**Transesophageal Echocardiographic Hemodynamic Monitoring during Preoperative Acute Normovolemic Hemodilution**

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**Background:** Preoperative acute normovolemic hemodilution may compromise oxygen transport. The aims of our study were to describe the hemodynamic effects of normovolemic hemodilution and to determine its effect on systolic and diastolic cardiac function by transesophageal echocardiography.

**Methods:** In eight anesthetized patients (aged 13–51 yr) without heart disease, hemoglobin was reduced in steps from 123 ± 8 (mean ± SD) to 98 ± 3 and to 79 ± 5 g/l. Hemodynamic measurements (intravascular pressures, thermodilution cardiac output, and echocardiographic recordings) were obtained during a stabilization period and at each level of hemodilution. Left ventricular wall motion was monitored continuously, and Doppler variables, annular motion, and changes in ejection fractional area were analyzed off-line.

**Results:** During hemodilution, cardiac output by thermodilution increased by 16 ± 7% and 26 ± 10%, corresponding well to the increase in cardiac output as measured by Doppler (difference, 0.32 ± 1.2 1/min). Systemic vascular resistance fell 16 ± 14% and 23 ± 9% and pulmonary capillary wedge pressure increased slightly (2 ± 2 mmHg), whereas other pressures, heart rate, wall motion, and diastolic Doppler variables remained unchanged. Ejection fractional area change increased from 44 ± 7% to 54 ± 10% and 60 ± 9% as a result of reduced end-systolic and increased end-diastolic left ventricular areas.

**Conclusions:** A reduction in hemoglobin to 80 g/l during acute normovolemic hemodilution does not normally compromise systolic or diastolic myocardial function as determined by transesophageal echocardiography. Preload, left ventricular ejection fraction, and cardiac output increase with a concomitant fall in systemic vascular resistance. (Key words: Noninvasive monitoring; stroke volume; blood viscosity; general anesthesia.)

PREOPERATIVE acute normovolemic hemodilution (ANH) is used to reduce the loss of blood cells during intraoperative bleeding and to avoid or reduce the need for homologous blood transfusion. The efficacy of hemodilution for this purpose is somewhat controversial.1,2 The hemodynamic effects of ANH using non-oxygen-carrying exchange solutions have been studied mainly in animal models3,4 and have been shown to cause a reduction in blood viscosity, decreased systemic vascular resistance, and increased venous return.5,6 These changes result in increased stroke volume (SV) and cardiac output (CO).7 There is little central hemodynamic data available in humans,8–12 and even such an essential, previously generally accepted compensatory mechanism as increased CO has recently been contradicted.13

Echocardiography offers noninvasive or less invasive alternatives for monitoring the hemodynamic variables previously derived only invasively. The accuracy of transesophageal echocardiography (TEE) measurements has been evaluated against invasive measurements of CO14–16 and pressure measurements and compares favorably.17–19 During hypervolemic hemodilution, TEE has been found to be accurate in measuring CO and volume loading,20 and TEE has been used to follow the left ventricular (LV) systolic regional function in patients with vascular disease during normovolemic hemodilution.21

The use of TEE during ANH for hemodynamic monitoring, including the assessment of LV regional systolic and diastolic function and loading conditions, has not to our knowledge been evaluated in humans. In the current
study we used multiplane TEE and invasive measurements to evaluate the hemodynamic effects of ANH.

**Material and Methods**

**Patients**

We studied eight patients (seven women, mean age, 28 yr; range, 13–61 yr; mean weight, 55 kg; range, 44–71 kg) who had ANH before operations for scoliosis, which was expected to cause substantial blood loss. We excluded anyone with evidence of heart disease in the history, on physical examination, on electrocardiography, or on transthoracic echocardiography. The patients had no history of problems with swallowing or hemo-stasis and no esophageal disease. All patients were informed and gave consent to participate in the study, which was approved by the Medical Ethics Committee at our institution.

**Anesthesia, Preparation and Acute Normovolemic Hemodilution**

The patients were premedicated with diazepam 5 mg orally, and anesthesia was induced with fentanyl 2.5 μg/kg, thiopental 5 mg/kg, and vecuronium 0.1 mg/kg. After tracheal intubation, the lungs were ventilated with 70% nitrous oxide in oxygen, and minute volume was adjusted to achieve normocapnia. While the patient remained in the supine position, anesthesia was maintained with isoflurane (end-tidal concentration, 0.8%). No additional fentanyl was given during the study. Warmed (37°C) Ringer's acetate replacement and a heating blanket were used to maintain body temperature at basal value. In each patient a central venous catheter, a pulmonary arterial thermodilution (TD) catheter (Swan-Ganz, Arrow 7.5 French), a right radial artery catheter, and additional peripheral venous catheters were introduced. A transesophageal multiplane ultrasound probe (3.5 MHz imaging, 2.5 MHz spectral Doppler, CFM800, Vingmed Sound, Horten, Norway) was introduced. Heart rate (HR), a five-lead electrocardiogram (X, Y, Z, V2, V5), radial mean systemic arterial pressure, mean pulmonary artery pressure, and central venous pressure were monitored continuously (Hewlett Packard M1092; Palo Alto, CA). We also measured pulmonary capillary wedge pressure (PCWP) at end expiration and CO by TD (Hewlett Packard M1012) simultaneously with the echocardiographic Doppler recordings at five stages, as described in Data Acquisition. We used the mean of three or more consecutive CO_TD measurements (10 ml isotonic saline at room temperature).

After induction of anesthesia and a 15- (n = 2) or 45- (n = 6) min stabilization period, ANH was undertaken. To achieve hemoglobin values of 100 and 80 g/l (in two steps), the blood volume to be withdrawn was preoperatively calculated from weight and hemoglobin level, using a nomogram based on the formula: volume lost = blood volume · ln(hemoglobin_start/hemoglobin_final), where ln denotes the natural logarithm. Blood withdrawal and volume substitutions were accomplished simultaneously within 15 min. The total infused volume was twice that withdrawn and consisted of equal quantities of Ringer's acetate and 4% albumin solution. The patient was allowed to equilibrate 5 min before hemoglobin was measured and hemodynamic recordings were performed. The planned operation did not start until ANH and all measurements were completed.

**Data Acquisition**

We monitored electrocardiography, HR, two-dimensional (2D) TEE, and mean systemic arterial, mean pulmonary arterial, and central venous pressures continuously. Additional parallel noninvasive and invasive measurements were made at five time points: first at 15 (n = 8), 30 (n = 6), and 45 (n = 6) minutes during the stabilization period, and second during the two stages of ANH (n = 8). Each stage of hemodilution was completed within 15 min, and measurements were made 5 min later after a stabilization period. Data on the continuously measured pressures, PCWP, and CO_TD were stored digitally in the memory of the Hewlett Packard system.

An experienced echocardiographer monitored the heart continuously by TEE and made recordings on a super-VHS video recorder and as paper printouts. From an esophageal longitudinal four-chamber view, we recorded 2D images of mitral, tricuspid, and aortic annuli as well as pulsed-wave spectral Doppler images of mitral inflow (sample volume located at the tip of the open valve) and pulmonary venous flow (sample volume 1 cm upstream in the upper left pulmonary vein). By advancing the probe, we obtained transgastric long-axis and short-axis 2D views of the LV and the LV outflow tract. We recorded flow velocities from the LV outflow tract by continuous-wave spectral Doppler.

**On-line Transesophageal Echocardiography Analysis**

Real-time, 2D, transgastric, short-axis images of the left ventricle at the level of the papillary muscles were mon-
monitored continuously when no specific measurements were made. We used this view in combination with a transgastric long-axis view and esophageal 2D views to analyze wall motion in all 16 segments of the LV.23 Envelope-traced Doppler recordings from the LV outflow tract and aorta (velocity time integral) served as repeated checks on changes in SV.

Off-line Transesophageal Echocardiography Analysis

Two-dimensional and Doppler images were also analyzed off-line from video recordings. The on-line wall motion analysis was repeated off-line by one independent observer. End-diastolic (LVEDA) and end-systolic (LVESA) left ventricular areas, defined as the largest and smallest area of the LV cavity at the level of the papillary muscles, were measured off-line. High-quality images from three cardiac cycles were averaged. Fractional left ventricular area changes (FAC; LVEDA – LVESA)/LVEDA were calculated in percentages. The longitudinal ventricular shortening (in millimeters) was measured from esophageal 2D images24 as maximum (end-systolic – end-diastolic) lateral mitral annulus motion and lateral tricuspid annulus motion.

To calculate CO_{Doppler}, the velocity time integral was multiplied by HR and flow area. Flow area was assumed to be circular and constant and was calculated from the systolic inner-to-inner diameter of the aortic annulus.14 In one patient, the quality of the Doppler signal was inadequate for the calculation of CO.

For estimation of LV diastolic properties, the following spectral Doppler data were collected and analyzed: mitral early filling maximal flow velocity (MEV\textsubscript{max}) and deceleration time; mitral atrial filling maximal flow velocity (MAV\textsubscript{max}) and duration (MA\textsubscript{dur}); isovolumic relaxation time (aortic closure to mitral opening); maximal systolic and diastolic forward pulmonary venous flow; and the maximal velocity and duration of atrial pulmonary flow reversal. All measurements were repeated once by an independent experienced observer.

Statistical Analysis

Statview computer software (Abacus Concepts, Inc., Berkeley, CA) was used for statistical analysis. We used analysis of variance for repeated measures to identify any changes during the stabilization period or during ANH. Significance was defined as \( P < 0.05 \). For comparisons, regression analyses and correlation coefficients were calculated, and to compare invasive and noninvasive measurements of CO, the mean difference (bias) and its SD were calculated.25 For echocardiographic reproducibility, interindividual coefficients of variation of repeated measurements were calculated by dividing the SD of the differences by the mean value of the samples.

Results

Preoperative ANH was completed in all patients according to the protocol without complications, and all patients recovered uneventfully from the operations for scoliosis.

During Stabilization Period

Fifteen, 30, and 45 min after induction of anesthesia, no hemodynamic changes were recorded.

During Acute Normovolemic Hemodilution Monitoring and Invasive Measurements. HR, mean systemic arterial pressure, and mean pulmonary artery pressure remained constant during ANH (table 1). No electrocardiographic changes were recorded. CO increased in steps from 16 to 26%, and the change in CO correlated with the change in hemoglobin concentration (\( r = 0.85 \)). HR remained constant, and the increase in CO was a result of an increased SV. Calculated systemic vascular resistance ([mean systemic arterial pressure – central venous pressure]/CO) accordingly fell, but central venous pressure remained at the same level. A slight increase in PCWP was seen by the end of ANH.

Echocardiographic Measurements. No regional abnormalities in motion of the wall of the LV were observed (table 1). Aortic velocity time integral did increase at both stages (16–27%), reflecting the increase in CO. A 20% decrease in LVESA and an 11% increase in LVEDA resulted in an increase in FAC from 44 to 60% (fig. 1). Lateral mitral annulus motion increased by 38%, mostly at the final stage of the ANH, and correlated with the changes in FAC (\( r = 0.78 \)). Of the diastolic variables, only MEV\textsubscript{max} and maximal systolic forward pulmonary venous flow changed significantly compared with baseline values, but the trend for MAV\textsubscript{max} and maximal diastolic forward pulmonary venous flow was also upward in all patients during ANH. Deceleration time and isovolumic relaxation time did not change significantly, and the ratio between duration of atrial-related mitral and pulmonary venous flow26 remained around 2 throughout the study.
Comparative Studies

CO_Doppler measurements correlated with CO_TD (n = 36, r = 0.78, SEE = 1.1), and the line (y = 0.71x + 1.1) did not differ significantly from the line of identity (fig. 2). The change in MEV_max correlated poorly with the change in CO (r = 0.42), but the degree of hemodilution correlated well with the change in CO (r = 0.85), the change in FAC (r = −0.81), and the percentage change in systemic vascular resistance (r = 0.80). There was no correlation between LVEDA and PCWP (r = 0.07) despite the fact that both increased slightly but significantly during hemodilution (individual data in fig. 3).

Reproducibility

The interindividual coefficients of variation for measurements of aortic annulus diameter, velocity time integral, and LV areas were small (<4%; table 2). For diastolic Doppler measurements, the reproducibility was less, especially for pulmonary venous flow variables and isovolumic relaxation time.

Discussion

Hemodilution reduces the viscosity of the blood and causes dilatation in some vascular beds, which then

Table 1. Results from Hemodynamic Monitoring during ANH (n = 8)

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>HD1</th>
<th>HD2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin (g/l)</td>
<td>122.1 (7.9)</td>
<td>98.0 (3.2)*</td>
<td>79.4 (4.9)*</td>
</tr>
<tr>
<td>Volume withdrawn (ml)</td>
<td></td>
<td>742 (425)</td>
<td>714 (254)</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>75 (14)</td>
<td>75 (14)</td>
<td>74 (13)</td>
</tr>
<tr>
<td>Invasively</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>83 (10)</td>
<td>80 (8)</td>
<td>82 (9)</td>
</tr>
<tr>
<td>CVP (mmHg)</td>
<td>11.4 (3.0)</td>
<td>11.9 (4.3)</td>
<td>12.4 (4.4)</td>
</tr>
<tr>
<td>MPAP (mmHg)</td>
<td>18.3 (4.5)</td>
<td>20.2 (3.6)</td>
<td>21.1 (3.7)</td>
</tr>
<tr>
<td>PCWP (mmHg)</td>
<td>12.0 (4.8)</td>
<td>13.8 (2.9)</td>
<td>14.5 (3.3)*</td>
</tr>
<tr>
<td>CO_TD (l/min)</td>
<td>4.9 (1.5)</td>
<td>5.6 (1.7)*</td>
<td>6.1 (1.7)*</td>
</tr>
<tr>
<td>SVr (ml)</td>
<td>66 (22)</td>
<td>77 (24)*</td>
<td>84 (27)*</td>
</tr>
<tr>
<td>SV_Doppler (ml)</td>
<td>1290 (470)</td>
<td>1070 (420)*</td>
<td>980 (350)*</td>
</tr>
<tr>
<td>LVSW (g/m)</td>
<td>69 (17)</td>
<td>79 (23)*</td>
<td>89 (20)*</td>
</tr>
<tr>
<td>RVSW (g/m)</td>
<td>13 (2)</td>
<td>17 (4)</td>
<td>20 (5)*</td>
</tr>
<tr>
<td>By echocardiography</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VTI (cm)</td>
<td>19.0 (4.1)</td>
<td>22.0 (5.5)*</td>
<td>24.5 (6.5)*</td>
</tr>
<tr>
<td>SV_Doppler (cm²)</td>
<td>60 (17)</td>
<td>70 (23)*</td>
<td>78 (25)*</td>
</tr>
<tr>
<td>LVEDA (cm²)</td>
<td>8.8 (2.1)</td>
<td>7.7 (2.3)*</td>
<td>7.0 (1.8)*</td>
</tr>
<tr>
<td>LVEDA (cm²)</td>
<td>15.8 (3.7)</td>
<td>16.6 (3.6)</td>
<td>17.6 (3.7)*</td>
</tr>
<tr>
<td>FAC%</td>
<td>44 (7)</td>
<td>54 (10)*</td>
<td>60 (9)*</td>
</tr>
<tr>
<td>LMAM (cm)</td>
<td>1.6 (0.4)</td>
<td>1.9 (0.2)</td>
<td>2.2 (0.3)*</td>
</tr>
<tr>
<td>LTAM (cm)</td>
<td>1.9 (0.6)</td>
<td>2.2 (0.5)</td>
<td>2.3 (0.7)</td>
</tr>
<tr>
<td>IVRT (ms)</td>
<td>68 (9)</td>
<td>57 (14)</td>
<td>58 (6)</td>
</tr>
<tr>
<td>DT (ms)</td>
<td>109 (23)</td>
<td>103 (27)</td>
<td>101 (18)</td>
</tr>
<tr>
<td>MEV_max (m/s)</td>
<td>0.53 (0.04)</td>
<td>0.61 (0.12)</td>
<td>0.70 (0.12)*</td>
</tr>
<tr>
<td>MAV_max (m/s)</td>
<td>0.37 (0.16)</td>
<td>0.36 (0.10)</td>
<td>0.42 (0.14)</td>
</tr>
<tr>
<td>MAV_max (ms)</td>
<td>135 (31)</td>
<td>131 (15)</td>
<td>126 (14)</td>
</tr>
<tr>
<td>ARV_max (m/s)</td>
<td>0.17 (0.07)</td>
<td>0.12 (0.06)</td>
<td>0.14 (0.08)</td>
</tr>
<tr>
<td>ARV_max (ms)</td>
<td>58 (34)</td>
<td>61 (40)</td>
<td>63 (43)</td>
</tr>
<tr>
<td>PV (-m/s)</td>
<td>0.41 (0.10)</td>
<td>0.50 (0.11)</td>
<td>0.57 (0.13)*</td>
</tr>
<tr>
<td>PV (-m/s)</td>
<td>0.36 (0.10)</td>
<td>0.38 (0.14)</td>
<td>0.52 (0.19)</td>
</tr>
</tbody>
</table>

Baseline measurements and measurements after the first (HD1) and second (HD2) stage of hemodilution (mean values [SD]).

ARV_max = maximal duration of atrial pulmonary flow reversal; ARV_max = maximal velocity of atrial pulmonary flow reversal; CO_TD = cardiac output measured by thermodilution; CVP = central venous pressure; DT = mitral inflow deceleration time; FAC = fractional left ventricular area change; IVRT = isovolumic relaxation time; LMAM = lateral mitral annular motion; LTAM = lateral tricuspid annular motion; LVEDA = end-diastolic cross-sectional area of the left ventricular cavity; LVEDA = end-diastolic cross-sectional area of the left ventricular cavity; LVSW = left ventricular stroke work; MA_TAD = mitral atrial filling flow duration; MAP = mean arterial pressure; MAV_max = mitral atrial filling maximal flow velocity; ME = mitral early filling wave; MEV_max = mitral early filling maximal flow velocity; MPAP = mean pulmonary arterial pressure; PCWP = pulmonary capillary wedge pressure; PVD = maximal diastolic forward pulmonary venous flow velocity; PVR = pulmonary vascular resistance; PVS = maximal systolic forward pulmonary venous flow velocity; RVSW = right ventricular stroke work; SV = stroke volume; SVR = calculated systemic vascular resistance; VTI = Doppler velocity-time integral of the aortic flow.

* Indicates a significant change (P < 0.05).

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changes the pressure profile in the series-coupled vascular components and causes alterations in capillary pressures and hence changes in net filtration. Additionally, the sympathetic and myogenic tone of the vessels may change. At a constant blood volume, the cardiac filling pressure increases. If normovolemic hemodilution is done in such a way as to keep the cardiac filling pressure constant, this could entail some degree of hypovolemia that might in turn infringe on clinical safety margins. Laks et al. reported an increase in blood volume following administration of Ringer’s acetate and colloids on the same volume basis that we adopted, but the osmotic pressure of the protein solution they used was significantly higher. The colloid osmotic pressure of the 4% albumin solution has been reported to be only one half of that of normal human serum. Following hemodilution, there may be a net loss of blood volume as a result of the enhanced transcapillary extravasation rate of albumin and fluid, which by itself would require extra volume supplementation. It is not correct to claim normovolemia in the sense that it represents a stable equilibrium unless blood volume is repeatedly measured. Hence, substitution with an identical volume of the 4% albumin solution alone would not be useful for “isovolemic” hemodilution. Crystalloid is generally considered to be administered on a 3–4:1 basis to prevent volume deficit. To obtain a clinically safe and approxi-

<table>
<thead>
<tr>
<th>CV%</th>
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<tr>
<td>VTI</td>
</tr>
<tr>
<td>LV areas</td>
</tr>
<tr>
<td>Aortic annulus diameter</td>
</tr>
<tr>
<td>DT</td>
</tr>
<tr>
<td>IVRT</td>
</tr>
<tr>
<td>MAV_max</td>
</tr>
<tr>
<td>MEV_max</td>
</tr>
<tr>
<td>PVS</td>
</tr>
<tr>
<td>PVD</td>
</tr>
</tbody>
</table>

CV = coefficient of variation; DT = mitral inflow deceleration time; IVRT = isovolumic relaxation time; LV = left ventricular; MAV_max = mitral atrial filling maximal flow velocity; MEV_max = mitral early filling maximal flow velocity; PVD = maximal diastolic forward pulmonary venous flow velocity; PVS = maximal systolic forward pulmonary venous flow velocity; VTI = Doppler velocity; time integral of the aortic flow.

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mately normovolemic hemodilution, we therefore re-
placed the withdrawn blood with an equal volume of 4% 
albumin solution and the same volume of crystalloid 
solution. Anesthesia itself affects the cardiovascular re-
sponse to ANH, and the effect of anesthesia was not evalu-
ated in this study. During the prolonged anesthesia, how-
ever, hemodynamic variables did not change before ANH.

The decrease in systemic vascular resistance and in-
crease in CO have been attributed to reduced blood vis-
cosity\(^5,6\) and increased venous return. In our study, as 
in earlier human studies of ANH,\(^8,9\) HR, mean systemic 
arterial pressure, and mean pulmonary artery pressure re-
maincd constant. Weiskopf et al.\(^{12}\) reported in con-
scious humans that ANH 30 min after administration of 
propofol 50–150 µg/kg/min induced the same increase 
in SV as we found but also induced a concomitant con-
tinuous increase in HR. We did not see any increase in 
HR, but we did not evaluate the effect of anesthetics.

The low dose of fentanyl at induction together with 
thiopental and the prolonged stabilization period in our 
study makes it less plausible that anesthetics could ex-
plain all the difference. Increased HR during ANH and 
hypovolemia is likely to occur when the amount of 
volume given is targeted at keeping filling pressures 
constant, as in the study by Weiskopf et al.\(^{12}\) As expect-
ed,\(^5\) within the ranges of hemoglobin we saw in our 
study (70–138 g/l) the changes in CO were proportional 
to the degree of hemodilution.

During hemodilution, mixed venous oxygen saturation 
is not considered a reliable indicator of tissue hypoxia,\(^ {50}\) 
and it was not measured in this study. Animal studies 
have shown that normal coronary arteries respond to 
acute anemia by substantial dilatation before oxygen 
delivery is affected.\(^{31}\) This is in agreement with our 
study; when hemoglobin was reduced to less than 80 g/l 
and cardiac stroke work increased, neither systolic re-
gional motion of the LV wall nor CO deteriorated. Catoire 
et al.\(^{21}\) an improved hemodynamic tolerance to 
aortic clamping by a similar degree of hemodilution in 
patients with coronary artery disease.

The measurement of diastolic function during ANH 
may help to avoid inadequate oxygen delivery and up-
take. The deterioration of diastolic function is one of 
the earliest signs of acute myocardial ischemia,\(^ {32–34}\) and 
unlike regional deterioration of systolic function, there is 
no mechanism during advanced hemodilution to com-
 pensate for regional diastolic dysfunction in territories in 
which the coronary flow is compromised.\(^ {35,36}\) During 
ANH in our study, increased end-diastolic areas of the 
left ventricle were seen in all but one patient, and PCWP 
also increased slightly. In individual patients, however, 
PCWP was not predictable from LVEDA increase or from 
the diastolic echocardiographic measurements of mitral 
inflow and pulmonary venous flow. These findings sug-
gest that, in physiologically normal individuals, the in-
crease in preload during ANH is an effect of increased 
venous return and is not due to decreased LV diastolic 
function. At those low-normal filling pressures, the 
changes in pressures are small, and normal variations in 
compliance may play an important part\(^ {18,37}\) and may 
contribute to the interindividual discordance between 
volume and pressure. The diastolic LV pressure–volume 
correlation accurately describes the ventricular relax-
ation and filling.\(^ {32}\) Although the mitral and pulmonary 
vein echocardiographic measurements are strongly depen-
dent on left atrial pressure and pressure gradients,\(^ {38,39}\) no 
significant changes were detected, suggesting that diastolic 
function was unaltered. The combination of a limited 
change in pressures, a lower reproducibility of diastolic 
Doppler measurements, and a limited number of patients 
may, however, all contribute to the result.

We have confirmed that CO measurements using trans-
gastric Doppler from the LV outflow tract is possible 
with reproducibility similar to that reported with trans-
 thoracic echocardiographic studies.\(^ {40}\) We conclude that 
a degree of hemodilution to 80 g/l does not compromise 
the systolic or diastolic myocardial function in normal 
humans and confirm that ANH causes a decrease in 
vascular resistance and an increase in CO. This increase 
in CO is proportional to the degree of hemodilution and 
is generated by an increased SV resulting from increased 
ejection fraction and increased preload.

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