Case Reports/Case Series

Case report: Retroperitoneoscopic pheochromocytoma removal in an adult with Eisenmenger's syndrome

[Présentation de cas : Ablation rétropéritonéoscopique d'un phéochromocytome chez un adulte souffrant du syndrome d'Eisenmenger]

Geoff A. Bellingham MD,* Achal K. Dhir MD FRCA,* Patrick P. Luke MD FRCS(C)†‡§

Purpose: Patients with uncorrected or palliated, complex congenital heart lesions requiring surgery can benefit from laparoscopic techniques, but retroperitoneal insufflation may render them hemodynamically unstable. Alterations in cardiopulmonary physiology during retroperitoneal insufflation have been studied, yet there are no cases detailing this approach in patients with congenital heart lesions. We present a case of a pheochromocytoma removal via retroperitoneoscopy in a patient with a palliated, complex heart lesion.

Clinical features: A 28-yr-old woman was admitted for removal of a pheochromocytoma through retroperitoneoscopy. The main feature of her heart disease was a complete atrioventricular canal defect. She eventually developed Eisenmenger's syndrome and became chronically cyanotic. Retroperitoneal insufflation with CO_2 gas did not change hemodynamic variables. Significant increases in peak airway pressures were encountered, possibly due to the distending effects of insufflation, or due to increasing the minute ventilation to reduce exogenous CO_2. Arterial CO_2 remained stable, but a significant increase between end-tidal and arterial levels became apparent with insufflation. Tumour manipulation led to systemic (and possibly pulmonary) hypertension, which exacerbated ventricular dysfunction. This condition resulted in atrioventricular valve regurgitation, as seen on transesophageal echocardiography, and diminished pulmonary blood flow with subsequent desaturation. These changes resolved with antihypertensive medications. The patient's trachea was extubated four hours postoperatively, and she recovered uneventfully.

Conclusion: Patients with altered cardiopulmonary physiology may tolerate retroperitoneoscopic insufflation with relative hemodynamic stability. Appropriate use of short-acting, vasoactive drugs and aggressive monitoring of $PaCO_2$ and hemodynamic variables is required.

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Objectif *: Les patients ayant des lésions cardiaques congénitales complexes non corrigées ou palliées nécessitant une chirurgie peuvent profiter de techniques laparoscopiques ; toutefois, un rétropneumopéritoine peut rendre leur hémodynamie instable. Les modifications de la physiologie cardiopulmonaire pendant un rétropneumopéritoine ont été étudiées, mais il n'existe aucun cas décrivant cette approche chez des patients souffrant d'anomalies cardiaques congénitales. Nous présentons un cas d'ablation de phéochromocytome par rétropéritonéoscopie chez un patient ayant une anomalie cardiaque congénitale complexe palliée.*

Éléments cliniques *: Une femme de 28 ans a été admise pour une ablation de phéochromocytome par rétropéritonéoscopie. Une déficience totale de son canal atrio-ventriculaire constituait l'élément principal de sa maladie cardiaque. Elle a finalement développé un syndrome d'Eisenmenger et est devenue chroniquement cyanotique. Un rétropneumopéritoine avec du gaz CO_2 n'a pas modifié les variables hémodynamiques. Des augmentations significatives dans les pics de pression ventilatoire ont été observées,*

From the Department of Anesthesia and Perioperative Medicine,* University of Western Ontario, the Division of Urology,† University of Western Ontario, the Multi-Organ Transplant Program (MOTP),‡ and the Canadian Surgical Technologies and Advanced Robotics (CSTAR),§ London, Ontario, Canada.

Address correspondence to: Dr. Achal K. Dhir, Department of Anesthesia and Perioperative Medicine, London Health Sciences Centre and the University of Western Ontario, 339 Windermere Road, London, Ontario N6A 5A5, Canada. Phone: 519-663-3022; Fax: 519-663-2957; E-mail: adhir2@uwo.ca

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possiblement provoquées par les effets distensifs de l'insufflation ou par l'augmentation de la ventilation minute pour réduire le CO_2 exogène. Le CO_2 artériel est resté stable, mais une augmentation significative entre les niveaux télé-expiratoire et artériel est apparue lors de l'insufflation. La manipulation tumorale a provoqué une hypertension systémique (et possiblement pulmonaire), ce qui a exacerbé la dysfonction ventriculaire. La conséquence de cette condition a été une régurgitation des valves atrio-ventriculaires, telle qu'observées par échocardiographie transoesophagienne, et un débit sanguin pulmonaire réduit avec une désaturation subséquente. Ces modifications ont pu être contrées avec des médicaments anti-hypertenseurs. La trachée de la patiente a été extubée quatre heures après l'opération, et elle s'est rétablie normalement.

***Conclusion** : Les patients présentant une physiologie cardio-pulmonaire altérée pourraient tolérer un rétropneumopéritoine et maintenir une hémodynamie relativement stable. L'utilisation adéquate de médicaments vasoactifs à action courte et une surveillance agressive de la $PaCO_2$ et des variables hémodynamiques sont nécessaires.*

We present a case which describes the use of retroperitoneoscopy to remove a pheochromocytoma from a 28-yr-old patient with complex congenital heart disease and Eisenmenger's syndrome. This report serves to demonstrate that retroperitoneoscopic surgery may not be associated with major, adverse hemodynamic or ventilatory effects, and it highlights the feasibility of this approach for patients with congenital heart lesions. The Institutional Ethics Board of the University of Western Ontario approved reporting of this case.

Case report

The patient was a 28-yr-old female (height 157 cm, weight 37.7 kg) born with a complete atrioventricular (AV) canal defect, a common AV valve, a bilateral superior vena cavae, and a common atrium. At eight months of age, pulmonary artery (PA) banding was performed to diminish PA blood flow. At five years of age, cardiac catheterization revealed pulmonary hypertension (84/48 mmHg) which was 75% of systemic levels. A concurrent, right upper lobe lung biopsy revealed pulmonary arterioles with intimal thickening and fibrosis with narrowing of the vascular lumen. Elevated PA pressures led to the development of Eisenmenger's syndrome and chronic cyanosis. She eventually required the use of home oxygen at 4 $L \cdot min^{-1}$ via nasal prongs. Her baseline oxygen saturation on supplemental oxygen ranged between 72% and 80%. At the time of her most recent cardiac assessment, the patient could walk one flight of stairs with mild dyspnea.

During routine cardiac follow-up, a right adrenal mass was identified as an incidental finding on echocardiography. In retrospect, when questioned about symptoms, the patient admitted to experiencing paroxysms of dyspnea, diaphoresis, and palpitations. A computed tomography scan of the abdomen showed a mass in the right adrenal gland measuring 3.5 × 4 cm, and a 123-iodine, meta-iodobenzylguanidine body survey confirmed the diagnosis of a single pheochromocytoma. Blood work revealed increased levels of plasma norepinephrine (69,300 $pmol \cdot L^{-1}$; normal ≤ 2,800 $pmol \cdot L^{-1}$), plasma epinephrine (583 $pmol \cdot L^{-1}$; normal ≤ 320 $pmol \cdot L^{-1}$), and plasma dopamine (274 $pmol \cdot L^{-1}$; normal ≤ 210).

Echocardiography confirmed the previous cardiac anomalies, in addition to a post-stenotic aneurysmal dilation of the main PA measuring 50 mm in width (Figures 1 and 2). The pressure gradient across the banded area was 45–50 mmHg. The left ventricle was mildly dilated with moderate systolic impairment; while the right ventricle had moderate systolic dysfunction with significant hypertrophy (echocardiography image loops 1–5, available as Additional Material at www.cja-jca.org).

Her blood pressure was 115/64 mmHg, heart rate 110 $beats \cdot min^{-1}$, and oxygen saturation 72% on 6 $L \cdot min^{-1}$ of oxygen. Doxazosin 2 mg *po* bid, metoprolol 25 mg *po* bid, and hydrochlorothiazide 12.5 mg *po* OD were used for preoperative management of heart rate and blood pressure, and the patient was discharged home until an operative date could be arranged.

The day prior to surgery, the patient was admitted to the intensive care unit to assess her hemodynamic status and to provide further optimization, if needed. The baseline vital signs were as summarized in the Table. The patient did not experience any paroxysms of tachycardia or hypertension over 24 hr, and she did not demonstrate significant orthostatic hypotension, indicating euvolemia. From these clinical findings, adrenergic blockade was deemed adequate. Laboratory investigations revealed hemoglobin of 186 $g \cdot L^{-1}$, hematocrit of 0.57, and platelet count of 212 × $10^9 \cdot L^{-1}$. Her international normalized ratio and partial thromboplastin time were 1.2 and 32, respectively. Arterial blood gases showed a partial pressure of oxygen and CO_2 of 37 and 48 mmHg, respectively; her pH was 7.41.

Anesthetic induction was achieved using doses of propofol, midazolam, and fentanyl titrated to blood

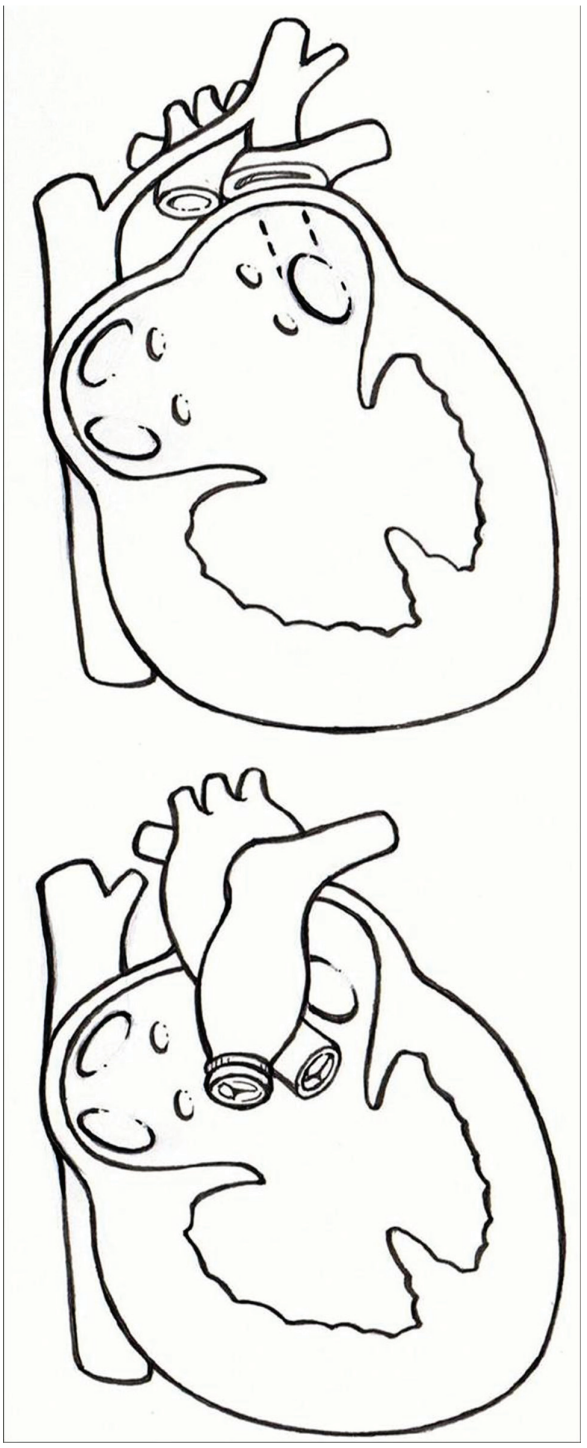

FIGURE 1 Schematic representation of cardiovascular pathology in this patient. *Upper Panel:* Aorta and pulmonary artery removed, showing a complete atrioventricular (AV) canal defect, common AV valve, bilateral superior vena cavae, common atrium, absence of coronary sinus, and right ventricular hypertrophy. Pulmonary veins return to both left and right atria. *Lower Panel:* Pulmonary artery banding with post-stenotic aneurysmal dilation.

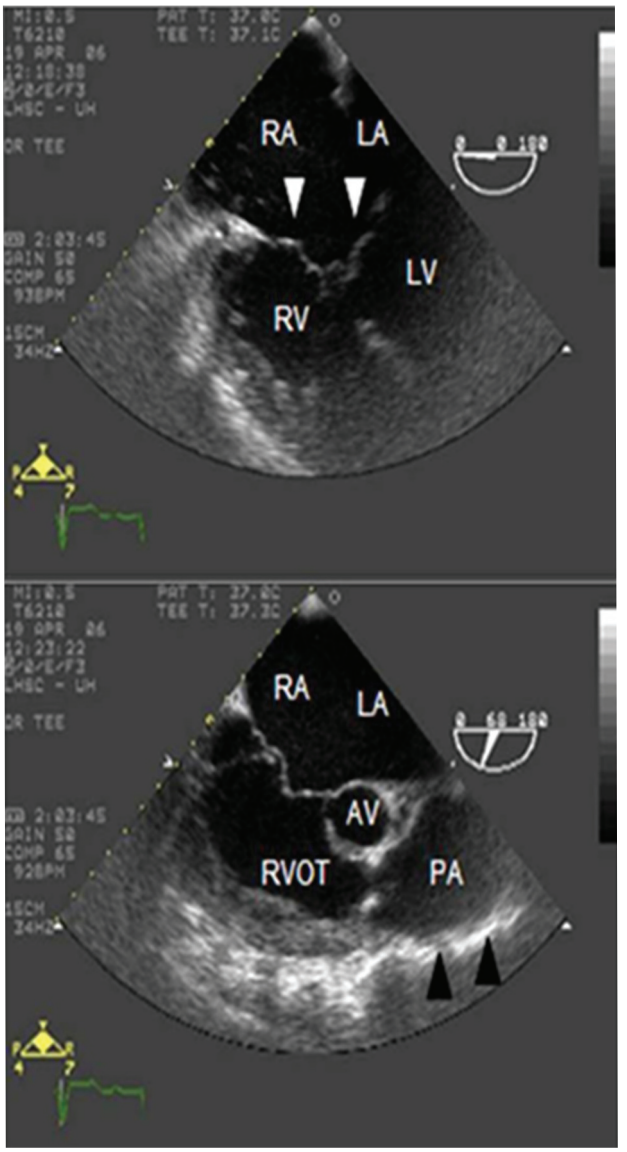

FIGURE 2 Intraoperative, transesophageal echocardiography: *Upper Panel:* Four- chamber view showing complete AV canal defect and common AV valve (white arrows). RA = right atrium; LA = left atrium; RV = right ventricle; LV = left ventricle. *Lower Panel* RV inflow/outflow view showing post-stenotic aneurysmal dilation of the main pulmonary artery (black arrows) measuring 50 mm in width. AV = aortic valve; RVOT = right ventricular outflow tract; PA = pulmonary artery.

pressure measured via an arterial line. The patient was intubated without significant changes in hemodynamics (Table). Standard monitors were applied, in addition to a 5 lead electrocardiogram (ECG) for ST segment monitoring.

Given the complexity of her cardiovascular physiology, an adult sized transesophageal echocardiography

TABLE

	Baseline	*Pre-induction*	*Post-intubation*	*Incision*	*Pre-insufflation*	*Post-insufflation**	*End of surgery**	*1 hr post surgery**
Heart rate($\cdot min^{-1}$)	79	115	95	110	100	98	120	85
Blood pressure (mmHg)	120/70	145/85	110/70	130/85	125/80	120/75	86/45	110/60
Central venous pressure (mmHg)	14	18	24	31	31	35	20	17
F_IO_2	4 $L\cdot min^{-1}$ via np§	4 $L\cdot min^{-1}$ via np§	60%	60%	60%	60%	90%	60%
Oxygen saturation	80%	74%	74%	70%	70%	72%	82%	80%
End-tidal CO_2 (mmHg)			49	35	36	32	30	

*Patient placed on norepinephrine infusion at 3 $\mu g\cdot min^{-1}$ to support blood pressure after adrenal vein clipping. §Patient breathing spontaneously with oxygen supplementation at 4 $L\cdot min^{-1}$ via nasal prongs (np).

(TEE) probe was used to assess cardiac function. Focus of the TEE study was global biventricular function and the degree of AV regurgitation. AV regurgitation was examined, since it acted as an indicator of increased myocardial strain from increasing afterload.

A PA catheter was not used, since the septal defect and the stenotic PA would have made placement very difficult. There was additional concern that a PA catheter could potentially cause vascular injury of the PA aneurysm. Furthermore, cardiac output measurements using thermodilution would be inaccurate in the presence of an intracardiac shunt. Unfortunately, given the rapidly changing hemodynamic profiles, it was not possible to calculate values for cardiac output and vascular resistances quickly enough using TEE during the case.

Since there was no monitor to detect increasing PA pressures and vascular resistance, oxygen saturation and central venous pressures (CVP) were used as surrogate markers for these variables. Rising pulmonary vascular resistance (PVR) would cause right ventricular dysfunction and decreased pulmonary blood flow, resulting in; increased right to left shunting, increased CVP, increased AV regurgitation, and a decrease in oxygen saturation.

Prior to the diagnosis of pheochromocytoma, the patient was being considered for pulmonary vasodilating therapy. Although vasoreactivity tests, via right heart catheterization, were postponed until after the surgery, it was decided that use of nitric oxide (NO) and milrinone could be beneficial, intraoperatively, to help decrease PVR.

Nitric oxide was started immediately after intubation at 20 ppm. Next, low dose milrinone was administered at 0.3 $ug\cdot kg^{-1}\cdot min^{-1}$ and titrated carefully, to prevent a decrease in blood pressure associated with its systemic vasodilating properties.

Anesthesia was maintained with sevoflurane (end-tidal concentrations ranging from 1.7 to 2.3%) in a 1:1 air/oxygen mixture. Intravenous fentanyl boluses were administered throughout the case to provide analgesia. The patient was placed in a Trendelenberg (20° head-down), left lateral decubitus position with flexion of the operating table.

The posterior perinephric space was developed with a balloon expander. Retroperitoneal insufflation of CO_2 gas to 15 mmHg was not accompanied with changes in hemodynamic variables (Table), although peak airway pressure increased from 22 to 37 cm H_2O while using volume controlled ventilation (500 mL tidal volume with 5 cm H_2O of positive end-expiratory pressure). This increase was possibly due to the tissue distending effects of insufflation, or due to increasing minute ventilation (by increasing the respiratory rate) from 7.9 $L\cdot min^{-1}$ to a maximum of 13.4 $L\cdot min^{-1}$, in anticipation of elevated levels of CO_2. The baseline arterial CO_2 was 53 mmHg before insufflation and increased to a maximum of 56 mmHg during insufflation. The 7 mmHg difference between end-tidal CO_2 and arterial CO_2, that was present initially, increased to 14 mmHg during insufflation.

The operating time was four hours. Tumour manipulation was accompanied by increases in blood pressure and heart rate (maximum blood pressure and heart rate values were 140/95 mmHg and 125 $beats\cdot min^{-1}$, respectively). These hemodynamic changes were managed using phentolamine (1 mg incremental boluses to a maximum of 4 mg), in addition to esmolol and nitroprusside infusions. There were no ST segment changes. Oxygen saturation decreased (nadir = 65 %) during hypertensive episodes. These changes were accompanied by a rise in CVP from 20 to 41 mmHg. Reducing insufflation pressures from 15 to 10 mmHg failed to reduce CVP. Transesophageal

echocardiography findings revealed worsening of the AV valve regurgitation jet (transesophageal image loop 5, available as Additional Material at www.cja-jca.org). Once the blood pressure improved, the regurgitation diminished and oxygen saturation and CVP returned to baseline values. Clipping of the adrenal vein was associated with a sudden decrease in blood pressure to 88/50 mmHg, and hemodynamic support was initiated with a norepinephrine infusion.

Postoperatively, the patient was transferred to the intensive care unit, and four hours later, when she was awake and responding appropriately to command, her trachea was extubated. Her early and late postoperative course was uneventful, and she was discharged from hospital on postoperative day eight with stable hemodynamics and mild hypoglycemia, secondary to the removal of the pheochromocytoma. At one year follow-up, she reported dramatic improvements in her symptomology, specifically noting a lack of paroxysms of tachycardia, diaphoresis, and dyspnea. Her oxygen saturation, on room air, was 72%, with a heart rate of 88 beats·min^{-1} and blood pressure of 120/60 mmHg.

Discussion

Important hemodynamic goals for patients with Eisenmenger's syndrome are to prevent increases in PVR and to maintain an adequate, systemic vascular resistance (SVR). If SVR were to decrease, or if PVR were to increase, there would be an augmentation of right to left shunting leading to worsening cyanosis.[1] Retroperitoneal insufflation with CO_2 may lead to changes that compromise these goals.

In this case, many of the anesthetic interventions were aimed at minimizing the amount of right to left intracardiac shunting, resulting from increases in PVR secondary to hypercapnia or due to elevated catecholamine levels. Although the patient may have had established pulmonary hypertension, it was believed that any benefits provided by vasodilator therapy would outweigh the potential risks of the pharmacological interventions. Previous reports describe the utility of vasodilating therapies in similar patient populations.[2,3] This encouraged our use of NO and milrinone, intraoperatively. Milrinone was also beneficial, since it could have improved myocardial contractility through phosphodiesterase III inhibition, rather than acting on beta receptors, which were likely to have been down regulated and desensitized secondary to chronic heart failure. Of note, pulmonary vasoreactivity was previously examined in 23 adult patients with congenital heart disease and Eisenmenger's syndrome.[3] Hemodynamic profiles were evaluated using Swan-Ganz catheters to calculate total pulmonary resistance, cardiac index, and baseline shunt fraction. It was found that NO inhalation induced pulmonary vasodilation in 29% of patients. These studies suggested that the use of NO and milrinone would be beneficial for our patient.

A central concern for this patient was the potential to elicit pulmonary vasoconstriction from elevated $PaCO_2$ levels secondary to the CO_2 insufflation.[4] Carbon dioxide absorption, examined in several studies during retroperitoneoscopy, showed greater gas uptake compared to intraperitoneal insufflation.[5–7] Despite this, we managed to prevent an escalation in arterial CO_2 levels by increasing the respiratory rate during volume controlled ventilation. Frequent arterial blood gas sampling was done to ensure that increases in minute ventilation were adequate to diminish the possibility of hypercapnic pulmonary vasoconstriction. However, without direct monitoring of PVR, we were unable to document changes in pulmonary vasoreactivity.

The necessity for frequent CO_2 monitoring is further highlighted by the fact that end-tidal CO_2 measurements may not provide accurate representations of $PaCO_2$ in patients with cyanotic heart disease.[8,9] With a mixed cardiac lesion, alterations in pulmonary blood flow during anesthesia may lead to changes in the efficiency of CO_2 elimination. As seen in cyanotic children, systemic venous blood mixes with pulmonary venous blood and, as a result, increases $PaCO_2$ above end-capillary CO_2 levels ($PcCO_2$).[10,11] In this case, an increase in the end-tidal to arterial CO_2 gradient was noted during insufflation. Measurement of end-expiratory CO_2 is of limited value as an indirect measurement of $PcCO_2$, due to the variable arterial/end-expiratory gradient caused by anesthesia. However, the measurement of end-tidal CO_2 can be useful for recording changing trends in $PcCO_2$.[12]

The greatest change noted with retroperitoneal insufflation was an increase in peak airway pressures. This observation may have been due to tissue distension imposing a displacement of the diaphragm or due to the effects of increasing minute ventilation by increasing respiratory rate. Increased airway pressures can be encountered by increasing respiratory rate through augmentation of the turbulent component of airway resistance. Further concern regarding increased respiratory rate centered on the generation of breath stacking, which could enhance PVR by overdistending the lung tissue.[13] To avoid this complication and to allow for adequate expiration time, the inspiratory: expiratory ratio was held constant at 1:2 throughout the duration of the case.

Increased airway pressures were a concern, in this

case, since studies have demonstrated the potential to decrease right ventricular output with positive pressure ventilation.[14,15] However, we did not observe any appreciable difference in either the patient's hemodynamic values or in her oxygen saturation during the period of insufflation. Differences between intraperitoneal and retroperitoneal effects on the pulmonary system have been documented in a study examining respiratory changes in a porcine model.[16] Significant changes from baseline values were noted for both groups with respect to peak airway pressures, mean PA pressures, and PVR. However, changes in mean PA pressure and in peak airway pressures were significantly less for the retroperitoneal group, with no difference in PVR between groups. Intraperitoneal insufflation causes an initial augmentation in blood flow to the right heart.[16,17] This effect is secondary to extravascular pressure placed on large venous capacitance vessels forcing blood from the abdomen to the thorax. Increased preload could force a weakened myocardium to a less favourable position on the Starling curve, which could have led to acute congestive heart failure in our patient.

A previous study examining this phenomenon focused on changes in caval vein pressure gradients in humans during prone retroperitoneoscopy.[18] In contrast to intraperitoneal insufflation, retroperitoneal insufflation did not produce a pressure gradient in the inferior vena cava during transition from abdominal to thoracic compartments. This suggests that there may be no initial transfer of venous capacitance blood to the right heart, which may have been important in this case for maintaining cardiac stability and for avoiding an episode of acute congestive heart failure. In our case, we observed a consistent CVP during surgery, except during tumour manipulation.

There was little concern regarding the effects that retroperitoneoscopy might have on SVR. Hemodynamic changes have been documented in seven patients undergoing retroperitoneoscopic adrenalectomy, and little variability was reported.[19] This is important to note since, if SVR were to decrease, right to left shunting could increase exacerbating the cyanosis.[1] In the case we present, blood pressure remained stable until the time of tumour manipulation. As the blood pressure rose during tumour dissection, the regurgitant jet from the common AV valve worsened leading to an increase in CVP. These changes were successfully managed with the antihypertensive medications. The only time during this case, in which oxygen saturation decreased, occurred during hypertensive episodes. This may have been due to worsening ventricular dysfunction from increased PVR and SVR. Forward blood flow was likely diminished during these episodes. Treatment with vasodilating agents (phentolamine and nitroprusside), in addition to NO and milrinone, helped to increase oxygen saturation.

A limitation to the interpretation of this case is the lack of evidence for pulmonary vasoreactivity. There had been no recent, preoperative, right heart catheterization, and a Swan-Ganz catheter was not used for the reasons alluded to previously. Additionally, TEE could not be used to its full potential to help derive hemodynamic indices. This was due to the rapidly changing hemodynamics, intraoperatively, and the fact that such calculations require time to derive; this being a difficulty encountered during concurrent management of this complicated case.

In conclusion, this report highlights that, for patients with significant cardiopulmonary physiological alterations, who are subject to prolonged retroperitonoscopy procedures, hemodynamic stability is feasible with appropriate pharmacological interventions and with careful control of ventilatory parameters. In order to minimize alterations in vasomotor tone, frequent blood gas analysis is important to ensure adequate control of CO_2 levels. Additionally, TEE may serve as a useful guide to the tailoring of specific drug therapies to maintain perioperative hemodymamic stability.

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