HYPERTENSION PULMONAIRE :

MONITORING ET PRISE EN CHARGE DE L'INSTABILITÉ HÉMODYNAMIQUE

VINCENT GÉNÉREUX
R2 ANESTHÉSIE - UNIVERSITÉ DE MONTRÉAL

JOURNÉE SCIENTIFIQUE DES RÉSIDENTS
3 MAI 2014

Université de Montréal

CAS CLINIQUE

- Un soir de garde au CHRTR
- Grossesse ectopique rompue annoncée

CAS CLINIQUE

- F 32 , HAP associée à une sclérodermie (forme CREST)
  - Histoire : CF III / IV, 6MWT : 300 m, évolution clinique rapide en 1 an dysphagie aux solides et RGO fréquent.
  - Examen : orientée, dyspnéique, TA 110/65 FC 115, FR 30 SpO2 96 % (VM 50%), cou et visage cartonnés, ouverture buccale 2-3 cm. OMI bilatéral modeste.

CAS CLINIQUE

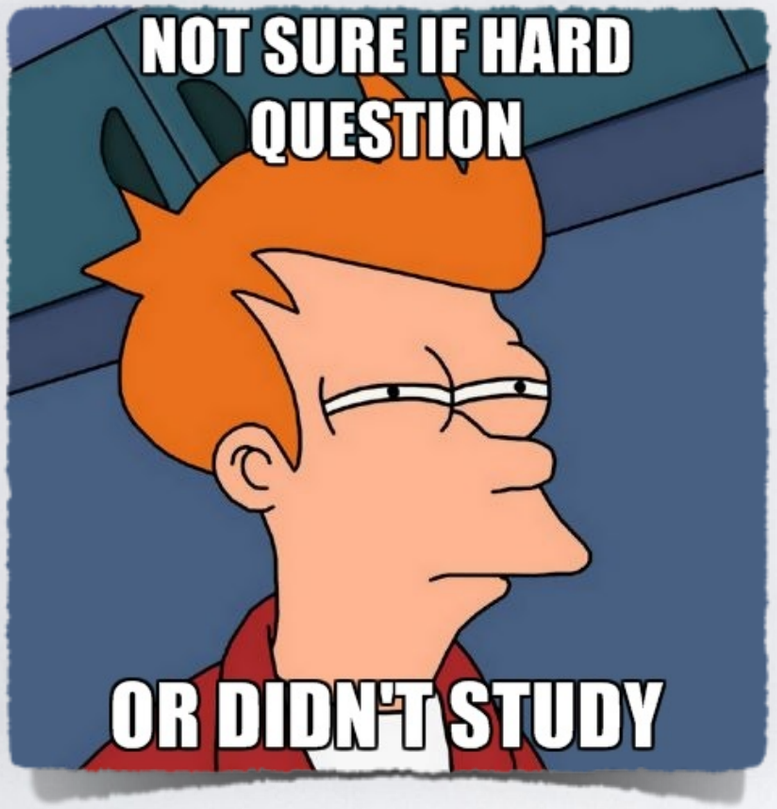
- Médication : HCTZ, epoprostenol, bosentan
- Test para-cliniques :
  - ECG - tachycardie sinusale, ondes P pointues et signes HVD
  - Gaz artériel : pH 7,35 PaO_2 80, $PaCO_2$ 30, HCO_3 18, lactates 4

OBJECTIFS

- Principaux :
  - Interprétation du monitoring invasif
    - **Traitement approprié de l'instabilité hémodynamique**
- Secondaires :
  - Évaluation préopératoire de la sévérité de l'hypertension pulmonaire

MA QUESTION POUR VOUS

- Advenant une instabilité hémodynamique peropératoire chez cette patiente, quel paramètre doit être pris en charge en priorité ?



A. TVC

B. PAP

C. RVP

D. PAPO

E. TAM

CLASSIFICATION HTAP

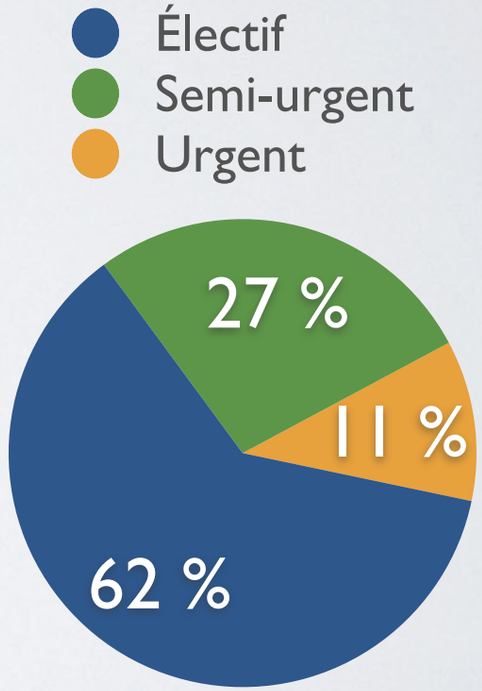
1. **Hypertension artérielle pulmonaire (HAP)**
    1. Idiopathique
    2. Associée à une maladie systémique
2. Secondaire à un atteinte ventriculaire gauche
3. Secondaire à maladie du parenchyme pulmonaire ou l'hypoxie
4. Maladie thromboembolique chronique
5. Étiologies multifactorielles et autres

PRISE EN CHARGE MÉDICALE

- Anticoagulation, O_2 à domicile, diurétiques
- BCC oraux
- ERA (bosentan) ou iPDE-5 (sildenafil)
- Prostanoïdes (epoprostenol, treprostinil, iloprost)
- Combinaisons

HTAP ET CHIRURGIE NON-CARDIAQUE

- Mortalité élevée en chirurgie urgente (15% vs 2%)
- Facteurs de risque morbidité/mortalité :
  - chirurgie urgente - OR 2,4
  - 6MWD < 400 m - OR 2.2



Eur Respir J 2013; 41: 1302–1307
DOI: 10.1183/09031936.00089212

Université de Montréal

PÉRIODE PRÉOPÉRATOIRE

- Tenter une **approche multidisciplinaire** :
  - Risque/bénéfice de l'intervention, surtout en urgence*
  - Gestion de l'anticoagulation
- Évaluer la cause et la sévérité de l'HTAP
  - Hx, Ex. phys., ECG, RXP, 6MWT, **ETT/ETO**, **cathétérisme cardiaque droit**
- Évaluer les comorbidités
- Optimiser/**poursuivre le traitement pour l'HTAP** si possible

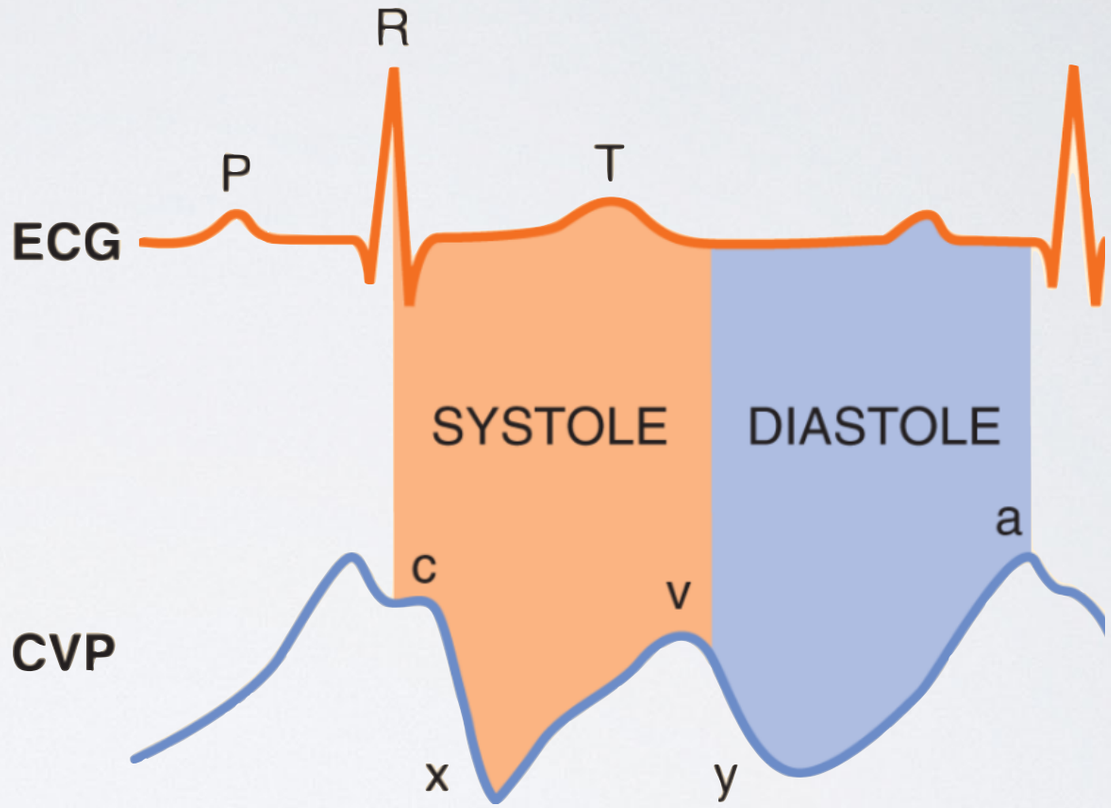
PEROPÉRATOIRE

MONITORING CLASSIQUE

- Standard
- Canule artérielle
- Voie centrale
- Cathéter artériel pulmonaire
- Échocardiographie

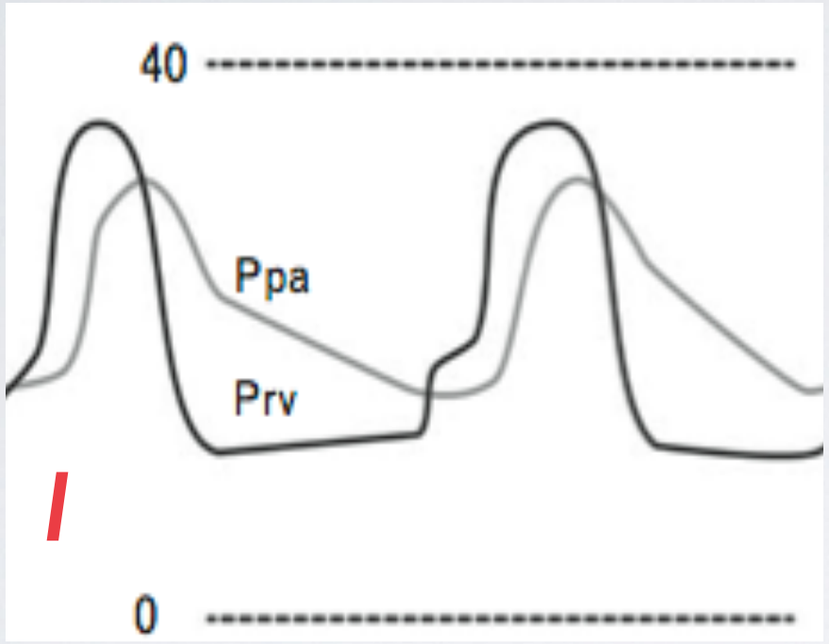
QUOI D'AUTRE ?

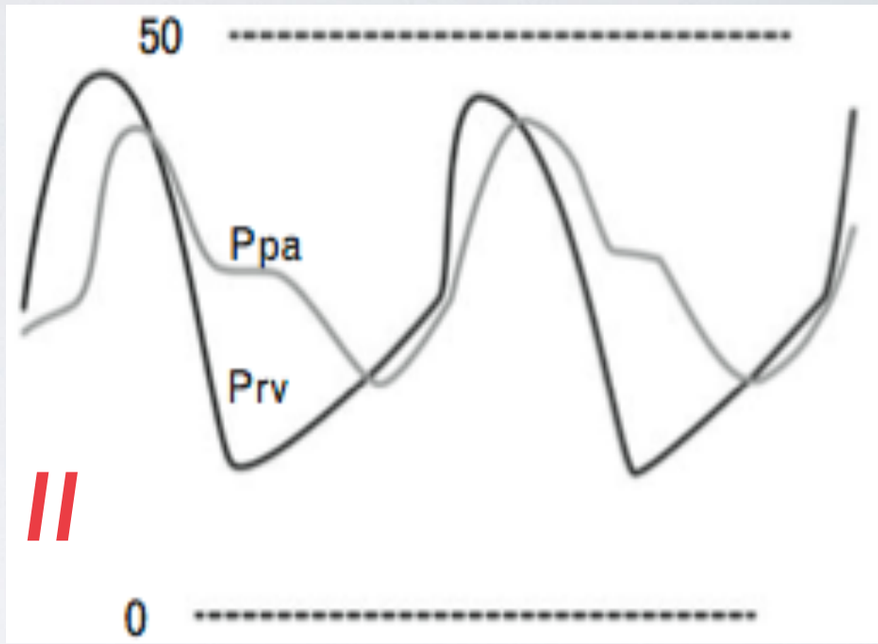
- **Courbe TVC**

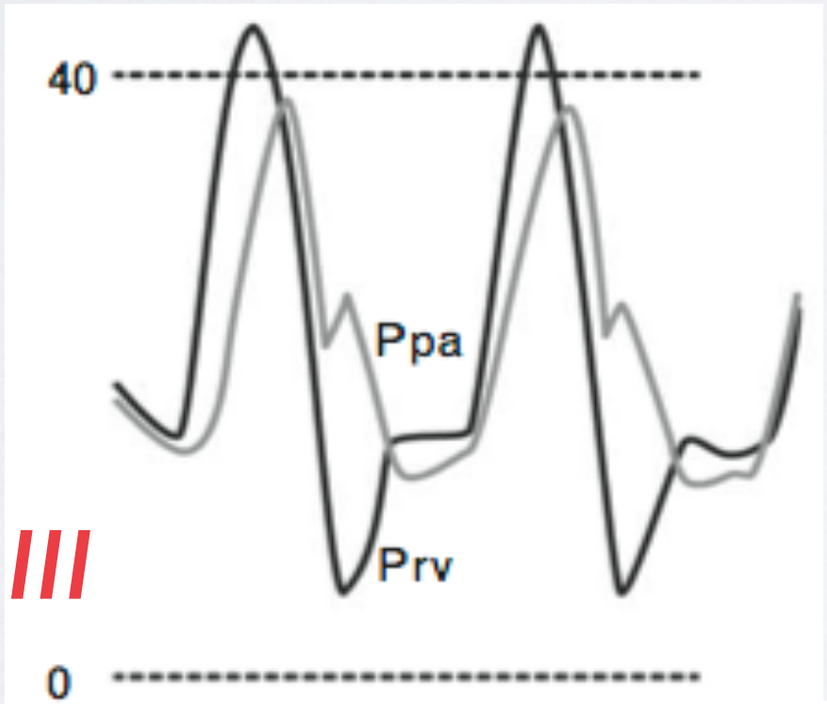


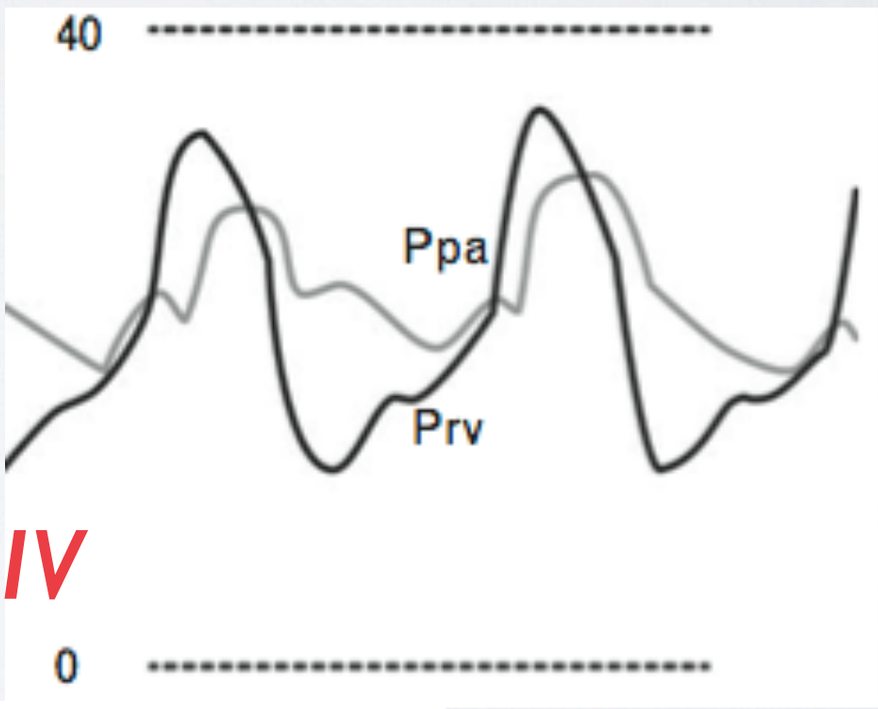
- **Saturométrie cérébrale**

COURBE VENTRICULAIRE DROITE









CURRENT OPINION **Perioperative right ventricular dysfunction**

André Yvan Denault[a,b], Francois Haddad[c], Eric Jacobsohn[d], and Alain Deschamps[a]

Université de Montréal

RÉFÉRENCES HÉMODYNAMIQUES

Paramètre	Normal	Anormal
PAPs	15-30 mmHg	> 30-40 mmHg
PAPm	9-16 mmHg	> 25 mmHg
RVP	1.1-1.4 WU 90-120 dyn*s*cm	modéré : 200-300 dyn*s*cm sévère : > 600 dyn*s*cm
ratio PAPm/TAM	< 25%	modéré : 33-50% sévère : >50%
ratio TAM/PAPm	> 4	< 4

Current Cardiology Reviews, 2010, 6, 1-14

PHYSIOPATHOLOGIE DE L'INSTABILITÉ HÉMODYNAMIQUE

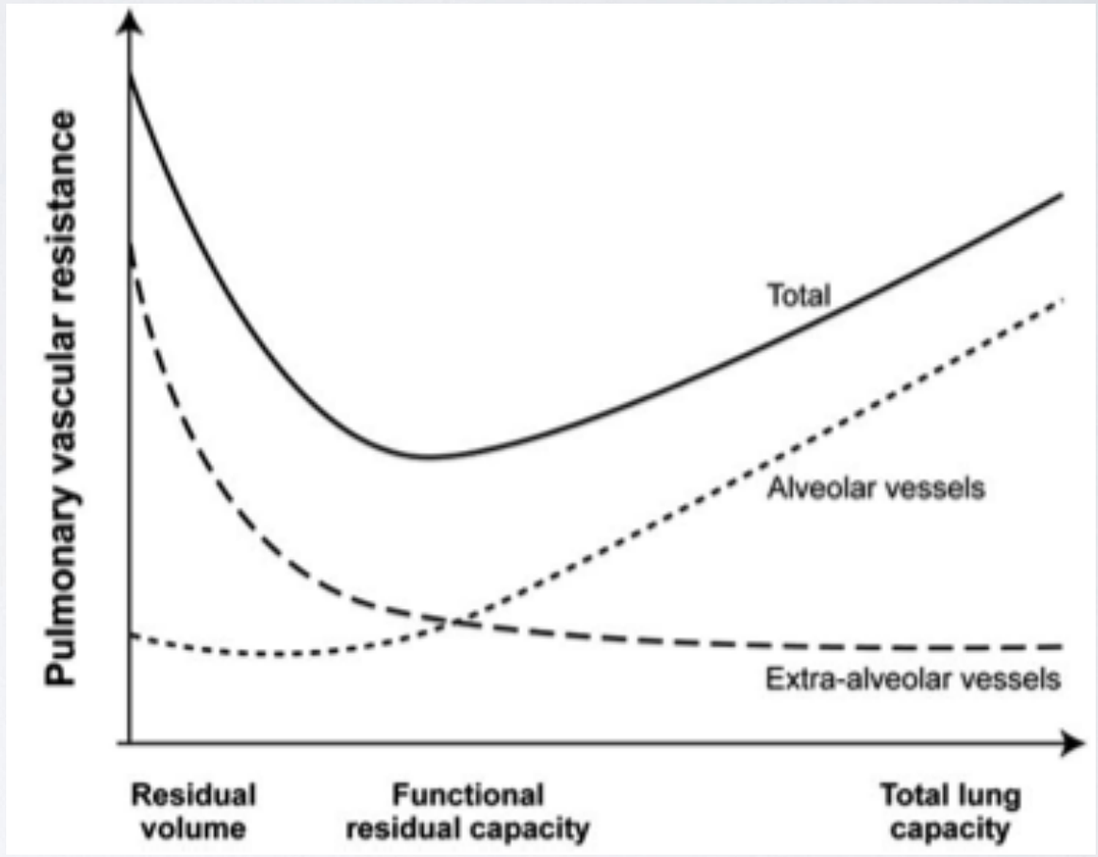
- Ventricule droit fragilisé
  - Post-charge
  - Perfusion myocardique droite
    - Sensible à la baisse du retour veineux
- Interdépendance ventriculaire
  - Dysfonction diastolique gauche induite
  - Contractilité gauche
  - Risque de shunt droit - gauche si FOP

PRÉVENTION DYSFONCTION VD - PRINCIPES GÉNÉRAUX

- Soutenir la TAM*
- Minimiser la RVP
- Autres buts hémodynamiques :
  - Maintenir précharge droite
  - Maintenir contractilité
  - Maintenir rythme sinusal
  - Viser une fréquence cardiaque 80-100 bpm

RÉSISTANCE VASCULAIRE PULMONAIRE

- Hypoxie / Hypercapnie
- Hypothermie
- Acidose
- Douleur / Adrénergisme
- Paramètres ventilatoires
- Phénomènes emboliques
- Arrêt de vasodilatateurs pulmonaires



AGENTS D'INDUCTION

Etomidate **Midazolam**	**Ketamine** **Propofol**	**Agents Inhalés**
Opioïdes		
Rocuronium **Succinylcholine**		

MAINTIEN

- Cocktail anesthésique sécuritaire:
  - Agents inhalés (sauf N_2O) ou IV
    - Opioïdes
    - Bloqueurs neuromusculaires
- Méthodes pharmacologiques et non-pharmacologiques d'optimisation de l'hémodynamie…

ÉMERGENCE / POST-OPÉRATOIRE

- **Analgésie** locorégionale et non-opioïde optimisée
- **Ventilation** optimale malgré analgésie
- **Euvolémie** et stabilité hémodynamique
- Sevrage/poursuite **vasodilatateurs pulmonaires**
- Surveillance/Prévention des complications :
  - Défaillance droite
  - Embolie pulmonaire

ET L'INSTABILITÉ…

APPROCHE / PRISE EN CHARGE

- Influencées par le monitoring et l'étiologie suspectée.
- Le rétablissement de la pression de perfusion systémique est prioritaire en attente d'une prise en charge étiologique

Perioperative Risk and Management in Patients With Pulmonary Hypertension

Omar A. Minai, MD, FCCP; Jean-Pierre Yared, MD; Roop Kaw, MD; Kathirvel Subramaniam, MD; and Nicholas S. Hill, MD, FCCP

CHEST / 144 / 1 / JULY 2013

Systemic Hypotension

Hemodynamic Characteristics	CO	↔ or ↑	↓	↓	↓
	CVP	↔	↓↓	↑	↑
	PCWP	↔ or ↓	↓	↓	↔
	PAP	↔ or ↑	↓	↔ or ↑	↓
Etiology		↓ SVR	↓ RV preload	↑ PVR ↑ RV afterload	↓ RV contractibility
Management		– Treat cause of ↓ SVR eg. sepsis – Combination inotropics, vasopressors, and pulmonary vasodilators – Cautious volume infusion	– Volume infusion	– Correct cause of ↑ PVR eg. hypoxia, acidosis, etc. – Pulmonary vasodilators	– Treat cause eg. RV infarction – Pulmonary vasodilators – Inotropic agents

VASOPRESSEURS

- Agents pharmacologiques centraux
  - Rapides et efficaces
  - Contribuent à éviter l'hypervolémie
  - Complémentaires à certains inotropes ou vasodilatateurs
  - Meilleurs choix : **noradrénaline** et **vasopressine**

Kwak et al. (2002) Anaesthesia
Jeon et al. (2006) Eur J Cardio-Thor Surg

INOTROPES

- **Dobutamine**
  - Profil favorable jusqu'à 10 ug/kg/min
  - Tachycardie, hypotension
- **Milrinone**
  - Profil inodilatateur
  - Tachycardie légère, hypotension*
- **Adrénaline**
  - Tachycardie, arythmies

Pagnamenta et al. (2003) Crit Care Med
Kerbaul et al. (2004) Crit Care
Jeon et al. (2006) Eur J Cardio-Thor Surg

VASODILATATEURS PULMONAIRES

- Voie systémique
  - Altération de la RVS
    - Diminution de la vasoconstriction hypoxique (RVP vs PaO2)
- **Voie inhalée**
  - Altération sélective de la RVP « sans » altération de la RVS
  - Moins d'altération V/Q

NO INHALÉ

- Réduit RVP et améliore débit cardiaque en HTAP, ischémie VD et post-opératoire
  - Combinaison bénéfique avec prostanoïdes et iPDE-5
- Utilisation limitée par la disponibilité, le coût et les effets secondaires
  - HTAP rebond à l'arrêt
  - Toxicité

Goldstein et al. (2012) Thrombosis J
Wang et al. (2003) Can J Anesth

PRÉCURSEURS DU NO

- **Nitroglycérine inhalé**
  - Métabolisme en NO et effet vasodilatateur local
  - Effet bénéfique démontré chez l'adulte et l'enfant
- Nitroprussiate inhalé
- Citrulline

Yurtseven et al. (2003) Anesthesiology
Goyal et al. (2006) British J Anesth
Fattouch (2005) J Card Surg
Barr et al. (2007) J Thor Cardiovasc Surg

PROSTANOÏDES

- **Epoprostenol** (Flolan)
  - Tx médical à long terme
  - Périopératoire : efficace, sécuritaire, simple et peu dispendieux
    - Voie inhalée à favoriser
      - Efficacité équivalente au NO et effet additif NO + epoprostenol inhalé
  - Altération plaquettaire
  - Précautions d'entreposage et d'administration

Haraldsson et al (2000) Intensive Care Med
Khan et al. (2009) J Thor Cardiovasc Surg
Della Rocca et al. (2001) J Cardiothor Vasc Anesth
De Wet et al. (2004) J Thor Cardiovasc Surg

INHIBITEURS PDE

- **Sildenafil** oral
  - Prévention ou traitement de l'HTAP rebond
  - Vasodilatateur pulmonaire
  - Instabilité hémodynamique possible via diminution RVS
- **Milrinone** inhalée
  - Effet pulmonaire sans atteinte de la TAM ou RVS
  - Moins de mismatch V/Q, meilleure PaO2/FiO2

Sablotzki et al. (2005) Can J Anesth
Wang et al. (2009) Adv Ther
Namachivayam et al. (2006) AJRCCM
Shim et al. (2006) J Thor Cardiovasc Surg
Lamarche et al. (2007) Eur J Cardiothor Surg

RETOUR SUR LE CAS CLINIQUE

- Hypertension pulmonaire sévère = morbi-mortalité importante
    - Priorité des considérations anesthésiques
    - Autant que possible, évaluer et optimiser l'hémodynamie en préopératoire.
    - Canule artérielle, voie centrale, cathéter artériel pulmonaire, ETT
    - Volume/produits sanguins, vasopresseurs, inotropes, vasodilatateurs pulmonaires

REMERCIEMENTS

- Dr. André Y. Denault, ICM
- Dr. Tian You An, CHUM
- Dr. Alain Gauthier, CHUM
- Équipe d'inhalothérapeutes du CHUM - HND

RÉFÉRENCES

- **Articles de revue**
- Minai, O. A., Yared, J.-P., Kaw, R., Subramaniam, K., & Hill, N. S. (2013). Perioperative risk and management in patients with pulmonary hypertension. CHEST Journal, 144(1), 329–340.
- Denault, A. Y., Haddad, F., Jacobsohn, E., & Deschamps, A. (2013). Perioperative right ventricular dysfunction. Current Opinion in Anaesthesiology, 26(1), 71–81.
- Price, L. C., Wort, S. J., Finney, S. J., Marino, P. S., & Brett, S. J. (2010). Pulmonary vascular and right ventricular dysfunction in adult critical care: current and emerging options for management: a systematic literature review. Critical Care, 14(5), R169
- Gordon, C., Collard, C. D., & Pan, W. (2010). Intraoperative management of pulmonary hypertension and associated right heart failure. Current Opinion in Anaesthesiology, 23(1), 49–56.
- Denault, A., Deschamps, A., Tardif, J.-C., Lambert, J., & Perrault, L. (2010). Pulmonary hypertension in cardiac surgery. Current Cardiology Reviews, 6(1), 1–14.
- Chin, K. M., & Rubin, L. J. (2008). Pulmonary arterial hypertension. Journal of the American College of Cardiology, 51(16), 1527–1538.

- McLaughlin, V. V., Archer, S. L., Badesch, D. B., Barst, R. J., Farber, H. W., Lindner, J. R., et al. (2009). ACCF/AHA 2009 expert consensus document on pulmonary hypertension a report of the American College of Cardiology Foundation Task Force on Expert Consensus Documents and the American Heart Association developed in collaboration with the American College of Chest Physicians; American Thoracic Society, Inc.; and the Pulmonary Hypertension Association. (Vol. 53, pp. 1573–1619). Presented at the Journal of the American College of Cardiology.

- Galiè, N., Hoeper, M. M., Humbert, M., Torbicki, A., Vachiery, J.-L., Barbera, J. A., et al. (2009, October). Guidelines for the diagnosis and treatment of pulmonary hypertension: the Task Force for the Diagnosis and Treatment of Pulmonary Hypertension of the European Society of Cardiology (ESC) and the European Respiratory Society (ERS), endorsed by the International Society of Heart and Lung Transplantation (ISHLT). European Heart Journal.

- Meyer, S., McLaughlin, V. V., Seyfarth, H.-J., Bull, T. M., Vizza, C. D., Gomberg-Maitland, M., et al. (2013). Outcomes of noncardiac, nonobstetric surgery in patients with PAH: an international prospective survey. European Respiratory Journal, 41(6), 1302–1307.

- Strumpher, J., & Jacobsohn, E. (2011). Pulmonary hypertension and right ventricular dysfunction: physiology and perioperative management. Journal of Cardiothoracic and Vascular Anesthesia, 25(4), 687–704.

- Hill, N. S., Roberts, K. R., & Preston, I. R. (2009). Postoperative pulmonary hypertension: etiology and treatment of a dangerous complication. Respiratory Care, 54(7), 958–968

- **Vasopresseurs et inotropes**

- Kwak, Y. L., Lee, C. S., Park, Y. H., & Hong, Y. W. (2002). The effect of phenylephrine and norepinephrine in patients with chronic pulmonary hypertension. Anaesthesia, 57(1), 9–14
- Price, L. C., Forrest, P., Sodhi, V., Adamson, D. L., Nelson-Piercy, C., Lucey, M., & Howard, L. S. (2007). Use of vasopressin after Caesarean section in idiopathic pulmonary arterial hypertension. British Journal of Anaesthesia, 99(4), 552–555
- Pagnamenta, A., Fesler, P., Vandinivit, A., Brimioulle, S., & Naeije, R. (2003). Pulmonary vascular effects of dobutamine in experimental pulmonary hypertension. Critical Care Medicine, 31(4), 1140–1146.
- Kerbaul, F., Rondelet, B., Motte, S., & Fesler, P. (2004). Effects of norepinephrine and dobutamine on pressure load-induced right ventricular failure*. Critical Care.
- Jeon, Y., Ryu, J. H., Lim, Y. J., Kim, C. S., Bahk, J.-H., Yoon, S. Z., & Choi, J. Y. (2006). Comparative hemodynamic effects of vasopressin and norepinephrine after milrinone-induced hypotension in off-pump coronary artery bypass surgical patients. European Journal of Cardio-Thoracic Surgery : Official Journal of the European Association for Cardio-Thoracic Surgery, 29(6),

- **NO inhalé , Nitroglycérine, Nitroprussiate**

- Goldstein, B., Baldassarre, J., & Young, J. N. (2012). Effects of inhaled nitric oxide on hemostasis in healthy adults treated with heparin: a randomized, controlled, blinded crossover study. Thrombosis Journal, 10(1), 1
- Wang, T., Kebir, El, D., & Blaise, G. (2003). Inhaled nitric oxide in 2003: a review of its mechanisms of action. Canadian Journal of Anaesthesia, 50(8), 839–846.

- Yurtseven, N., Karaca, P., Kaplan, M., Ozkul, V., Tuygun, A. K., Aksoy, T., et al. (2003). Effect of Nitroglycerin Inhalation on Patients with Pulmonary Hypertension Undergoing Mitral Valve Replacement Surgery. Anesthesiology, 99(4), 855.
- Goyal, P., Kiran, U., Chauhan, S., Juneja, R., & Choudhary, M. (2006). Efficacy of nitroglycerin inhalation in reducing pulmonary arterial hypertension in children with congenital heart disease. British Journal of Anaesthesia, 97(2), 208–214.
- **Prostanoïdes**
- Rocca, G. D., Coccia, C., Pompei, L., & Ruberto, F. (2001). Hemodynamic and oxygenation changes of combined therapy with inhaled nitric oxide and inhaled aerosolized prostacyclin. Journal of Cardiothoracic ….
- Antoniou, T., Koletsis, E. N., Prokakis, C., Rellia, P., Thanopoulos, A., Theodoraki, K., et al. (2013). Hemodynamic effects of combination therapy with inhaled nitric oxide and iloprost in patients with pulmonary hypertension and right ventricular dysfunction after high-risk cardiac surgery. Journal of Cardiothoracic and Vascular Anesthesia, 27(3), 459–466.
- Winterhalter, M., Simon, A., Fischer, S., Rahe-Meyer, N., Chamtzidou, N., Hecker, H., et al. (2008). Comparison of Inhaled Iloprost and Nitric Oxide in Patients With Pulmonary Hypertension During Weaning From Cardiopulmonary Bypass in Cardiac Surgery: A Prospective Randomized Trial. Journal of Cardiothoracic and Vascular Anesthesia, 22(3), 406–413.
- Hoeper, M. M., & Olschewski, H. (2000). A comparison of the acute hemodynamic effects of inhaled nitric oxide and aerosolized iloprost in primary pulmonary hypertension. Journal of the …

- De Wet, C. J., Affleck, D. G., Jacobsohn, E., Avidan, M. S., Tymkew, H., Hill, L. L., et al. (2004). Inhaled prostacyclin is safe, effective, and affordable in patients with pulmonary hypertension, right heart dysfunction, and refractory hypoxemia after cardiothoracic surgery. The Journal of Thoracic and Cardiovascular Surgery, 127(4), 1058–1067.
- Khan, T. A., Schnickel, G., Ross, D., Bastani, S., Laks, H., Esmailian, F., et al. (2009). A prospective, randomized, crossover pilot study of inhaled nitric oxide versus inhaled prostacyclin in heart transplant and lung transplant recipients. The Journal of Thoracic and Cardiovascular Surgery, 138(6), 1417–1424.
- Haraldsson, A., Kieler-Jensen, N., Wadenvik, H., & Ricksten, S. E. (2000). Inhaled prostacyclin and platelet function after cardiac surgery and cardiopulmonary bypass. Intensive Care Medicine, 26(2), 188–194.
- **iPDE**
- Sablotzki, A., Starzmann, W., Scheubel, R., Grond, S., & Czeslick, E. G. (2005). Selective pulmonary vasodilation with inhaled aerosolized milrinone in heart transplant candidates. Canadian Journal of Anesthesia 52(10), 1076–1082.
- Wang, H., Gong, M., Zhou, B., & Dai, A. (2009). Comparison of inhaled and intravenous milrinone in patients with pulmonary hypertension undergoing mitral valve surgery. Advances in Therapy.
- Namachivayam, P., Theilen, U., Butt, W. W., Cooper, S. M., Penny, D. J., & Shekerdemian, L. S. (2006). Sildenafil prevents rebound pulmonary hypertension after withdrawal of nitric oxide in children. American Journal of Respiratory and Critical Care Medicine, 174(9), 1042–1047.
- Shim, J. K., Choi, Y. S., Oh, Y. J., Kim, D. H., Hong, Y. W., & Kwak, Y. L. (2006). Effect of oral sildenafil citrate on intraoperative hemodynamics in patients with pulmonary hypertension undergoing valvular heart surgery. The Journal of Thoracic and Cardiovascular Surgery, 132(6), 1420–1425.
- Lamarche, Y., Perrault, L. P., Maltais, S., Tétreault, K., Lambert, J., & Denault, A. Y. (2007). Preliminary experience with inhaled milrinone in cardiac surgery. European Journal of Cardio-Thoracic Surgery : Official Journal of the European Association for Cardio-Thoracic Surgery, 31(6), 1081–1087.

- **Autres**

  - Côté, G., & Denault, A. (2008). Transesophageal echocardiography-related complications. Canadian Journal of Anaesthesia = Journal Canadien D'anesthésie, 55(9), 622–647.

- Boyd, K. D., Thomas, S. J., & Gold, J. (1983). A prospective study of complications of pulmonary artery catheterizations in 500 consecutive patients. CHEST Journal.

CLASSIFICATION

Table 4 Updated clinical classification of pulmonary hypertension (Dana Point, 2008[1])

1 Pulmonary arterial hypertension (PAH)
- 1.1 Idiopathic
- 1.2 Heritable
  - 1.2.1 BMPR2
  - 1.2.2 ALK1, endoglin (with or without hereditary haemorrhagic telangiectasia)
  - 1.2.3 Unknown
- 1.3 Drugs and toxins induced
- 1.4 Associated with (APAH)
  - 1.4.1 Connective tissue diseases
  - 1.4.2 HIV infection
  - 1.4.3 Portal hypertension
  - 1.4.4 Congenital heart disease
  - 1.4.5 Schistosomiasis
  - 1.4.6 Chronic haemolytic anaemia
- 1.5 Persistent pulmonary hypertension of the newborn

1′ Pulmonary veno-occlusive disease and/or pulmonary capillary haemangiomatosis

2 Pulmonary hypertension due to left heart disease
- 2.1 Systolic dysfunction
- 2.2 Diastolic dysfunction
- 2.3 Valvular disease

3 Pulmonary hypertension due to lung diseases and/or hypoxia
- 3.1 Chronic obstructive pulmonary disease
- 3.2 Interstitial lung disease
- 3.3 Other pulmonary diseases with mixed restrictive and obstructive pattern
- 3.4 Sleep-disordered breathing
- 3.5 Alveolar hypoventilation disorders
- 3.6 Chronic exposure to high altitude
- 3.7 Developmental abnormalities

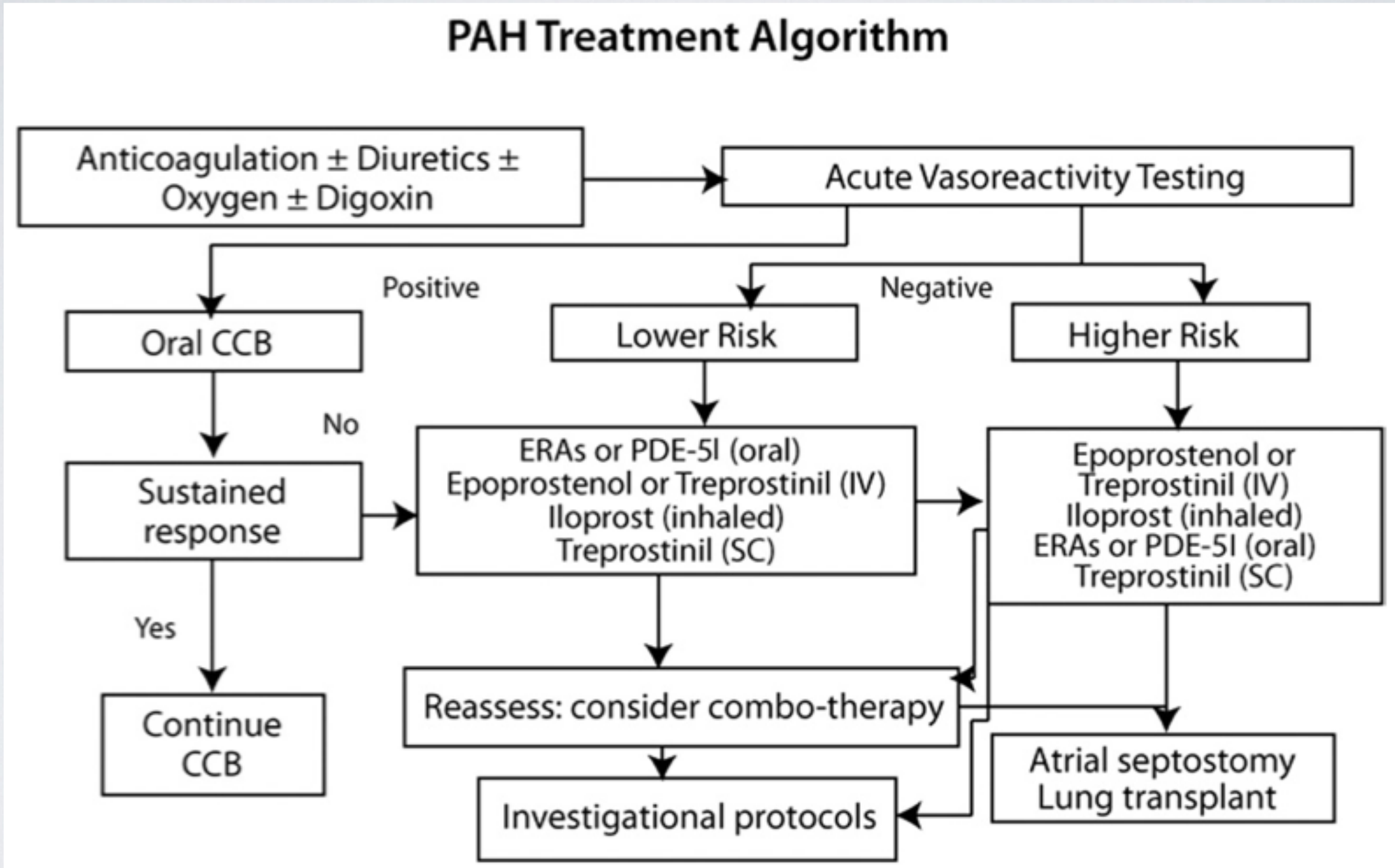
4 Chronic thromboembolic pulmonary hypertension

5 PH with unclear and/or multifactorial mechanisms
- 5.1 Haematological disorders: myeloproliferative disorders, splenectomy.
- 5.2 Systemic disorders: sarcoidosis, pulmonary Langerhans cell histiocytosis, lymphangioleiomyomatosis, neurofibromatosis, vasculitis
- 5.3 Metabolic disorders: glycogen storage disease, Gaucher disease, thyroid disorders
- 5.4 Others: tumoural obstruction, fibrosing mediastinitis, chronic renal failure on dialysis

ALK-1 = activin receptor-like kinase 1 gene; APAH = associated pulmonary arterial hypertension; BMPR2 = bone morphogenetic protein receptor, type 2; HIV = human immunodeficiency virus; PAH = pulmonary arterial hypertension.

European Heart Journal (2009) **30**, 2493–2537
doi:10.1093/eurheartj/ehp297

PRISE EN CHARGE MÉDICALE



American Heart Association. JACC Vol. 53, No. 17, 2009
April 28, 2009:1573–619

Université de Montréal

PRONOSTIC MÉDICAL

Table 2. PAH*: Determinants of Prognosis

Determinants of Risk	Higher Risk (Poor Prognosis)
Clinical evidence of RV failure	Yes
Progression of symptoms	Rapid
WHO class†	IV
6MW distance‡	Shorter (less than 300 m)
CPET	Peak VO_2 less than 10.4 mL/kg/min
Echocardiography	Pericardial effusion, significant RV enlargement/dysfunction, right atrial enlargement
Hemodynamics	RAP greater than 20 mm Hg, CI less than 2.0 L/min/m^2
BNP§	Significantly elevated

American Heart Association. JACC Vol. 53, No. 17, 2009
April 28, 2009:1573–619

ET LA CHIRURGIE CARDIAQUE…

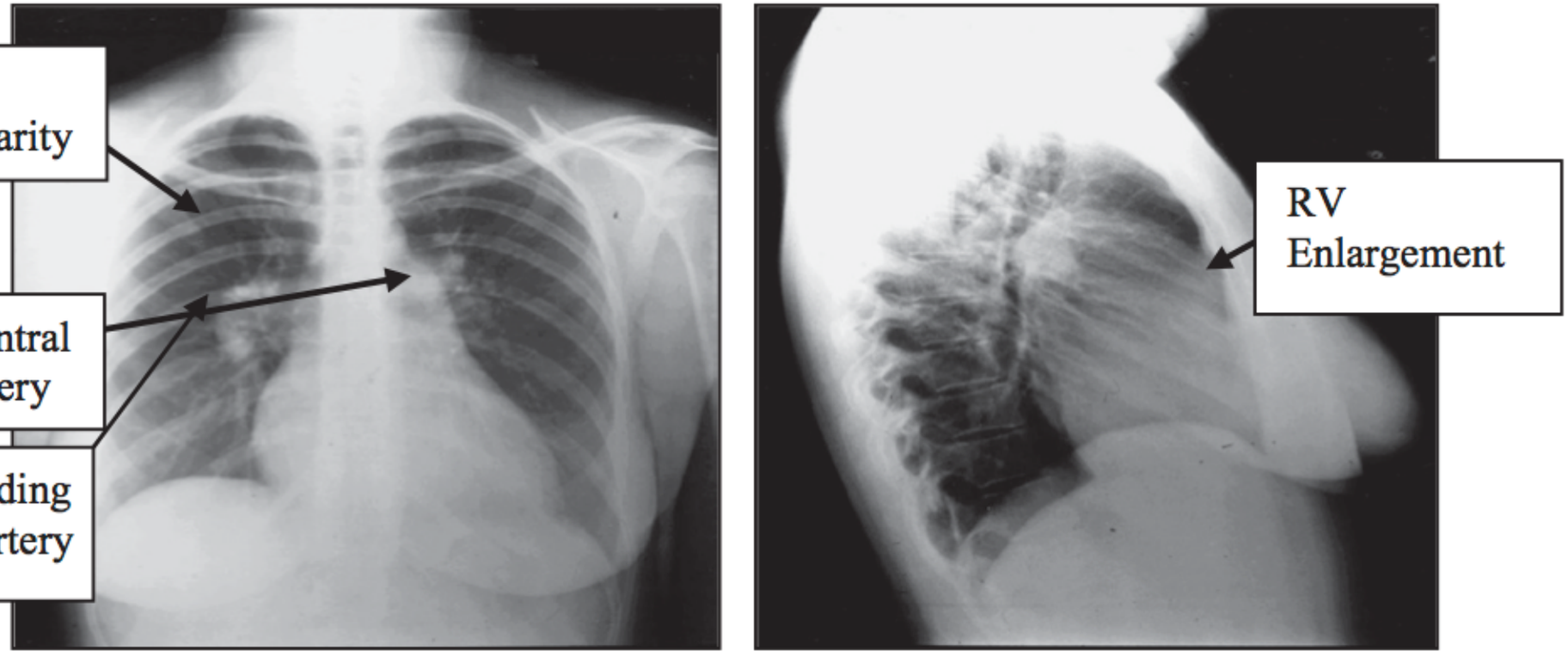
- Mortalité jusqu'à 25%
- Prédicteurs de morbi-mortalité :
  - PAPs > 60 mmHg, PAPm > 30 mmHg
  - TAM / PAPm < 4
- HTAP responsable de 19% de la mortalité et de 50% des complications en transplantation cardiaque

Table 4. Features of the Physical Examination Pertinent to the Evaluation of PH

Sign	Implication
Physical Signs That Reflect Severity of PH	
Accentuated pulmonary component of S_2 (audible at apex in over 90%)	High pulmonary pressure increases force of pulmonic valve closure
Early systolic click	Sudden interruption of opening of pulmonary valve into high-pressure artery
Midsystolic ejection murmur	Turbulent transvalvular pulmonary outflow
Left parasternal lift	High right ventricular pressure and hypertrophy present
Right ventricular S_4 (in 38%)	High right ventricular pressure and hypertrophy present
Increased jugular "a" wave	Poor right ventricular compliance
Physical Signs That Suggest Moderate to Severe PH	
Moderate to severe PH	
Holosystolic murmur that increases with inspiration	Tricuspid regurgitation
Increased jugular v waves	
Pulsatile liver	
Diastolic murmur	Pulmonary regurgitation
Hepatojugular reflux	High central venous pressure
Advanced PH with right ventricular failure	
Right ventricular S_3 (in 23%)	Right ventricular dysfunction
Distention of jugular veins	Right ventricular dysfunction or tricuspid regurgitation or both
Hepatomegaly	Right ventricular dysfunction or tricuspid regurgitation or both
Peripheral edema (in 32%)	
Ascites	
Low blood pressure, diminished pulse pressure, cool extremities	Reduced cardiac output, peripheral vasoconstriction
Physical Signs That Suggest Possible Underlying Cause or Associations of PH	
Central cyanosis	Abnormal V/Q, intra-pulmonary shunt, hypoxemia, pulmonary-to-systemic shunt
Clubbing	Congenital heart disease, pulmonary venopathy
Cardiac auscultatory findings, including systolic murmurs, diastolic murmurs, opening snap, and gallop	Congenital or acquired heart or valvular disease
Rales, dullness, or decreased breath sounds	Pulmonary congestion or effusion or both
Fine rales, accessory muscle use, wheezing, protracted expiration, productive cough	Pulmonary parenchymal disease
Obesity, kyphoscoliosis, enlarged tonsils	Possible substrate for disordered ventilation
Sclerodactyly, arthritis, telangiectasia, Raynaud phenomenon, rash	Connective tissue disorder
Peripheral venous insufficiency or obstruction	Possible venous thrombosis
Venous stasis ulcers	Possible sickle cell disease
Pulmonary vascular bruits	Chronic thromboembolic PH
Splenomegaly, spider angiomata, palmary erythema, icterus, caput medusa, ascites	Portal hypertension

JACC Vol. 53, No. 17, 2009
April 28, 2009:1573–619
American Heart Association®

A



B

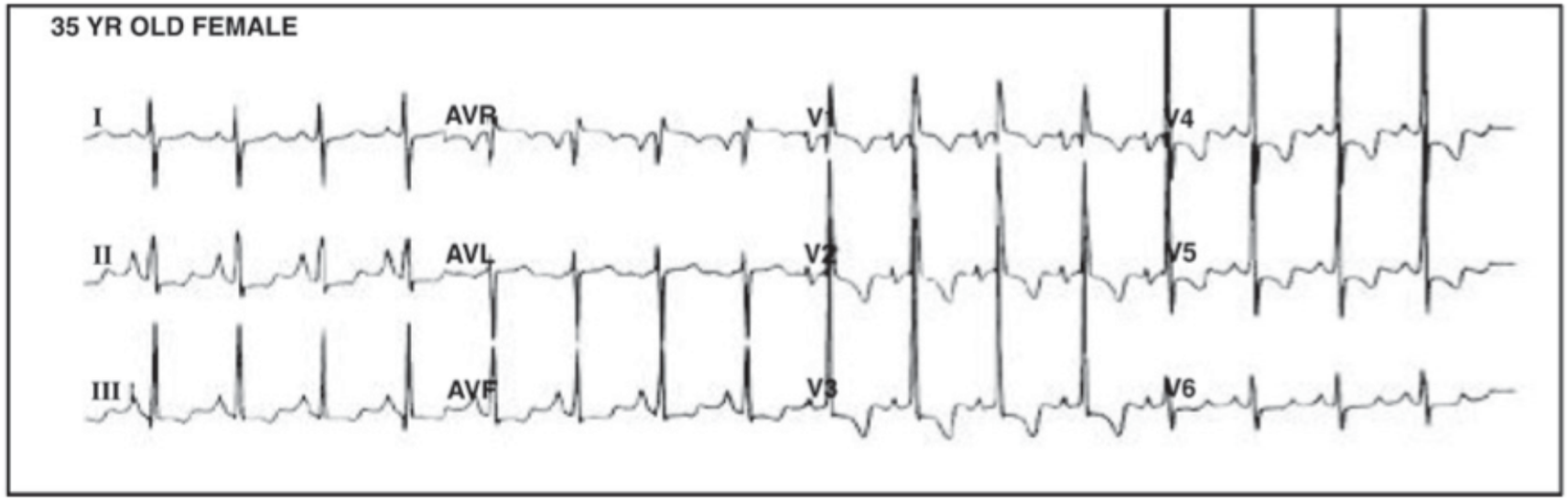
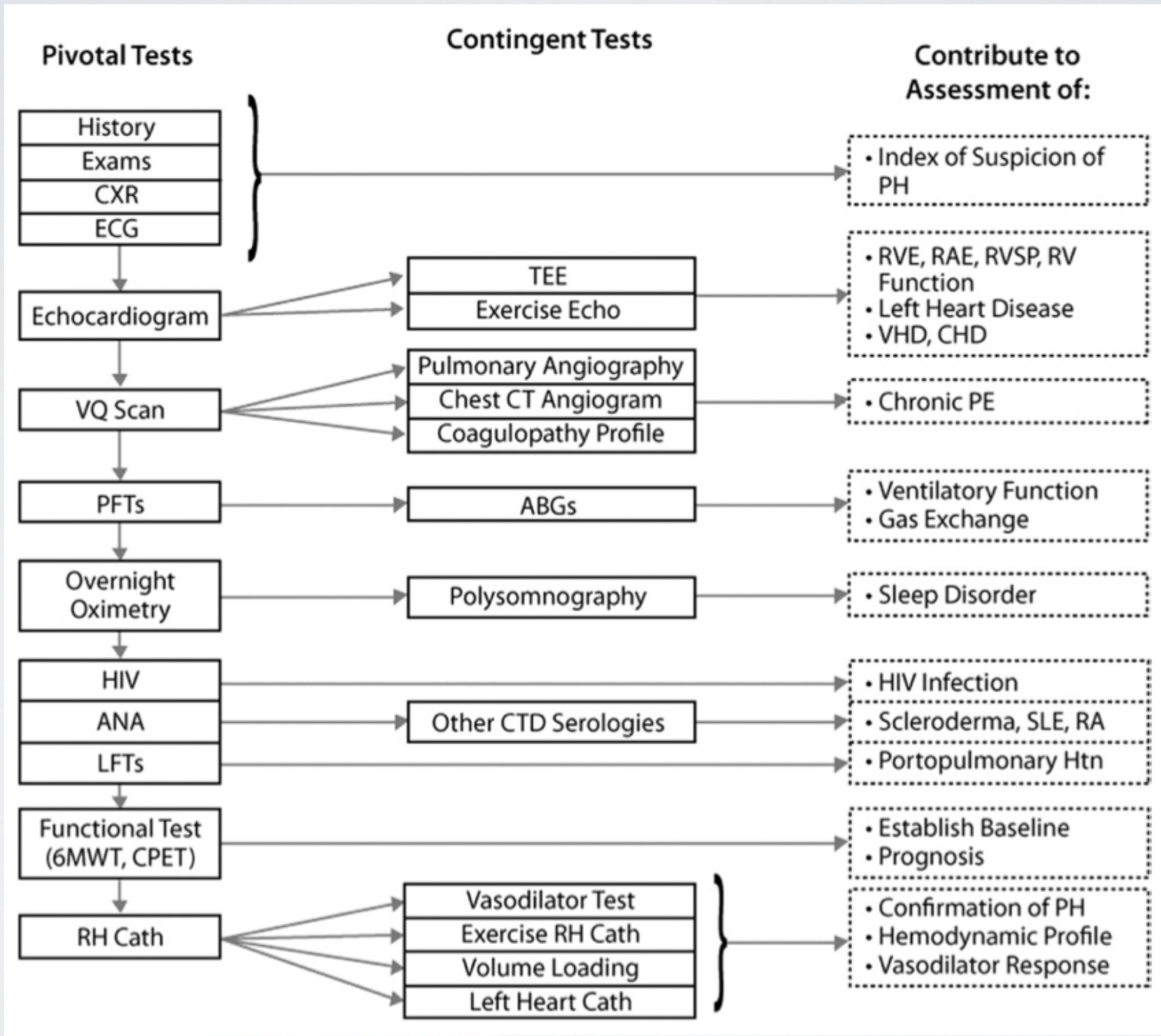


Figure 4. Chest X-Ray and ECG in PAH

Postero-anterior and lateral chest X-ray (top) shows decreased peripheral lung vascular markings, hilar pulmonary artery prominence, and right ventricular enlargement of a patient with idiopathic PAH. ECG (bottom) of the same patient reveals right atrial enlargement, right ventricular hypertrophy and strain, and right axis deviation of the QRS complex. ECG indicates electrocardiogram; and PAH, pulmonary arterial hypertension.

American Heart Association. JACC Vol. 53, No. 17, 2009
April 28, 2009:1573–619

BILAN HYPERTENSION PULMONAIRE



American Heart Association. JACC Vol. 53, No. 17, 2009
April 28, 2009:1573–619

Université de Montréal

ÉPREUVE DE VASORÉACTIVITÉ

Table 7. Agents for Acute Vasodilator Testing

	Epoprostenol	Adenosine	Nitric Oxide
Route of Administration	Intravenous infusion	Intravenous infusion	Inhaled
Dose Titration	2 ng/kg/min every 10 to 15 min	50 mcg/kg/min every 2 min	None
Dose Range	2 to 10 ng/kg/min	50 to 250 mcg/kg/min	10 to 80 ppm
Side Effects	Headache, nausea, lightheadedness	Dyspnea, chest pain, AV block	Increased left heart filling pressure in susceptible patients

American Heart Association. JACC Vol. 53, No. 17, 2009
April 28, 2009:1573–619

TABLE 3 Univariate analysis of risk factors for major complications after surgery in patients with pulmonary arterial hypertension

	OR (95% CI)	p-value
Age years	0.9 (0.9–1.0)	0.52
6MWD m	2.2 (1.1–3.7)	0.04
NYHA functional class	1.04 (0.5–2.0)	0.72
mRAP mmHg	1.1 (1.0–1.3)	0.01
Cardiac index $L \cdot min^{-1} \cdot m^{-2}$	1.6 (0.92–4.9)	0.36
mPAP mmHg	0.97 (0.8–1.1)	0.21
PVR $dyn \cdot s \cdot cm^{-5}$	0.91 (0.78–1.1)	0.22
SvO_2 %	1.2 (0.9–1.3)	0.07
Surgery performed in PH centre	0.2 (0.05–1.0)	0.06
General *versus* spinal anaesthesia	1.9 (0.3–2.7)	0.50
Use of vasopressors	1.5 (1.2–2.7)	0.03
Emergency procedure	2.4 (1.4–3.6)	0.01

6MWD: 6-min walking distance; NYHA: New York Heart Association; mRAP: mean right artery pressure; mPAP: mean pulmonary artery pressure; PVR: pulmonary vascular resistance; SvO_2: mixed venous oxygen saturation; PH: pulmonary hypertension.

Eur Respir J 2013; 41: 1302–1307
DOI: 10.1183/09031936.00089212

Table 26-2 Hemodynamic Changes after Induction of Anesthesia with Nonbarbiturate Hypnotics

	Etomidate*	Ketamine	Midazolam	Propofol
HR	–5 ± 10%	0-59%	–14 ± 12%	–10 ± 10%
MBP	0-17%	0 ± 40%	–12-26%	–10-40%
SVR	–10 ± 14%	0 ± 33%	0-20%	–15-25%
PAP	–9 ± 8%	+44 ± 47%	Unchanged	0-10%
PVR	–18 ± 6%	0 ± 33%	Unchanged	0-10%
PAO	Unchanged	Unchanged	0-25%	Unchanged
RAP	Unchanged	+15 ± 33%	Unchanged	0-10%
CI	–20 ± 14%	0 ± 42%	0-25%	–10-30%
SV	0-20%	0-21%	0-18%	–10-25%
LVSWI	0-33%	0 ± 27%	–28-42%	–10-20%
dP/dt	0-18%	Unchanged	0-12%	Decreased

COURBE TVC NORMALE

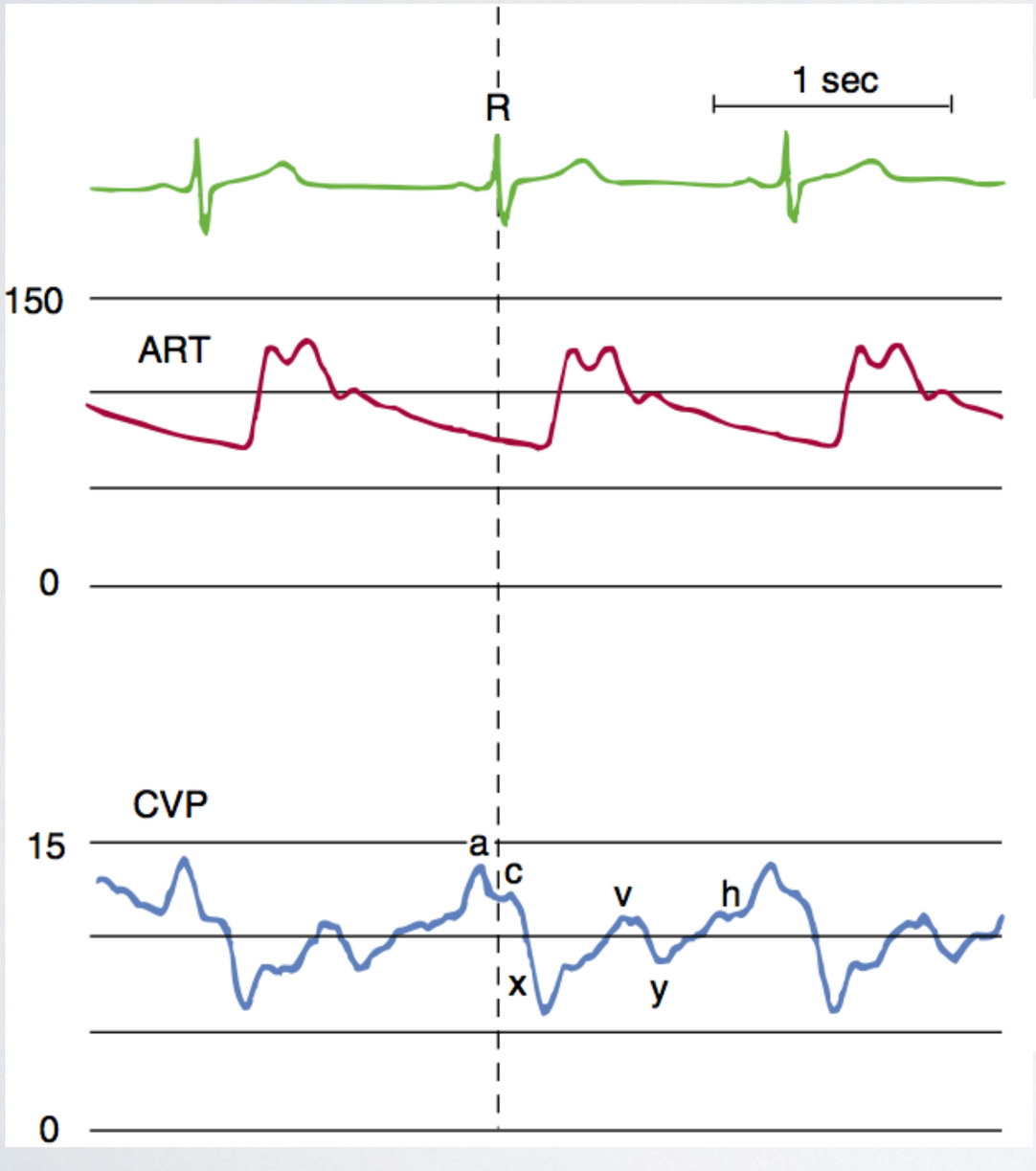


Table 40-3 Central Venous Pressure Waveform Components

Waveform Component	Phase of Cardiac Cycle	Mechanical Event
a wave	End diastole	Atrial contraction
c wave	Early systole	Isovolumic ventricular contraction, tricuspid motion toward the right atrium
v wave	Late systole	Systolic filling of the atrium
h wave	Mid to late diastole	Diastolic plateau
x descent	Mid systole	Atrial relaxation, descent of the base, systolic collapse
y descent	Early diastole	Early ventricular filling, diastolic collapse

COURBE TVC - ANORMAL

FA + régurgitation tricuspidienne

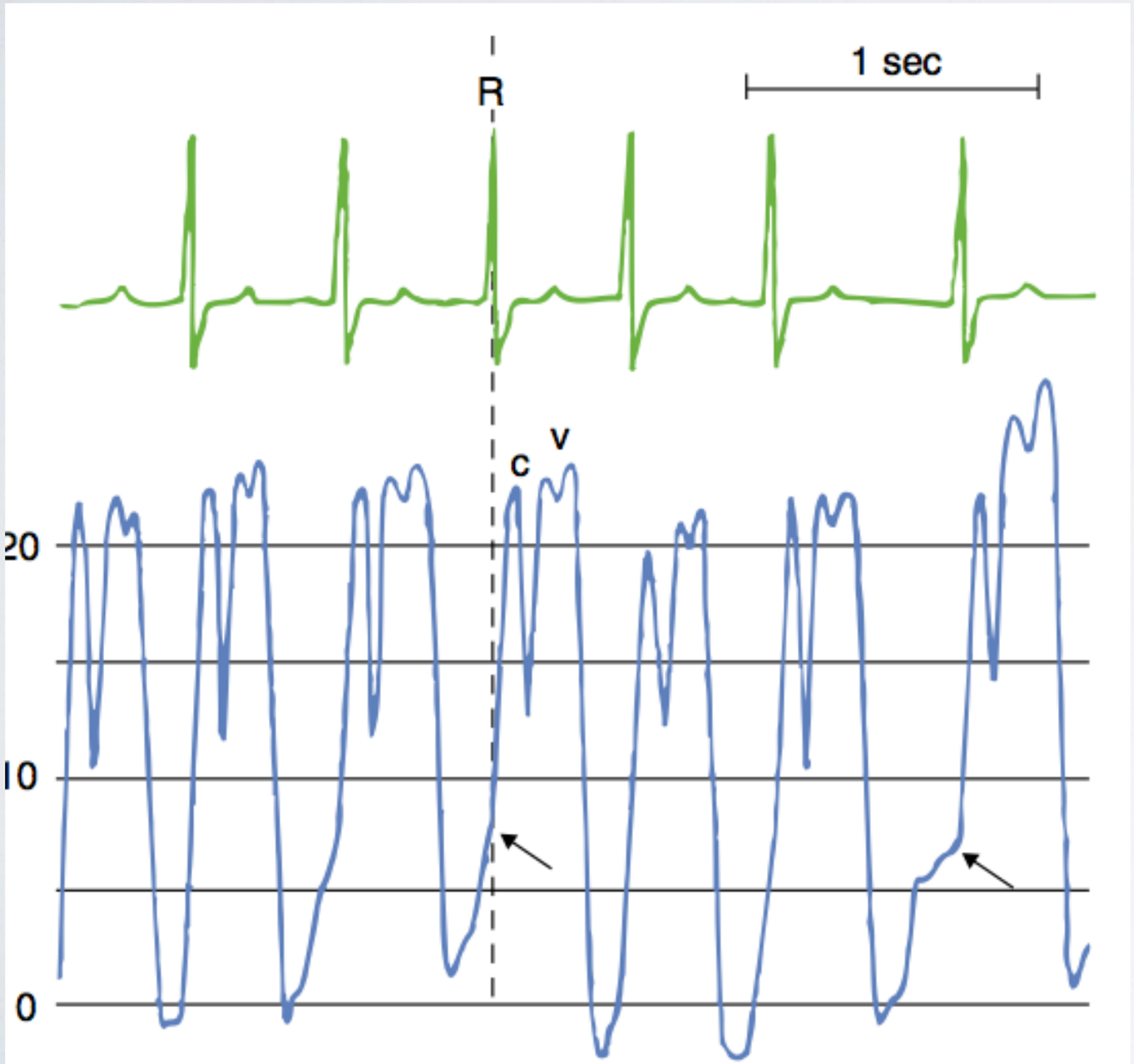


Table 40-4 Central Venous Pressure Waveform Abnormalities

Condition	Characteristics
Atrial fibrillation	Loss of a wave Prominent c wave
Atrioventricular dissociation	Cannon a wave
Tricuspid regurgitation	Tall systolic c-v wave Loss of x descent
Tricuspid stenosis	Tall a wave Attenuation of y descent
Right ventricular ischemia	Tall a and v waves Steep x and y descents M or W configuration
Pericardial constriction	Tall a and v waves Steep x and y descents M or W configuration
Cardiac tamponade	Dominant x descent Attenuated y descent
Respiratory variation during spontaneous or positive-pressure ventilation	Measure pressures at end-expiration

Ronald D. Miller
Miller's Anesthesia
SEVENTH EDITION

Université de Montréal

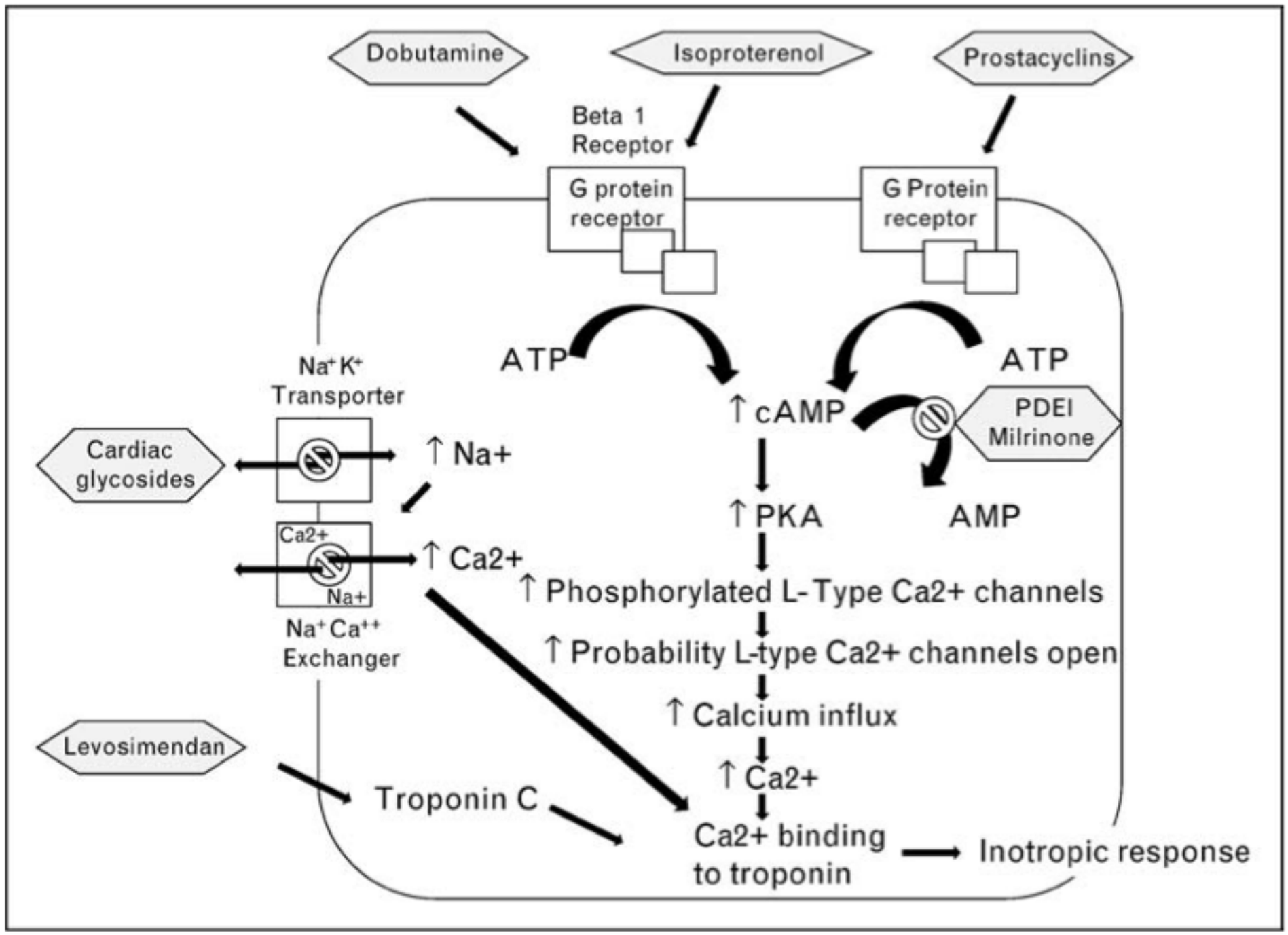
Table 3. Risk Factors for Postoperative Complications in Patients With Preoperative Pulmonary Hypertension

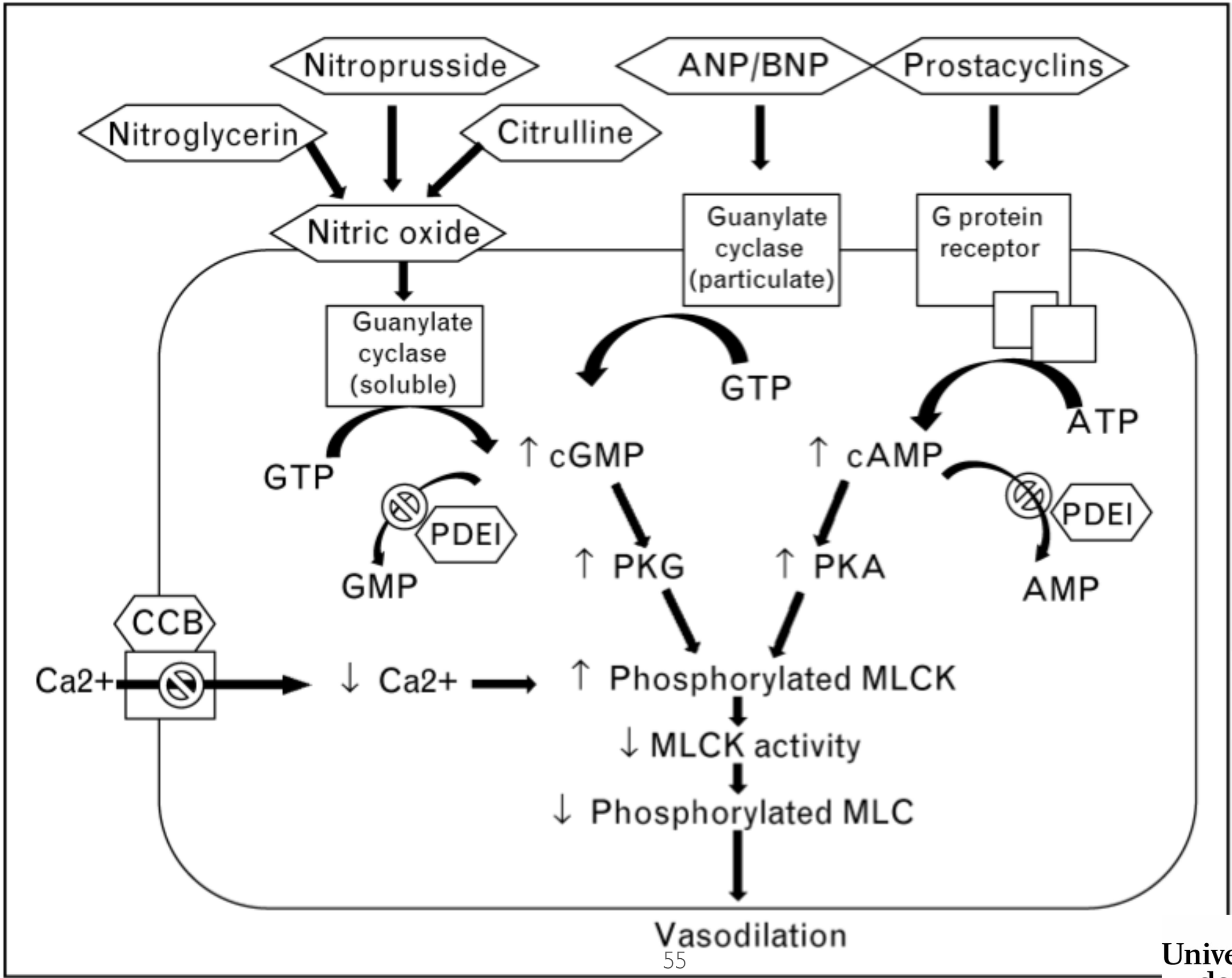
Non-cardiac surgery
High estimated pulmonary arterial systolic pressure (> 70 mm Hg)
Underlying coronary artery disease
Emergency surgery
Intermediate to high risk surgery
New York Heart Association functional class ≥ 2
History of pulmonary embolism
Prolonged anesthesia (> 3 h)
Right-axis deviation or right-ventricular hypertrophy on electrocardiogram
Ratio of right-ventricular to systemic systolic blood pressure > 0.66
Need for vasopressor

Table 4. Basic Principles of Managing Postoperative Pulmonary Hypertension

Optimize fluid balance
Avoid excessive fluid in patient with distended inferior vena cava and right ventricle
Optimize metabolic state
Correct acidosis, hypoxemia, anemia
Treat respiratory failure
Intubated patient
Lung-protective ventilation strategy (tidal volume 6 mL/kg, plateau pressure < 30 cm H_2O)
Optimize oxygenation
Maintain systemic perfusion pressure
Mean systemic blood pressure $>$ mean pulmonary arterial pressure
Pressors if necessary
Optimize cardiac output
Use inotropes to maintain cardiac index > 2 L/min/m^2
Attempt to reduce right-ventricular afterload
Avoid systemic vasodilators
Use pulmonary vasodilators: inhaled vasodilators are less likely to have adverse effects on systemic blood pressure or oxygenation.
Consider combinations

Figure 3 Schematic depicting inotropic agents used for augmenting right heart function in the setting of pulmonary hypertension





Intraoperative management of pulmonary hypertension and associated right heart failure

Claire Gordon[a], Charles D. Collard[a,b] and Wei Pan[a,b]

Table 1 Agents used for the therapeutic management of pulmonary hypertension

Drug	Dose
Intravenous	
Citrulline	150 mg/kg bolus at CPB initiation, with postoperative continuous infusion of 9 mg/kg/h
Epoprostenol	Continuous infusion of 1–50 ng/kg/min
Iloprost	Continuous infusion of 1–5 ng/kg/min
Nesiritide	2 μg/kg bolus followed by continual infusion of 0.01 μg/kg/min
Milrinone	50 μg/kg over 10 min followed by 0.375–0.75 μg/kg/min infusion
Sildenafil	10 mg in 20 ml buffer, administer throughout perioperative period
Inhaled	
Epoprostenol	10–20 μg/ml dilution, continuous nebulization with an oxygen flow of 2–3 l/min
Iloprost	2.5–5.0 μg, 6–9 times a day
Milrinone	60–90 μg/kg bolus followed by 0.08–0.11 μg/kg/min infusion
Nitric oxide	5–80 ppm continuous use
Sildenafil	10 mg in 20 ml buffer, administer throughout perioperative period
Nitroglycerin	2.5 μg/kg over 10 min
Treprostinil	18–54 μg q.i.d.
Subcutaneous	
Treprostinil	Continuous infusion of 1.25 ng/kg/min, titrated by 1.25 ng/kg/min weekly
Oral	
Bosentan	62.5–125 mg b.i.d.
Sildenafil	0.25–0.75 mg/kg every 4–6 h
Vardenafil	5 mg daily, then 5 mg b.i.d. after 4 weeks

vs 2,5 ug/kg/min (Yurtseven et al 2003 Anesthesiology)

Table 6 Agents used to reduce PVR in the ICU setting

Drug	Dose	Half-life (duration of action)	Potential adverse effects
Intravenous			
Prostacyclin (Epoprostenol, Flolan)	Start at 1 ng/kg/min; titrate upward in 2-ng/kg/min increments according to effect	3-5 minutes (10 minutes)	Systemic hypotension, worsening oxygenation (increased V/Q mismatch), antiplatelet effect, headache, flushing, jaw pain, nausea, diarrhea
Iloprost	1-5 ng/kg/min	30 minutes	Similar to Flolan; also syncope (5%)
Sildenafil [325] (NB off-license use in hemodynamically unstable patients)	Low dose, 0.05 mg/kg; high dose, 0.43 mg/kg) (comes as 0.8 mg/ml)	3-5 hours	Hypotension: caution if fluid depleted, severe LV-outflow obstruction, autonomic dysfunction. Hypoxemia due to V/Q mismatch. Common: headache, flushing, diarrhea, epistaxis, tremor. Rare but important: anterior ischemic optic neuropathy
Milrinone	50 μg/kg over 10 minutes followed by 0.375-0.75 μg/kg/min infusion	1-2 hours	Tachyarrhythmias, hypotension
Adenosine	50-350 μg/kg/min, titrate up in 50 μg/kg/min increments	5-10 seconds (2 minutes)	Bradycardia, bronchospasm, chest pain
Inhaled (preferred; Note variable absorption likely)			
Prostacyclin (Epoprostenol, Flolan) [286,303]	0.2-0.3 ml/min of 10-20 μg/ml nebulized into inspiratory limb of ventilator circuit (30-40 ng/kg/min)	3-5 minutes	As above but less hypotension and improved oxygenation compared with intravenous use
Iloprost [275]	2.5-5 μg 6-9 times/day, 1 mg/ml milrinone into the ventilator circuit at 0.2-0.3 ml/min for 10-20 minutes	30 minutes	As above and bronchospasm
Milrinone [176,178,179]	5-80 ppm continuously	1-2 hours	Less systemic hypotension than with IV milrinone
NO		15-30 seconds (5 minutes)	Methemoglobinemia; withdrawal PH
ORAL (rarely in ICU)			
Bosentan	62.5-125 mg b.d.	5 hours	Liver-function test abnormalities; drug interactions; edema
Sildenafil	0.25-0.75 mg/kg 4 hrly	3-4 hours	As above; less hypotension and hypoxemia in stable patients

Laura C Price[1*†], Stephen J Wort[1†], Simon J Finney[1], Philip S Marino[1], Stephen J Brett[2]

Table 5 Pulmonary vascular properties of vasoactive agents

	CI	PVR	SVR	PVR/SVR	Tachycardia
Vasopressors				Dose related	
NE	+	+	++	+/-	+
PHE	-	++	+	+	-
Low-dose AVP	+/-	+/-	++	-	-
Inotropes					
Dobutamine	++	-	-	-	+
<5 μg/kg/min					
Dopamine	+	+/-	+	+	++
Epinephrine	++	-	++	-	++
Inodilators					
PDE IIIs	++	-	-	-	+/-
Levosimendan	++	-	-	-	-

CRITICAL CARE

Pulmonary vascular and right ventricular dysfunction in adult critical care: current and emerging options for management: a systematic literature review

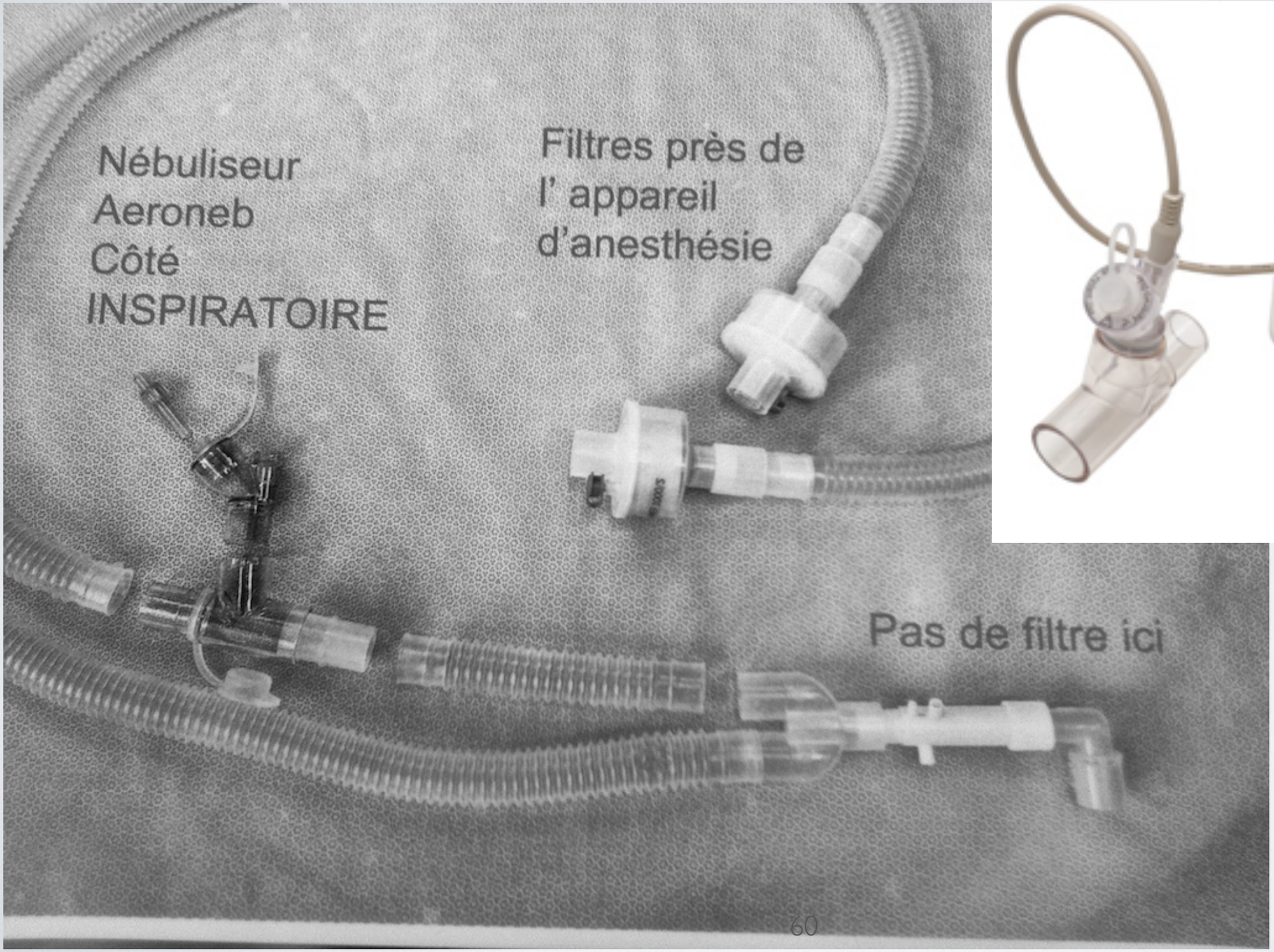
Laura C Price[1*†], Stephen J Wort[1†], Simon J Finney[1], Philip S Marino[1], Stephen J Brett[2]

AUTRES PROSTANOÏDES

- **Iloprost** (Ventavis)
  - Utilisé en périopératoire
  - Comparé à NO : efficacité supérieure mais diminue RVS
  - Combinaison NO + iloprost : possiblement additifs
  - Plus simple d'utilisation que l'epoprostenol

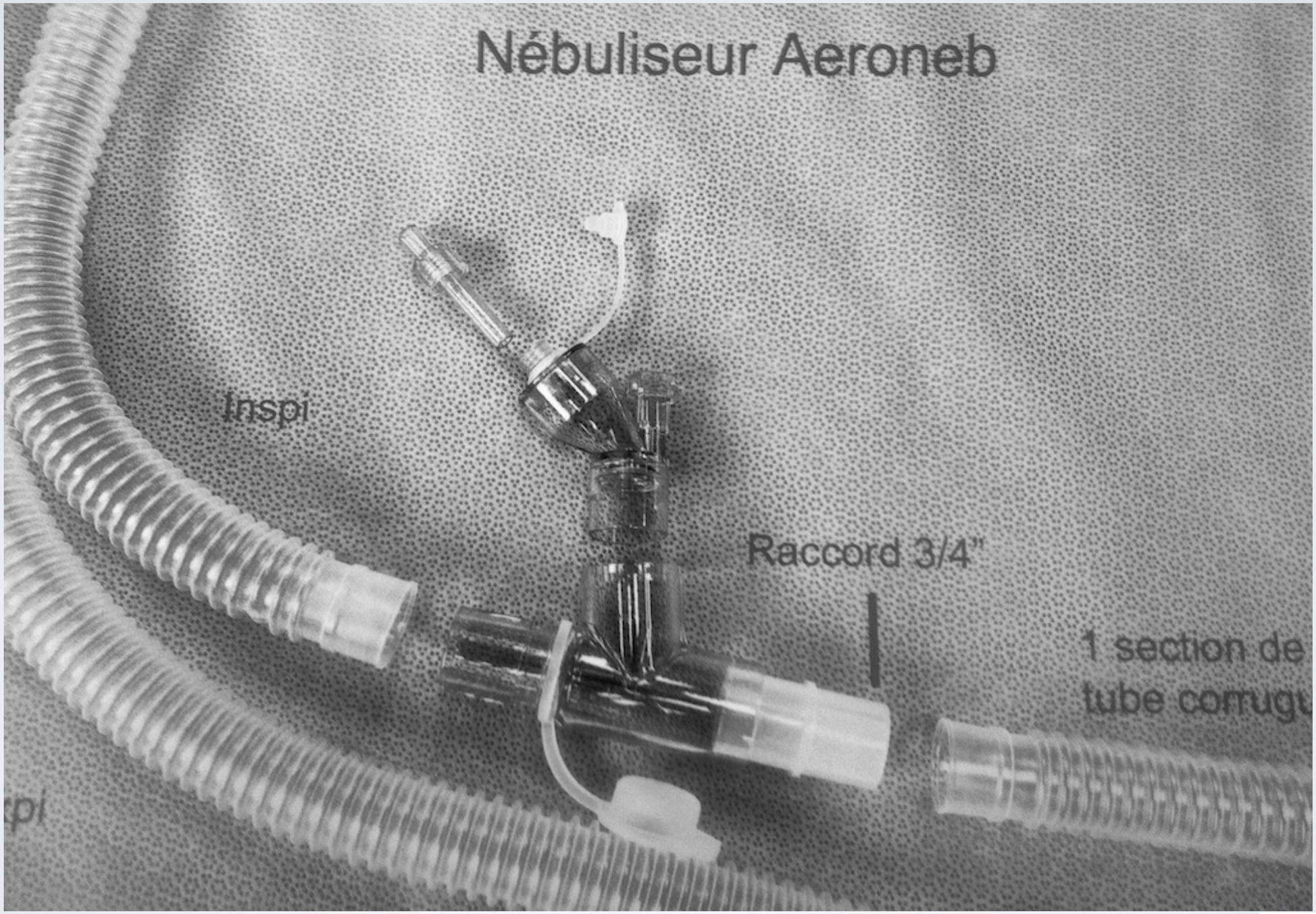
Hoeper et al. (2000) JACC
Winterhalter et al. (2008) J Cardiothor Vasc Anesth
Antoniou et al. (2013) J Cardiothor Vasc Anesth

MONTAGE NÉBULISEUR AERONEB



Université de Montréal

MONTAGE EPOPROSTENOL



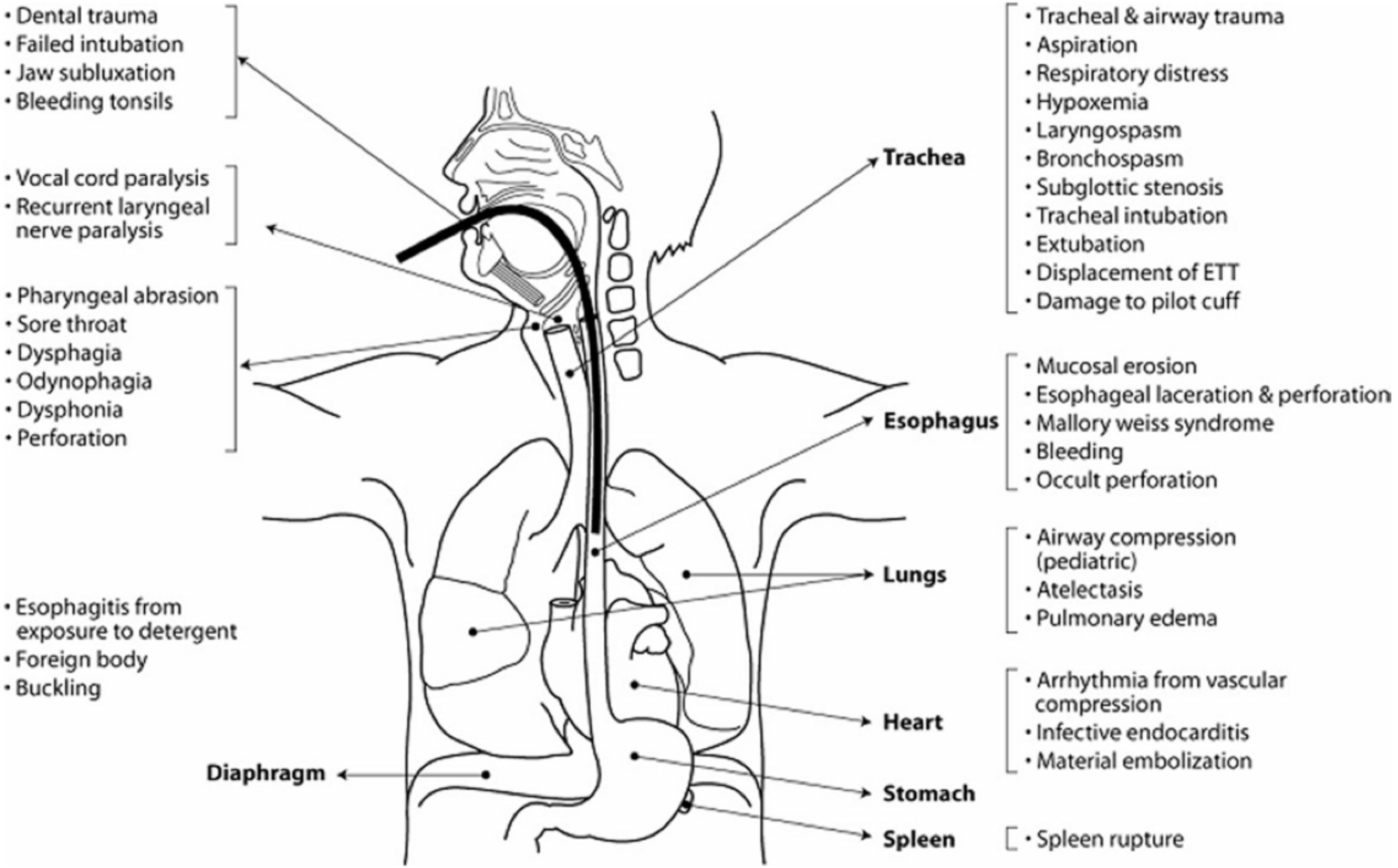


FIGURE 1 Transesophageal echocardiography (TEE)-related complications (ETT = endotracheal tube). With permission from Informa Healthcare (Denault *et al.* 2005).

CAN J ANESTH 2008 / 55: 9 / pp 622–647

TABLE III Contraindications to TEE

1. Absolute contraindications *

Lack of informed consent
Unwilling and uncooperative patient
Lack of expertise in intubation for TEE
Esophageal obstruction (cancer, stricture)
Gastric volvulus
Active upper gastrointestinal bleeding
Perforated viscus (known or suspected)
Full stomach
Suspected neck injury

2. Relative contraindications

2.1 Known esophageal pathology
- Esophageal varices without bleeding
- Esophageal diverticulum
- Transesophageal fistula
- Esophagitis/inflammatory process
- Gastric herniation
- Scleroderma
- Carcinoma
- Penetrating or blunt thoracic esophageal trauma
- History of previous esophageal surgery
- Esophagectomy
- Fundal-plication gastric surgery

2.2 Cervical abnormalities
- Severe cervical arthritis/osteophytes/severe cervical spondylosis
- Neck surgery/radiotherapy in the cervical region
- Severe oropharyngeal distortion

2.3 Miscellaneous
- Prior mediastinal irradiation
- Coagulopathy
- Nasal intubation
- History of nasal/nose surgery
- Septal deviation

CAN J ANESTH 2008 / 55: 9 / pp 622–647

Table 1—*Complications—Swan Ganz Catheters*
(528 Catheterizations)

	Number	Percent
Minor Complications		
Complications with Insertion		
Multiple sticks	27	5.1
Arterial punctures	8	1.5
Hematomas	15	2.8
Inability to cannulate right ventricle	3	0.6
Inability to wedge	11	2.1
Looped catheter	1	0.2
Cardiac Complications		
Atrial premature contractions	7	1.3
Ventricular premature contractions	58	11.0
Infections		
Inflamed puncture site	5	0.9
Positive blood culture (asymptomatic patients)	3	0.6
	138	
Serious Complications (Potentially Life Threatening)		
Ventricular tachycardia	8	1.5
Ventricular fibrillation	0	—
Endocarditis	0	—
Valve rupture	0	—
Septicemia	7	1.3
Pulmonary hemorrhage	1	0.2
Pulmonary infiltrates	7	1.3
	23	