From Figure 4B, the mean sac pressure associated with this flow range was between 75 and 87 mmHg, which may be sufficient to cause continued expansion of the aneurysm sac.[27,30]

These in vitro models provide an understanding of the effect of endoleak and aortic branch perfusion on intrasac pressure and help to resolve some of the conflicting in vivo observations. However, it is important to note that morphological aneurysm changes under the influence of pressure may also be dependent on the structure of the aneurysm wall. An aneurysm wall with highly altered structure may be less able to withstand even a small pressure and thus may continue to expand and rupture. Others may require considerable pressure to result in any noticeable dimensional change. Any residual pressure in the sac may pose a threat of further expansion and rupture if the wall structure is changed.

While the results of these experiments help to explain some of the discrepancies noted from clinical observations, they do not enable us to predict accurately which endoleaks require intervention. Clinical observations tell us that all type I and type III endoleaks require urgent secondary intervention, a policy that is supported by our results. However, changing sac morphology with continued or recurrent expansion continues to be the principal indication for secondary intervention in the presence of type II endoleaks.

In conclusion, we have shown that intrasac pressure is dependent on a number of factors, which include the perfusion rate into and out of the aneurysm sac and the pressure in the vascular beds associated with the afferent

and efferent vessels. Although large endoleaks are dangerous, they can usually be easily detected and may therefore be corrected through reintervention. On the other hand, small endoleaks may occur transiently and often are more difficult to detect by imaging. They can result in high intrasac pressure and are thus capable of causing endotension and rupture, but clinical experience to date suggests that this rarely occurs.

These in vitro studies provide a basic understanding of the individual effect of aortic branch perfusion, perigraft endoleaks, and graft-related endoleaks, as well as their combined effects on intrasac pressure after stent-graft repair of AAA. The true mechanisms, however, may be better understood if long-term monitoring of intrasac pressure in vivo is carried out, but the means of achieving this are not available at present.

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