A Novel Treatment Using an Intraventricular Stent Graft for Postinfarction Ventricular Septal Rupture in a Porcine Model

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Objective: Ventricular septal rupture (VSR) is a severe complication of acute myocardial infarction, and the conventional technique for repair is associated with high operative mortality. A novel intraventricular stent graft (IVSG) device was tested as a less invasive treatment for VSR; it does not require cardiopulmonary bypass, cardiac arrest, or left ventricular incision. Its effectiveness was assessed using animal experiments.

Methods: Six pigs were placed on cardiopulmonary bypass. The VSR model was created by making a hole in the interventricular septum via the right ventricle. Animals were weaned off the bypass. The sheath encasing the device was advanced over the guidewire, and the IVSG was placed in the left ventricle. Before and after rupture creation and after device deployment, left ventriculography was performed, hemodynamic data were collected, and Qp/Qs values were measured.

Results: All procedures were completed safely. The left-to-right shunt seen after rupture creation disappeared after device deployment. The pulmonary-to-systemic blood flow ratio after rupture was 3.35 ± 1.00, decreasing significantly to 1.09 ± 0.10 after device deployment ($P = 0.007$). Hemodynamic instability after rupture creation improved dramatically after deployment.

Conclusions: The use of our new IVSG in this VSR animal experiment model significantly decreased the left-to-right shunt. The new device was able to control the acute heart failure associated with VSR with a minimally invasive procedure during the hyperacute phase of heart failure. Potential improvements in VSR treatment outcomes are expected with its clinical application.

Key Words: Ventricular septal rupture, Acquired ventricular septal defect, Intraventricular stent graft, Acute myocardial infarction.

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Ventricular septal rupture (VSR) is a complication observed in 0.2% to 2% of acute myocardial infarctions and can lead to severe, acute, bilateral heart failure due to the onset of a severe left-to-right (L-R) shunt.[1] The mortality rate with medical therapy alone is 50% within 1 week and as high as 90% within 1 month.[2,3] Currently, intracardiac repair by inducing cardiac arrest with thoracotomy and extracorporeal circulation is the most common procedure; however, the risk associated with this surgery is equivalent to that in the acute phase of acute myocardial infarction. The surgical procedure itself is difficult because of preoperative factors, such as very poor cardiac function, excessive invasiveness, and the instability of the repair; the operative mortality rate is 20% to 87% despite advances in surgical techniques.[1–5] We think that the factors related to the poor results with the conventional methods are the excessive invasion of extracorporeal circulation in the acute phase of myocardial infarction and the cardiac dysfunction due to cardiac arrest during the operation and left ventricular (LV) direct incision. We contrived a new, less invasive treatment option for VSR that does not require extracorporeal circulation, cardiac arrest, or LV incision.

The salient features of this new device, including VSR closure from the LV, catheter insertion in a beating heart, its ability to follow LV wall movement because of its flexible structure, and decreased interference with the mitral valve chordae tendineae, are presented. Previously, we reported a case of the clinical application of this technique based on this research.[6] In the present study, its effectiveness as a new surgical procedure for VSR was evaluated in animal experiments.

MATERIAL AND METHODS

Construction of the Intraventricular Stent Graft

The intraventricular stent graft (IVSG) was designed to fit the LV structure of a pig weighing 60 to 70 kg. The design process involved the development of several different shapes and the selection of the optimal shape in pig-heart model experiments. Because stent graft interference with mitral valve tissue results in severe mitral regurgitation (MR), a slant cup shape was adopted, which decreased the interference with the mitral valve. The results of these basic animal experiments are currently being presented in a separate article.

The measurements required to determine the size of the IVSG were left ventricular dimension diastolic (LVDd), short side length, and long side length (Figs. 1A, B). The device diameter was established as approximately 1.1× LVDd. The diameter of the device used in this study was 36.6 ± 2.4 mm. The IVSG was set to the desired axis of implantation between the cardiac

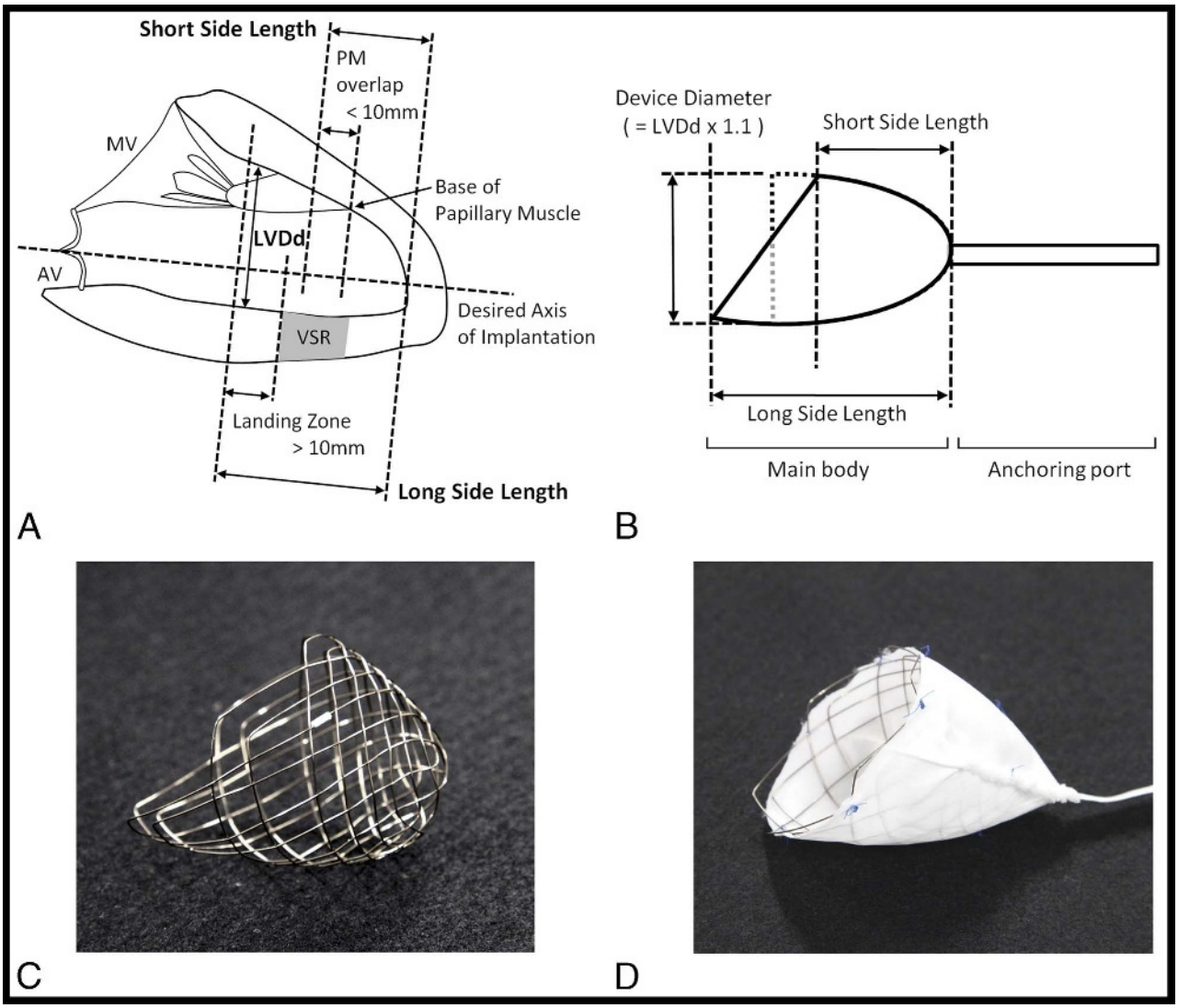

FIGURE 1. Design of the IVSG. A, B, Three measurements of LVDd, short side height, and long side height are needed to choose the optimal size of the IVSG. The device diameter was set up as approximately 1.1× LVDd. The short side extends from the cardiac apex to overlap 10 mm from the base of the papillary muscle. The long side extends from the cardiac apex to 10 mm or more from the outflow side edge of the VSR. The 10-mm zone above the VSR is termed the landing zone. The IVSG end toward the cardiac apex has a tubular anchoring port to facilitate the passage of devices such as guidewires. This port is ultimately used to fix the device to the cardiac apex. C, The framework for the IVSG is a superelastic nitinol stent shaped in a slant cup style. D, The IVSG is covered by polyester woven graft.

apex, where the stent was placed, and the center of the aortic valve. The lengths of the short and long sides were measured using a line that crossed this axis at right angles. The short side extended from the cardiac apex to overlap 10 mm from the base of the papillary muscle. The long side extended from the cardiac apex to 10 mm or more from the outflow side edge of the VSR. The 10-mm zone above the VSR was termed the landing zone. The IVSG end toward the cardiac apex had a tubular anchoring port to facilitate the passage of devices such as guidewires. This port was ultimately used to fix the device to the cardiac apex.

The framework for the IVSG is the Matsui-Kitamura (MK) stent (Fig. 1C), which is made from a single 0.35-mm-diameter superelastic alloy wire (Memoalloy; Tokin Inc, Tokyo, Japan) containing 51% nickel and 49% titanium by weight, and having a transformation temperature of less than 0°C. The nitinol wire was wound in a spiral fashion around a cup type stainless steel mandrel. The stent was annealed at 350°C in an oven for 25 minutes and then cooled to room temperature. The nitinol framework was then removed from the mandrel, and both ends of the mono wire were connected with a small, compressed platinum cylinder.

The intraventricular stent was covered with a seamless, cup-shaped, woven graft made of polyester fabric (Kitamura Manufacturing, Niigata, Japan) with a thickness of 0.2 mm (Fig. 1D). The porosity of the polyester fabric was approximately 100 mL/min/cm^2. The graft was attached to the stent with 5–0 interrupted polypropylene sutures. Platinum markers were attached to the anterior and posterior sides of the graft with 5–0 interrupted polypropylene sutures.

The IVSGs of diameter of 40 mm or less were placed in an 18-F sheath (Flexor Keller-Timmermans Introducer; Cook Medical, Bloomington, IN USA) to facilitate delivery. The IVSGs of diameter of more than 40 mm can be passed in a 20-F sheath.

Operative Procedure

This procedure was performed on 6 consecutive Yorkshire pigs. All animals received humane care in compliance with the Guide for the Care and Use of Laboratory Animals (www.nap.edu/catalog/5140.html) at the Takaramachi Campus of Kanazawa University.

The 6 pigs [mean ± standard deviation (SD) weight, 66.8 ± 4.5 kg] were sedated by means of an intramuscular injection of ketamine (10 mg/kg body weight). Anesthesia was maintained with 0.5% to 1.5% halothane in a mixture of oxygen, nitrous oxide gas, and intravenously injected propofol (1–2 mg/h/kg body weight) and pancuronium bromide (0.04 mg/kg body weight). The rate and volume of positive pressure ventilation (KMA-1300 IIS; ACOMA, Tokyo, Japan) were adjusted to maintain the arterial blood gases within the normal physiological range. Electrocardiographic monitoring was performed throughout the procedure. A median sternotomy was performed, and the pericardium was opened longitudinally along the midline. A cannula was placed in the common femoral artery, the main pulmonary artery, and the superior vena cava, and the systemic arterial pressure, pulmonary arterial pressure, and central venous pressure were monitored continuously. A PowerLab data acquisition system (AD Instruments, Milford, MA USA) was used for monitoring. The animal was then heparinized by injecting 200 IU/kg of heparin. Cardiopulmonary bypass was established with an aortic cannula and bicaval cannulation. A venting catheter was inserted into the right upper pulmonary vein. Continuous injection (2–15 μg/kg/min) of dopamine hydrochloride was necessary in all cases. If arrhythmias were easily induced before and after VSR creation, landiolol was used as a β-blocker to prevent atrial and ventricular arrhythmias before and after VSR creation.

Ventricular Septal Rupture Model

A longitudinal incision was made on the anterior surface of the right ventricle (RV) under total extracorporeal circulation, with the heart still beating. A VSR was created using a 20-mm puncher in the interventricular septum close to the RV apex (Fig. 2A). After confirming the size of the resected septal myocardium, the RV with the longitudinal incision was closed with a running 5–0 polypropylene suture. After VSR creation, the animals were weaned off cardiopulmonary bypass (CPB) temporarily.

Intraventricular Stent Graft Deployment

Intraventricular stent graft deployment was carried out under fluoroscopy. A 0.032-in guidewire (Terumo, Tokyo, Japan) was introduced into the LV apex and advanced across the aortic valve into the ascending aorta (Fig. 2B). A 5-F sheath (Medikit, Tokyo, Japan) was then inserted. The 5-F angiographic catheter was subsequently placed over the guidewire for left ventriculography (LVG). A 0.035-in Extra-Stiff Amplatz guidewire (Cook Medical) was introduced into the ascending aorta. After passing the guidewire through the IVSG center, the latter was placed in an 18-F sheath. The delivery system was advanced into the LV via the LV apex over the guidewire. Intraventricular stent graft deployment was performed using the anterior platinum marker fixed to the stent to correct the direction of the stent graft to match the direction of the VSR in the septum (Fig. 2C). No touch-up by balloon was performed after deployment. The anchoring ports of the IVSG penetrated the cardiac apex in one case. In this case, the cardiac apex was closed using purse-string sutures with felt pledgets. The anchoring port should be fixed to the LV apex when the IVSG has to be retained; however, fixation was not performed in this experiment.

Hemodynamics

The systemic arterial pressure, pulmonary arterial pressure, central venous pressure, and pulse rate were monitored continuously. Measured values were analyzed using the analysis software, LabChart7 (AD Instruments). "Control" refers to the

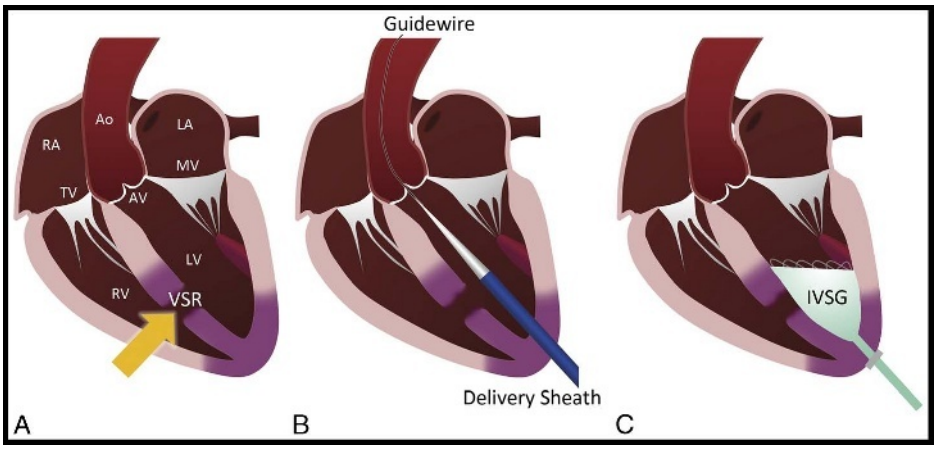

FIGURE 2. The VSR model and the deployment of the IVSG. A, An anterior-type VSR is created with a right ventricular approach. B, The guidewire is introduced into the (LV) apex and advanced across the aortic valve into the ascending aorta. The delivery sheath is advanced into the LV via the LV apex over the guidewire. C, Deployment of the IVSG into the LV via the apex. The anchoring port for the IVSG extends from the cardiac apex. The apex is closed by purse-string suture with felt pledgets.

phase before VSR creation, "VSR" to the phase after VSR creation, and "post-IVSG" to the phase after IVSG deployment. The hemodynamics of the VSR were measured as soon as possible after extracorporeal circulation was withdrawn, whereas those of post-IVSG were measured more than 30 minutes after weaning off extracorporeal circulation.

Imaging

Left ventriculography and echocardiography were performed before and after VSR creation and IVSG deployment. Angiography was performed by inserting a sheath into the LV apex and using a 5-F angiographic catheter before and after VSR creation and after IVSG deployment. Intraoperative echocardiography was performed via the apex or anterior RV wall directly using a gelatin gel pad.

Qp/Qs

Blood examinations of the peripheral artery, the superior and inferior venae cavae, and the pulmonary artery before and after VSR creation and after IVSG deployment were performed. The oxygen saturation (SO_2) of each was measured by blood gas analysis. Blood sampling was carried out when extracorporeal circulation was withdrawn. Oxygen saturation step-up between the right atrium and pulmonary artery was observed, and Qp/Qs was calculated. Qp/Qs was calculated by [arterial blood SO_2 (%) − mixed venous SO_2 (%)] ÷ [arterial blood SO_2 (%) − pulmonary artery SO_2 (%)]. Mixed venous SO_2 was calculated by [superior vena cava blood SO_2 (%) × 3 + inferior vena cava SO_2 (%)] ÷ 4.

Statistical Analysis

Statistical analysis was performed using IBM SPSS Statistics, Version 19 J (IBM Japan, Tokyo, Japan). The data are presented as means ± SD. Comparative analysis was performed with one-way repeated measures analysis of variance, followed by multiple comparisons using the Bonferroni adjustment method. Power was defined as 0.8 with a confidence interval of 95%. *P* values of less than 0.05 were considered significant.

RESULTS

All procedures were completed safely, and VSR creation and IVSG deployment were possible in all six cases. Ventricular tachycardia occurred immediately after IVSG deployment in

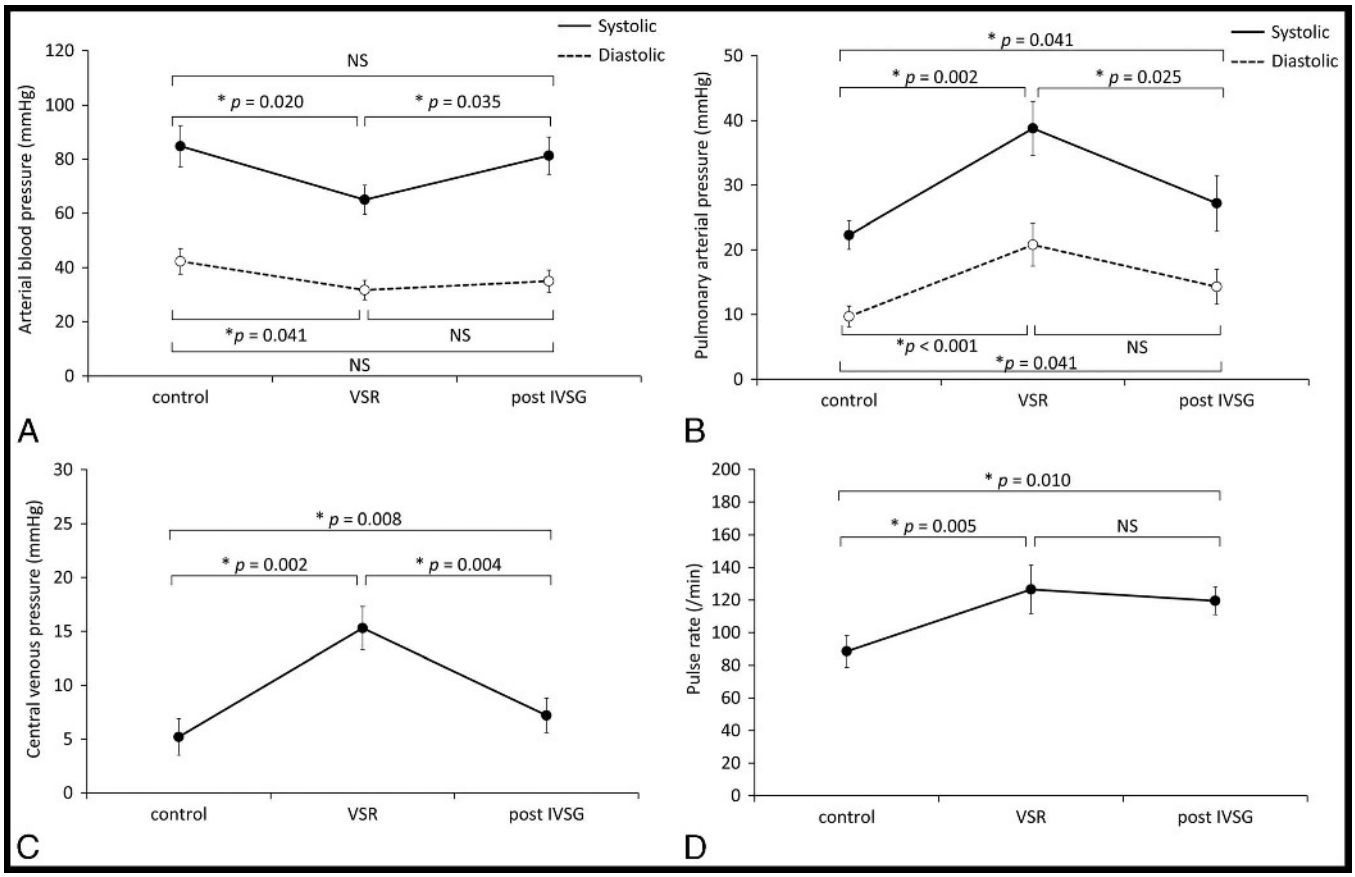

FIGURE 3. Hemodynamics. "Control" refers to the phase before VSR creation, "VSR" to the phase after VSR creation, and "post-IVSG" to the phase after IVSG deployment. A, The arterial systolic blood pressure decreases significantly after VSR creation and then increases significantly after IVSG deployment. B, Pulmonary arterial systolic blood pressure increases significantly after VSR creation and then decreases significantly after IVSG deployment. C, Central venous pressure increases significantly after VSR creation and then decreases significantly after IVSG deployment. D, The pulse rate increases significantly after VSR creation, but it is not significantly different after IVSG deployment.

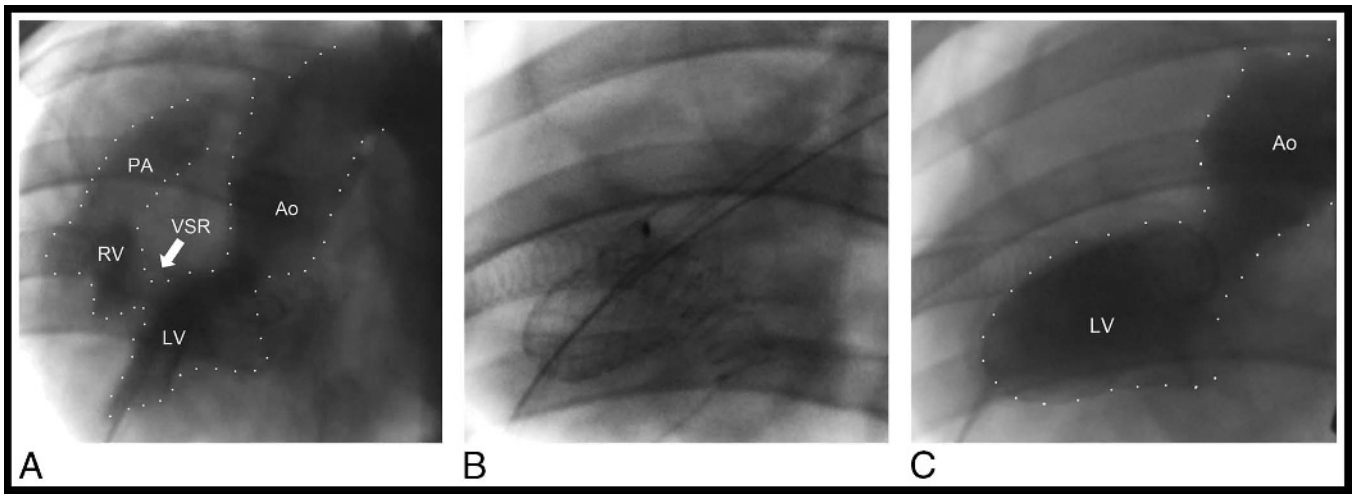

FIGURE 4. Left ventriculography. A, Left ventriculography shows the VSR L-R shunt flow (*arrows*). B, The IVSG is installed into the LV apex. C, Post-IVSG deployment, blood flow through the VSR has stopped.

one case, but sinus rhythm was restored with a 10-mg intravenous injection of xylocaine 2%. There were no intraoperative deaths.

Hemodynamics

Repeated measures analysis of variance showed a significant effect of the procedures (control, VSR, post-IVSG) on arterial systolic blood pressure ($P = 0.001$). Bonferroni-corrected multiple comparisons showed that arterial systolic blood pressure decreased to 65.0 ± 5.4 mm Hg, from 84.8 ± 7.6 mm Hg of the control, after VSR creation ($P = 0.002$), and that it went up to 81.3 ± 6.9 mm Hg after IVSG deployment ($P = 0.035$, Fig. 3A). Pulmonary arterial systolic blood pressure increased to 38.8 ± 4.2 mm Hg after VSR creation, from 22.3 ± 2.2 mm Hg of the control ($P = 0.002$). It decreased to 27.2 ± 4.3 mm Hg after the deployment of the IVSG ($P = 0.025$, Fig. 3B). Although central venous pressure increased from 5.2 ± 1.7 mm Hg to 15.3 ± 2.0 mm Hg ($P = 0.002$), it decreased to 7.2 ± 1.6 mm Hg later ($P = 0.004$, Fig. 3C). The pulse rate rose from 88.5 ± 9.8 per minute to 126.5 ± 14.9 per minute after VSR creation ($P = 0.005$), and it was 119.5 ± 8.5 per minute after the deployment of the IVSG; this difference was not significant ($P = 0.833$, Fig. 3D). Any pressure study parameter showing a worsening after VSR improved significantly after IVSG deployment. CPB could be withdrawn after VSR creation and IVSG deployment. Maintenance of hemodynamics was difficult after VSR creation but stabilized after IVSG deployment.

Imaging

Left ventriculography after VSR creation showed a defect hole in the interventricular septum and the flow of the contrast agent from the LV to the RV (Fig. 4A; Video 1, http://links.lww.com/INNOV/A96). The IVSG was placed in the LV apex (Fig. 4B). Left ventriculography after IVSG deployment showed that the L-R shunt flow had disappeared, and the flow of the contrast agent into the RV could not be seen (Fig. 4C). Left ventriculography and echocardiography showed that the LV apex was akinetic because of IVSG deployment, but this resulted in no obvious impairment of the movement of the basal, posterior, or lateral walls. The LV ejection fraction (LVEF) measured by echocardiography before and after VSR creation and after IVSG deployment was 67.7% ± 3.2%, 69.6% ± 5.5%, and 39.2% ± 9.8%, respectively. The LVEF after IVSG deployment was intentionally low compared with the other two groups ($P = 0.003$). After IVSG deployment, significant MR, defined as Sellers grade 2 or more, was not observed by LVG in any case; however, Sellers grade 1 MR was observed in two cases. Echocardiography showed mild MR in the same two cases with LVG.

Qp/Qs

Qp/Qs of the control was 1.02 ± 0.07, and Qp/Qs after VSR creation was 3.35 ± 1.00. Qp/Qs decreased significantly to 1.09 ± 0.10 after IVSG deployment (Fig. 5). Qp/Qs became significantly worse after VSR creation ($P = 0.007$), and it improved as expected after IVSG deployment ($P = 0.007$). The difference between Qp/Qs of the control and that of post-IVSG deployment was not significant ($P = 0.803$).

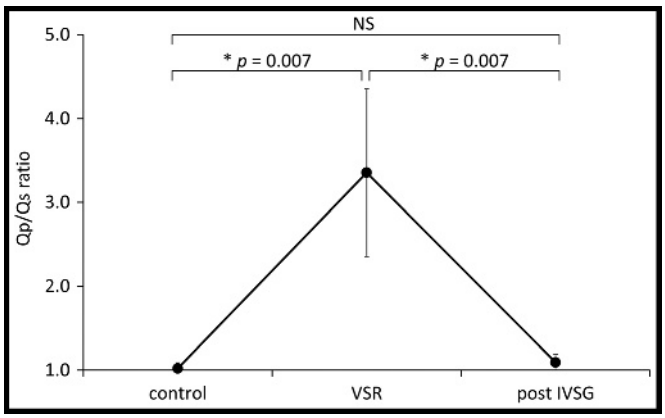

FIGURE 5. Qp/Qs. Qp/Qs deteriorates significantly because of VSR creation and improves as expected after IVSG deployment. The difference between control values and those after IVSG deployment is not significant.

DISCUSSION

The IVSG that was developed is a very effective device that can decrease the L-R shunt caused by an acute myocardial infarction–related VSR. Qp/Qs in the present VSR models was very high, at 3.35 ± 1.00, and the L-R shunt had a negative impact on the hemodynamics. The IVSG was specifically designed with a long septal side, so that the interventricular septum could be covered more broadly, and a shorter posterolateral side, so that interference with structures, such as the mitral valve and chordae tendineae, could be decreased. Although the frame of the MK stent has sufficient radial force to create flow control across the ventricular defect, the flexible structure did not interfere with LV function, as shown by pressure studies. We thought that this uniquely shaped stent graft device could certainly control the shunt flow from the VSR without MR and LV dysfunction. Qp/Qs after IVSG deployment improved significantly to 1.09 ± 0.10, and the slant cup-shaped stent graft placed in the LV dramatically decreased the amount of shunt by extensively covering the rupture in the interventricular septum. In addition, the hemodynamic instability that was seen after VSR creation improved dramatically after IVSG deployment.

Surgical repair for VSR has been widely performed since first reported by Cooley et al[7] in 1957, but the operative mortality rate remains high, at 20% to 87%, despite advances in surgical techniques.[1–5] There are also reports of necrotic myocardial tissue being extremely vulnerable in the hyperacute phase of myocardial infarction, surgical difficulty being very high, and early surgical treatment increasing the mortality rate.[2,3] Emergency surgery is often necessary with the deterioration in hemodynamics and general condition, despite the desire to perform surgery in the chronic phase.[8,9] In terms of hemodynamics, it is preferable to administer treatment for VSR as soon as possible after onset, although every effort should be made to perform surgery during the chronic phase when tissue scarring has progressed. Our IVSG does not require CPB and cardiac arrest. The procedure is far less invasive than traditional intracardiac repair, because placement inside the LV is possible without median sternotomy. Because vulnerable myocardial infarction foci are isolated from high LV pressure due to the IVSG, the risk of hemorrhage and free-wall rupture is greatly decreased. Damage to cardiac function can be minimized by not performing procedures such as direct myocardial resection. On the basis of these characteristics, early IVSG deployment may be possible even in the hyperacute phase of cardiac failure after the onset of myocardial infarction. Surgical repair performed during the stage in which scarring of the myocardium has progressed and stabilized is known to result in a good outcome.

The effectiveness of closure through placement of a percutaneous obturator has been demonstrated and clinically applied in recent years as a treatment for ventricular septal defect (VSD), which is a congenital heart disease. The Amplatzer Muscular VSD Occluder (AGA Medical, Plymouth, MA USA) is widely used.[10] There have been occasional reports on VSR closure associated with acute myocardial infarction using these devices.[11,12] This treatment is performed percutaneously, is minimally invasive, and has been proven to be effective. However, the following issues have been identified.[13–16] There are problems in determining the placement site because the defect holes in the VSR associated with acute myocardial infarction are often porous. The second issue is the lack of alternative applicable devices when the rupture is very large. The final issue is the possibility of dislocation of the device to the RV or the embolization of the device when fixation is insufficient, because the myocardium around the VSR is often vulnerable.

At our institution, we independently developed the MK stent graft before the widespread adoption of the thoracic endovascular aortic repair. There have been approximately 250 cases of its clinical use.[17,18] The clinical experience with the MK stent graft has allowed us to develop the IVSG. Our IVSG can cover a wide area, including the vulnerable ischemic myocardial region, with a graft. If the location of a large VSR is known, there is no need for detailed positioning.

The IVSGs of diameter of 40 mm or more were placed in an 18-F sheath to facilitate delivery. The IVSGs of diameter of 40 mm or more can be passed in a 20-F sheath. In the present animal experiments, the devices were placed using an approach from the LV apex where access is easier because of the fact that the femoral arteries of the animals used were anatomically narrow, and thoracotomy was always required to create the VSR models. If the deployment of the device is possible with 18- or 20-F sheaths, access from the femoral artery should be possible in patients with standard physiques. Possible future clinical applications include approaches from the femoral artery without sternal thoracotomy as the first choice, followed by approaches from the LV apex via left anterior small thoracotomy, depending on the situation. Thus, when considering 2-session LV repair and revascularization, median sternotomy over multiple sessions can be avoided, and the risk of chest reopening can be decreased.

The first of this study's limitations was the difference in intraventricular shape between the experimental animal model and humans. In particular, mitral valve tissue, such as the papillary muscles and chordae tendineae, were a factor causing severe MR associated with stent graft interference. We threedimensionally analyzed the LV cavities of pigs and selected a slant cup as the optimal stent shape that caused as little interference as possible with the mitral valve tissue, but we still need to analyze and investigate whether the same shape is effective in humans while performing contrast computed tomography of human LV cavities. The second limitation was that this experiment was not conducted in an acute myocardial infarction model. After IVSG deployment, the LVEF was intentionally low compared with control. This result may be due to apex wall motion was reduced by the stent of IVSG. However, we think that this result is not so important in this study. Because this experiment was not performed for acute myocardial infarction model and the preoperative cardiac function of these cases was normal, LV function after IVSG deployment become significantly worse as a matter of course. The pig model of anterior myocardial infarction has been known to show a tendency for developing fatal arrhythmias. It is, however, very difficult to create a VSR model after creating an acute myocardial infarction model. Thus, the effect of deployment of this device in the state of low LV function after myocardial infarction remains unknown. We are waiting for the results of our chronic phase experiment for effects on LV function of IVSG. The final limitation was the unknown durability of the IVSG. For long-term use of this device, it will be necessary to check its durability and perform chronic phase animal experiments. This new device may be

effective for the intermediate period until radical surgery can be planned. This device is suitable for installation during the early stages of VSR onset when physicians are hesitant to perform surgery. Left ventricular repair with median sternotomy would then be performed at a stage several months later when cardiac function has stabilized.

In conclusion, the L-R shunt and Qp/Qs were shown to be significantly decreased after the deployment of our newly developed IVSG in an animal model of VSR, which is normally a fatal complication of acute myocardial infarction. The IVSG was able to control the acute heart failure associated with VSR using a minimally invasive procedure in the hyperacute phase of cardiac failure. Potential improvements in VSR treatment outcomes from the clinical application of this device are awaited.

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CLINICAL PERSPECTIVE

This is an interesting experimental study of a novel intraventricular stent graft (IVSG) for the less invasive treatment of ventricular septal rupture (VSR). Nishida et al examined this novel technology on six pigs. The advantages of this device are that it can be placed without cardiopulmonary bypass, cardiac arrest, or a left ventricular incision. Ventricular septal rupture was created by making a hole in the inferior ventricular septum via the right ventricle. This device was placed via an 18F sheath in the left ventricular apex. The delivery system was advanced over a guidewire. The IVSG was placed across the VSR under fluoroscopic guidance. All procedures were performed safely, and the IVSG deployment was possible in all six cases. There was no flow across the defect after deployment, and there was no significant mitral regurgitation in any animal. The Qp/Qs value was decreased from 3.35 to 1.09 after deployment. These were excellent preliminary results.

However, it should be noted that there are significant limitations to the study. First of all, these experiments were carried out in normal animals with a small iatrogenic ventricular septal defect. In the clinical situation, there is often significant muscle necrosis, and the results with acute closure of VSR with percutaneous devices have been poor. Therefore, the relevance of this experimental model to the clinical situation is questionable. Moreover, there was a dramatic decrease in left ventricular ejection fraction after IVSG deployment, and this will need further experimental investigation. The final limitation of this study was that it did not address the long-term durability or efficacy of this stent graft.

With these limitations in mind, the authors are to be congratulated for developing a novel technology to help in the management of these very critically ill patients.

