EXPOSURE OF CIRCUMFLEX BRANCHES IN THE TILTED, BEATING PORCINE HEART: ECHOCARDIOGRAPHIC EVIDENCE OF RIGHT VENTRICULAR DEFORMATION AND THE EFFECT OF RIGHT OR LEFT HEART BYPASS

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Objective: In off-pump coronary surgery, exposure of posterior vessels via sternotomy causes deterioration of cardiac function. Changes in ventricular geometry, valve competence, and hemodynamics after retraction of the beating heart were studied. Subsequently, the modifying effect of right or left heart bypass was investigated. **Methods:** In six 80-kg pigs, an ultrasound probe was attached to the backside of the left ventricle and the heart was fully retracted with a suction tissue stabilizer. Five pigs underwent additional pump support. **Results:** During retraction, the right ventricle was squeezed between the pericardium and interventricular septum, thereby decreasing its diastolic cross-sectional area by $62\% \pm 6\%$ ($P < .001$) while, concomitantly, right ventricular end-diastolic pressure increased to $165\% \pm 19\%$ ($P = .004$) of basal values. Stroke volume and mean arterial pressure decreased by $29\% \pm 6\%$ and $23\% \pm 8\%$ ($P = .007$ and $P = .02$, respectively). Left ventricular shape became somewhat elliptic without changes in preload pressure, and its diastolic cross-sectional area decreased by $20\% \pm 3\%$ ($P = .001$). All valves were competent. Right heart bypass restored left ventricular cross-sectional area, stroke volume, and mean arterial pressure. In contrast, left heart bypass increased blood pressure only marginally. **Conclusions:** Ninety-degree anterior displacement of the beating porcine heart caused primarily right ventricular dysfunction as a result of mechanical interference with diastolic expansion without concurring valvular incompetence. Right heart bypass normalized stroke volume and mean arterial pressure by increasing left ventricular preload; in contrast, left heart bypass failed to restore systemic circulation. (J Thorac Cardiovasc Surg 1999;118:316-23)

Off-pump coronary bypass operations through a sternotomy^{1,2} have recently gained renewed interest for multivessel revascularization.³⁻⁶ Exposing posterior branches by displacing the beating heart (apex points anteriorly), however, tends to decrease arterial pressure considerably, both in the pig model^{7,8} and in patients.⁵ Currently, circulatory deterioration during exposure of

posterior vessels seems to prevent rapid expansion of coronary operations on the beating heart.⁹ Inotropic support,⁵ volume redistribution, or increasing preloads^{5,7,8} may, in part, mitigate hemodynamic deterioration during posterior wall revascularization. Ninety-degree displacement causes biventricular heart failure characterized by a major drop in stroke volume, despite elevation of right ventricular (RV) preload and unchanged left ventricular (LV) preload.^{7,8} Previously, it was found that the decreased pump function is not caused by impaired blood supply to the myocardium in a retracted beating heart.⁸ Visually, the right ventricle looks crumpled and folded on retraction.^{7,8} Pump failure may be worsened by the presence of concomitant valvular incompetence. It is conceivable that cardiac displacement causes strangulation of ventricles, but it is unclear which ventricle is more affected in its (diastolic) pump function by the maneuver. The precise mechanisms that cause deteriora-

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Fig 1. Exposure of posterior vessels by vertical displacement of the beating porcine heart with the Octopus tissue stabilizers,^{5,10} which were mounted to the sternal retractor. Note the omniplane ultrasound probe that was fixed between the suction tentacles (arrow).

tion of the circulation on lifting the beating heart are still not fully understood.

The objectives of *study I* were (1) to monitor changes in ventricular geometry, hemodynamics, and valvular function during vertical displacement of the beating porcine heart by the Octopus tissue stabilizer (Medtronic, Inc, Minneapolis, Minn)^{5,7,8,10} and (2) to assess the modifying effect of the Trendelenburg maneuver (whole-body head-down positioning) on the changes in ventricular geometry, hemodynamics, and valve function.

In *study II* we investigated the effect on hemodynamics of mechanically supporting either the right or the left ventricle during vertical displacement of the beating porcine heart.

Methods

Six Dutch landrace pigs (range 75-85 kg) were used. All animals received humane care in compliance with the "Guide for the Care and Use of Laboratory Animals" published by the National Institutes of Health (NIH publication No. 85-23, revised 1985). The study protocol was approved by the Animal Experimentation Committee of the Utrecht University.

Anesthesia and instrumentation. The animals were premedicated, anesthetized, and instrumented for hemodynamic measurements as described before.⁸ To reduce the mechanical irritability of the heart, we gradually administered intravenous propranolol (range 15-25 mg) to obtain a heart rate between 50 and 70 beats/min. After median sternotomy, the heart was suspended in a pericardial cradle. A bipolar pacing lead was sutured on the right atrial appendix, whereafter the heart was paced at a fixed rate of 80 beats/min. An ultrasound transit time flow probe (Transonic Inc, Ithaca, NY, size 20 or

Fig 2. Echographic cross-sectional areas (left panels) and M-mode images (right panels) obtained during 90-degree anterior displacement (*DISPLACED*) and subsequent head-down positioning (+ *TRENDELENBURG*). VS, Ventricular septum; RV, right ventricle; LV, left ventricle. The ultrasound probe was positioned between 2 Octopus tentacles and aligned to the posterior wall (*ANATOMIC*). Note the severe deformation of the RV on cardiac retraction (arrow, *DISPLACED*) and the partial recovery of RV cross-sectional area after the Trendelenburg maneuver.

24 mm) was placed around the aorta for on-line measurement of the cardiac output. Stroke volume was calculated by dividing cardiac output by heart rate (fixed rate 80/min). For stepwise cardiac retraction,⁸ two Octopus tissue stabilizers^{5,10} were used, with an omniplane ultrasound probe (6.2. MHz, Sonos 2000, Hewlett-Packard, Amstelveen, The Netherlands) fixed in between (Fig 1). The ultrasound probe was remotely steered to the heart surface until it touched the immobilized

Fig 3. For legend see page 319.

epicardium for the on-line collection of 2-dimensional and M-mode echocardiographic images (Fig 2). During maneuvers, the ultrasound probe remained exactly positioned in between and in the same plane as the Octopus tentacles. Four 3F air-filled balloon-tipped catheters, which served as echographic beacons, were placed in the lumen of the right ventricle and marked precisely the RV long-axis plane (first pair: RV outflow tract and RV apical portion) or the RV short-axis plane (second pair: border of tricuspid valve and midportion of interventricular groove). Care was taken that the plane of the short-axis view of the left ventricle crossed the apices of both LV papillary muscles.

A personal computer-based data acquisition system stored hemodynamic variables, which were simultaneously monitored on an 8-channel recorder (Gould Instrument System Inc, Valley View, Ohio).

After completion of the experimental protocol of study I (see further), in 5 animals (study II) the right atrium and pulmonary artery trunk and the left atrium and left carotid artery were cannulated for right and left heart bypass (RHB and LHB, respectively), as described before.¹¹ A single centrifugal pump (BP 80, Medtronic Bio-Medicus, Eden Prairie, Minn) was connected to the withdrawal and return cannulas by 2 Y-connectors. The pump circuit was used as either RHB or LHB by instantaneously changing tubing clamps. A test run was performed at maximum flow to bypass the right ventricle and the left ventricle (bypass flow > 4 L/min) in the anatomic position. It was assured that the tip of the 38F LHB withdrawal cannula was suspended in the left atrial lumen by applying slight tension to the purse-string suture while the heart was retracted anteriorly. By palpation, the tip position was again verified.

Experimental protocol

Study I ($n = 6$). Echocardiographic recordings were taken at each phase during at least 5 cardiac cycles and videotaped for analysis. In the same 2-dimensional view (probe at zero degrees), end-diastolic chamber dimensions (square millimeters) were obtained from the short-axis LV cross-sectional area and the long-axis RV cross-sectional area. In the M-mode view (position zero degrees), the distance between the LV posterior wall and RV free wall was measured in millimeters. After 90 degrees' rotation of the ultrasound omni-plane probe (probe in position 90 degrees), the RV short-axis cross-sectional area was measured. The LV long-axis view could not be consistently obtained in the same 2-dimensional plane and was further not measured. In addition, Doppler echocardiographic images were made of the mitral and aortic valves after each maneuver. The function of the pulmonary and tricuspid valves was judged in 2 separate experiments

with a hand-held ultrasound Doppler probe surfing over the epicardium using different echographic cross sections. Baseline cardiovascular values and echocardiographic measurements were recorded after fixation of the Octopus tentacles to the posterior wall after at least 15 minutes of pacing (phase 1, anatomic position). Subsequently, values were taken 3 minutes (phases 2-5) and 15 minutes (phases 3 and 5) after stabilization after each intervention.

The heart was vertically displaced by 90 degrees without delay with the Octopus tissue stabilizer (phase 2, first displacement) until the apex pointed upward. Circumflex branches on the back side of the heart became well exposed to the operative field^{5,7,8} without the aid of additional supportive tools. Subsequently, after stabilization of hemodynamics, the operating table was tilted 20 degrees in the head-down position (Trendelenburg maneuver) without changing the position of the heart relative to the body (phase 3). After the Trendelenburg maneuver, the operating table was returned to the horizontal position while the heart remained retracted for 3 minutes (phase 4, second displacement). Subsequently, the heart was put back in the pericardial cradle (phase 5, anatomic position). The entire experimental protocol was repeated after a period of stabilization.

The first 3 consecutive phases in the experimental protocol are schematically depicted on the abscissa of Fig 3.

Study II ($n = 5$). From baseline (anatomic position) the heart was retracted for 3 minutes (displacement). Subsequently, RHB was initiated whereby maximum RHB flow was set to optimally unload the right ventricle. After 3 minutes' stabilization (displacement + RHB), the pump was gradually stopped and the switch was made to LHB. LHB flow was increased until the left atrium was maximally drained and the atrial tissue collapsed (displacement + LHB).

Statistical analysis. First, data sets of 2 subsequently executed protocols from each animal were averaged and mean values were further used for analysis. Single video still frames at end-diastole and mid-systole were grabbed from original tapes and were off-line analyzed by an independent observer (C.M.M.). Data in Tables I and II are presented as mean $\pm$ standard deviation (SD) (absolute values). Hemodynamic variables and echocardiographic variables in the *Results* section and in Fig 3 are depicted as mean $\pm$ standard error of the mean (SEM) (percentage of basal values). Statistical analysis was performed by multivariate analysis of variance to assess the influence of displacement, and a paired Student *t* test was used to assess the modifying effect of the Trendelenburg maneuver or RHB/LHB compared with protocol control values.

Fig 3. Relative changes in hemodynamic parameters and RV and LV cross-sectional areas during vertical displacement of the beating porcine heart with the Octopus tissue stabilizer (study I) and the effect of mechanical circulatory support on displacement (study II). Statistical comparison with control values: * $P < .05$, ** $P < .01$, # $P < .001$. *n.m.*, Not measured; erroneous readings from the ultrasound aortic flow probe caused by turbulent flow from LHB cannula. *DIS*, Displaced; *LV*, left ventricle; *RV*, right ventricle; *RHB*, right heart bypass; *LHB*, left heart bypass.

Table I. Study I

	Basal values (phase 1)	Vertical displacement (phase 2)	P values	Trendelenburg 20-degree head-down (phase 3)	P values	Vertical displacement (phase 4)	Freely beating (phase 5)
SV (mL)	54 ± 9	37 ± 7	.007	50 ± 7	.5	37 ± 7	45 ± 3
HR (paced, beats/min)	80	80	—	80	—	80	80
CO (L/min)	4.3 ± 0.8	3.0 ± 0.5	.007	4.0 ± 0.6	.5	3.0 ± 0.6	3.6 ± 0.2
MAP (mm Hg)	66 ± 14	50 ± 12	.02	73 ± 16	.4	47 ± 9	55 ± 5
RVEDP (mm Hg)	6 ± 2	9 ± 2	.004	11 ± 2	.001	8 ± 1	6 ± 1
LVEDP (mm Hg)	10 ± 2	10 ± 5	.8	12 ± 3	.01	9 ± 3	8 ± 2
RAP (mm Hg)	7 ± 2	8 ± 2	.2	9 ± 2	.003	8 ± 2	7 ± 2
LAP (mm Hg)	11 ± 4	9 ± 3	.03	10 ± 2	.3	9 ± 3	11 ± 2
RVla, diastolic (mm ²)	1357 ± 262	494 ± 143	.001	895 ± 179	.01	494 ± 28	1219 ± 109
RVsa, diastolic (mm ²)	725 ± 95	287 ± 79	.001	412 ± 55	.01	254 ± 5	616 ± 108
LVsa, diastolic (mm ²)	1317 ± 90	1050 ± 127	.001	1271 ± 177	.5	952 ± 92	1094 ± 90
Distance, LV posterior wall– RV free wall (mm)	90 ± 5	75 ± 5	.001	82 ± 5	.008	75 ± 5	87 ± 2

Values are shown as means ± standard deviation ($n = 6$). Comparison was made with basal values where appropriate. SV, Stroke volume; HR, heart rate; CO, cardiac output; MAP, mean arterial pressure; RVEDP, right ventricular end-diastolic pressure; LVEDP, left ventricular end-diastolic pressure; RVla, right ventricular long-axis cross-sectional area; RVsa, right ventricular short-axis cross-sectional area; LVsa, left ventricular short-axis cross-sectional area.

Results

All animals survived the entire procedure without our needing to defibrillate or administer inotropic drugs. The paced heart remained in a regular rate of 80 beats/min throughout the experiment. M-mode measurement revealed that the myocardium in between the Octopus tissue stabilizers was well immobilized, and the full-thickness cardiac wall showed almost no wall motion (Fig 2, *right panel*). The experimental data of studies I and II are summarized in Tables I and II, respectively, and in Fig 3. Data in the text are given as a percentage of baseline values.

Study I. Echocardiography during displacement of the beating heart (Table I, Fig 2, and Fig 3, *left panel*). Displacement of the beating heart (phase 2) caused the same degree of deterioration of the circulation as observed before.^{7,8} RV end-diastolic pressure increased to 165% ± 19% of baseline values. In contrast, no significant change in LV end-diastolic pressure was observed (101% ± 9%). Echographically, a significant part of the RV free wall was pressed to the interventricular septum (Fig 2, *left panel, displaced*), whereas the RV outflow tract was somewhat narrowed but remained patent. Diastolic dimensions of the long-axis and short-axis RV cross-sectional areas decreased to 38% ± 6% and 40% ± 5%, respectively. Diastolic dimensions of the short-axis LV cross-sectional area decreased to 79% ± 3%, whereas the LV muscle was shaped elliptically. In M-mode, the distance between the LV posterior wall and RV free wall decreased by 17% ± 2%. Early diastolic LV septal motion was directed toward the RV free wall,

thereby further decreasing RV dimension. In no case was incompetence of any valve observed during the entire cardiac cycle. Specifically, the posterior leaflet of the mitral valve did not show prolapse. The 2-dimensional geometry of the cardiac valves seemed grossly unaffected. The superior and inferior caval veins remained fully patent on displacement.

Tilting whole-body head-down 20-degree Trendelenburg position. Three minutes into the Trendelenburg phase (phase 3), stroke volume had increased to 96% ± 8% and mean arterial pressure had normalized at the expense of increased ventricular preloads. Echocardiographically, a part of the RV free wall was released from the interventricular septum, and the RV outflow tract (in the short-axis view) increased its dimension. Diastolic dimensions of the long-axis and short-axis RV cross-sectional areas increased to 68% ± 8% and 57% ± 1%, respectively. Diastolic short-axis LV cross-sectional dimension increased to 96% ± 5%, whereas the left ventricle remained shaped elliptically. The distance between the LV posterior wall and RV free wall increased to 92% ± 2%. Again, in no case was dysfunction of any valve observed during the entire cardiac cycle after the preloads were increased.

Return of the operating table to the horizontal position. After the operating table had been returned to the horizontal position (phase 4, the heart remained retracted 90 degrees), most values returned to pre-Trendelenburg levels.

Return of the beating heart to the anatomic position. Shortly after return to the anatomic position (phase 5),

Table II. Study II

	Basal values	Vertical displacement	P values	RHB during displacement	P values	LHB during displacement	P values
SV (mL)	42 ± 7	17 ± 6	.02	33 ± 5	.09	—	—
HR (beats/min)	85 ± 10	81 ± 9	.07	93 ± 10	.1	94 ± 10	.06
CO (L/min)	3.4 ± 0.4	1.5 ± 0.4	.007	3.0 ± 0.5	.6	n.m.	—
MAP (mm Hg)	64 ± 9	32 ± 6	.006	64 ± 13	.9	46 ± 13	.04
RVEDP (mm Hg)	6 ± 2	9 ± 1	.02	7 ± 1	.07	9 ± 2	.06
LVEDP (mm Hg)	6 ± 1	7 ± 1	.07	11 ± 3	.04	6 ± 4	.8
RAP (mm Hg)	8 ± 2	10 ± 1	.007	7 ± 3	.5	10 ± 3	.07
LAP (mm Hg)	7 ± 1	8 ± 2	.4	12 ± 5	.1	5 ± 5	.4
RVla, diastolic (mm ²)	1249 ± 31	458 ± 159	.01	320 ± 98	.003	609 ± 122	.2
LVsa, diastolic (mm ²)	1155 ± 12	679 ± 214	.02	1376 ± 298	.1	506 ± 104	.002
Maximum pump flow (L/min)				3.1 ± 0.5	.009	1.6 ± 0.5	.001
Ratio, pump flow/baseline CO				0.91		0.55	

Values are shown as means ± standard deviation (*n* = 5). Comparison was made with basal values where appropriate. SV, Stroke volume; HR, heart rate; CO, cardiac output; MAP, mean arterial pressure; RVEDP, right ventricular end-diastolic pressure; LVEDP, left ventricular end-diastolic pressure; RAP, right atrial pressure; LAP, left atrial pressure; RVla, right ventricular long-axis cross-sectional area; LVsa, left ventricular short-axis cross-sectional area; n.m., Not measured.

the right ventricle was transiently dilated. Echocardiographically, RV diastolic dimensions were variably enlarged. Long-axis RV cross-sectional area increased from $1357 \pm 262 \text{ mm}^2$ to $1756 \pm 273 \text{ mm}^2$ (to $130\% \pm 6\%$, $P = .001$, vs baseline values). Central hemodynamic status improved rapidly and RV wall motion increased concomitantly. Diastolic RV dimension decreased to baseline values.

Study II. RHB or LHB in the retracted heart (Table II, Fig 3, right panel). The condition of the animal had somewhat deteriorated because of the previously executed protocol of study I, hemodilution, and subsequent cannulation. At 3 minutes' displacement, RHB was started. Average RHB flow reached 100% of baseline cardiac output, which increased diastolic short-axis LV cross-sectional dimension, LV preload, and cardiac output to baseline values. In contrast, LHB increased mean arterial pressure to only $72\% \pm 11\%$ ($P = .035$). Frequently, the LHB withdrawal cannula was pressed against the left atrial wall, which forced reduction of LHB flow. In 2 cases, air aspiration to the centrifugal pump occurred. Maximum LHB flow reached only 55% of baseline cardiac output.

Discussion

The principal findings of the study were as follows: First, in the supine animal, vertical displacement of the beating heart caused a major distortion in RV geometry but only a moderate change in LV shape. The decrease in stroke volume and in mean arterial pressure and the increase in RV preload coincided with diminished LV and RV dimensions.

Second, the borderline circulatory status during dis-

placement was markedly improved to almost baseline status by blood volume redistribution (Trendelenburg maneuver). At the same time, LV cross-sectional area, but not LV shape, returned to baseline.

Third, cardiac retraction did not cause valvular dysfunction.

Fourth, RHB effectively restored central hemodynamics in the tilted heart, whereas LHB only marginally improved circulatory status.

Epicardial 2-dimensional echocardiography of the beating heart. The immobilized epicardial surface contained a distal circumflex branch, and its presentation by the Octopus tissue stabilizers simulated full exposure for coronary surgery without the use of additional supportive tools (Fig 1).

In the 80-kg pig, 2-dimensional echocardiography of the beating heart proved technically feasible by fixing the ultrasound probe between two Octopus suction pods, which took hold of the posterior side of the heart (Fig 1). The area immobilized by the retractor/ultrasound system comprized approximately a small match-box area of cardiac tissue that does not affect cardiac function.⁷ Interestingly, cardiac wall motion and systolic wall thickening between the tentacles was almost absent transmurally in the line of measurement of the echo probe. The latter phenomenon was observed immediately after attachment of the suction device. The function of the mitral valve and the aortic valve could be adequately assessed in all experiments by Doppler ultrasound in the LV long-axis/RV short-axis view.

Displacement of the beating heart. The deterioration in circulation during displacement was characterized by severe reduction in stroke volume despite an

elevated RV preload without a concomitant change of LV preload and with unimpaired valve function. Thus cardiac retraction causes biventricular pump failure. We hypothesized that the function of the thin-walled right ventricle was primarily affected. When the beating heart was retracted anteriorly, RV luminal dimension in two planes decreased by about 60%, and RV geometry was considerably distorted, evidenced by crumpling of the muscle. For a substantial portion, the thin-walled RV muscle was squeezed between the left ventricle and the surrounding tissue, that is, the pericardial sac and right sternal border. A significant part of the RV free wall endocardium was pressed against the interventricular septum during the entire cardiac cycle. In diastole, the right ventricle did not expand sufficiently. A rim of RV tissue protruded over the interventricular groove, but that part of the ventricle in fact contributed well to RV output. The augmented RV preload corresponded to the absence of torque or compression of the caval veins and/or tricuspid valve dysfunction and/or pulmonary valve dysfunction. Apparently, the RV lumen available for diastolic expansion was reduced, which amounts to *an acquired temporary RV hypoplasia*.

In contrast to the severe changes in RV geometry, the changes in LV geometry were only moderate. The shape of the left ventricle changed from circular to elliptic, probably because of a thicker, less deformable muscle. LV function, however, also seemed impaired because stroke volume decreased despite unchanged LV preload and left atrial pressure. Load-independent indices of cardiac contractility were not assessed. Propranolol exerts negative inotropic effects on contractility and may worsen heart failure by displacement. Therefore a complete explanation of the changes of cardiac performance cannot be given.

RV support. In study II, we tested the hypothesis that RV failure is predominant over left heart failure in a retracted heart. In all cases, selectively increasing the preload of the tilted left ventricle by deviating blood around the displaced right ventricle by means of a short-circuit RHB system fully restored LV stroke volume and, as a result, mean arterial pressure. RHB flow in this study reached baseline cardiac output. In contrast to healthy pigs, patients with poor LV function and dilated hearts may not tolerate augmentation of LV preload in deformed tilted hearts (mean LV end-diastolic pressure increased from 6 to 11 mm Hg). These patients may need a form of LV mechanical support as well.

LV support. In contrast to RV support, LV support resulted in only marginal improvement of mean arterial pressure. Maximum LHB flow was comparable with

cardiac output in the displaced heart without any form of circulatory support. Attempts to fully restore systemic circulation failed in all cases because blood supply originating from the right ventricle was apparently insufficient. The left atrial wall easily collapsed and yielded a risk of air aspiration (in 2 of 5 cases). We rule out technical problems related to cannulation because the correct position of the tip of the withdrawal cannula was verified by palpation. From these observations we infer that the severe drop in stroke volume during anterior displacement of the beating porcine heart is primarily caused by right heart pump failure.

Effect of 20-degree Trendelenburg positioning. As reported earlier,^{7,8} the borderline circulatory status during displacement markedly improved almost to baseline status by blood volume redistribution (Trendelenburg maneuver). When the subject was tilted head down, the compressed right ventricle was (in part) mechanically pushed open by further augmenting filling pressure, demonstrated by an improved but incomplete recovery of RV dimensions, while the position of the heart relative to the body remained unchanged. At the same time, LV filling pressure increased, LV dimensions returned to baseline, and stroke volume normalized.

In selected patients, the Trendelenburg maneuver effectively mitigates circulatory deterioration on exposure of the posterior wall.⁵ In two thirds of the cases, however, inotropic support remained necessary.³

Recent observations by us showed that right oblique positioning of the pig after sternotomy facilitated exposure of the posterior LV wall and also mitigated the sequelae of cardiac retraction on mean arterial pressure.

The beating heart in the anatomic position. As before,^{7,8} we observed transient firm distention of the right ventricle on quickly replacing the heart into its anatomic position. The dilatation disappeared within several minutes and the central hemodynamic status improved. Apparently, the right ventricle needs to adjust after more than 30 minutes of deformation and increased preload.

Limitations. The pig's chest wall geometry differs from that of the human chest wall; that is, it is less barrel-shaped. Cardiac retraction was carried out in the normal healthy pig heart with its apex slightly pointing rightward. Patients with impaired LV function may not tolerate vertical displacement of the beating heart. Therefore inferences made from this experimental study may be applicable to human beings only after careful consideration.

Conclusion

Ninety degrees' anterior displacement of the beating porcine heart caused primarily RV deformation and

pump function without signs of valvular incompetence or inflow or outflow obstruction. The displacement prevented normal RV and LV diastolic expansion by pressing the heart against the surrounding tissue. It is inferred that vertical displacement affects mostly RV function because RHB effectively restored LV function whereas LHB failed to restore cardiac output and mean arterial pressure.

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