

Présentation
Cours science de base de cardiologie :
**Dissection aortique de type B
pour anesthésiologistes**

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Décembre 2013

Problème

- Ischémie MID chez patient de 65 ans avec dissection aortique type B

Plan

- Définition
- Pathophysiologie
- Classification
- Présentations cliniques
- Approche diagnostique initiale
- Considérations pré opératoires
- Traitement médical initial
- Indications d'interventions

Plan (suite)

- Choix de l'approche
- Prise en charge per opératoire
 - Monitoring/ Installation
 - Changements hémodynamiques
 - Considérations particulières pour TEVAR
 - Complications per opératoire et les méthodes de prévention
- Réponses QCM

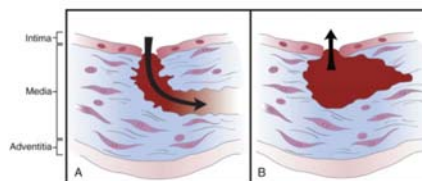
Dissection Aortique

Définition

- Perte de la continuité de la media
- Saignement à l'intérieur de la paroi aortique
- Séparation des couches aortiques

Dissection « classique » 90%

Causes dissection aortique



90% classique

FIGURE 60-66 Hypotheses of acute aortic dissection. **A**, Primary tear in the intima leads to blood entering the media (arrow) and the development of a cleavage plane (dissection) creating the true and false lumen. **B**, Primary rupture of the vasa vasorum leads to hemorrhage in the aortic wall, which then precipitates an intimal disruption (arrow), creating the intimal tear and aortic dissection.

Dissection Aortique

- Rare mais potentiellement catastrophique
- Difficile Dx
- 27 % mortalité intra hospitalière
- ACC/AHA
 - 40 % mort immédiate
 - 1% mortalité par hr
 - 20% mort péri opératoire

Dissection Aortique

Epidémiologie

- IRAD :
 - 464 cas (65% H, 63 ans)
 - mortalité 27.4%
 - Chx (26 %) Vs Rx (58 %) proximal Type A
 - Chx (31%) Vs Rx (10 %) distal Type B
 - Incidence 2,5-3 cas par 100000
 - 6000-10000 N cas par année

Dissection Aortique

Epidémiologie

- 75% mortalité à 2 sem sans traitement Type A
- 75% survie si traitement efficace à 5 ans
- 40-60% survie à 10 ans pour traitement Chx
- 90% survie type B avec Rx

Dissection Aortique

Physiopathologie

- Contraction cardiaque (43 millions battements par année)
- mouvements flexion Ao
- Stress de la paroi aortique
 - Intima
 - Media (tissue connective et muscle lisse)
 - Adventice

Dissection Aortique

Classification

Table 1 Classifications of AADs	
Classification of Aortic Dissection	
Stanford	
Type A	Involving the ascending aorta
Type B	Not involving the ascending aorta
DeBakey	
Type I	Originates in ascending aorta and extends to arch and often beyond into the descending aorta
Type II	Originates and localized to the ascending aorta
Type III	Originates and is localized to the descending aorta only
	Type IIIa: involves thoracic descending aorta only
	Type IIIb: extends beyond the diaphragm

Dissection Aortique

Classification

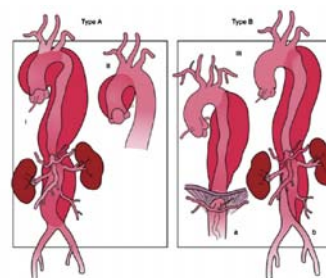
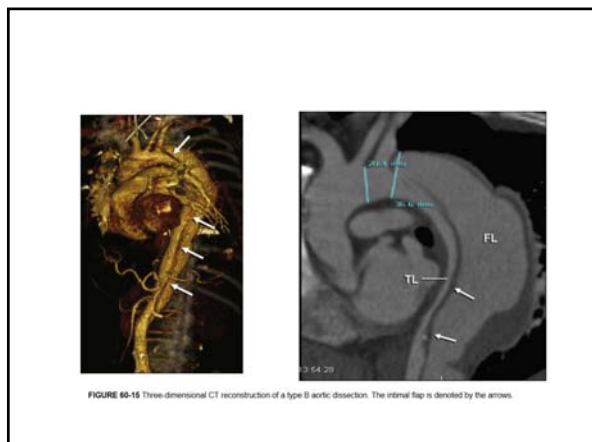


Fig. 1. Aortic dissections.



Dissection Aortique

Facteurs de risque

- Trauma, blunt or iatrogenic
 - Catheter or stent
 - Intra-aortic balloon pump
 - Aortic or vascular surgery
 - Motor vehicle accident
 - Coronary artery bypass surgery or aortic valve replacement
- Cocaine use
- Inflammatory or infectious disease
 - Giant cell arteritis
 - Takayasu arteritis
 - Behçet disease
 - Aortitis
 - Syphilis
- Pregnancy

Dissection Aortique

Facteurs de risque

- Hypertension
- Genetically triggered thoracic aortic disease
 - Marfan syndrome
 - Bicuspid aortic valve
 - Loeys-Dietz syndrome
 - Hereditary thoracic aortic aneurysm or dissection
 - Vascular Ehlers-Danlos syndrome
- Congenital diseases or syndromes
 - Coarctation of the aorta
 - Turner syndrome
 - Tetralogy of Fallot
- Atherosclerosis
 - Penetrating atherosclerotic ulcer

Dissection Aortique

Présentation clinique

Aortic Dissection	
Presenting Hemodynamics and Clinical Findings	Frequency/Finding
Hypertensive	32%
Normotensive	45%
Hypotensive	14%
Shock	13%
Cardiac tamponade	5%
Murmur of aortic insufficiency	45%
Pulse deficits	26%
Pericardial friction rub	2%
Cerebrovascular accident	8%
Ischemic peripheral neuropathy	3%
Ischemic spinal cord damage	2%
Ischemic lower extremity	10%
Coma/altered consciousness	12%
Congestive heart failure	5%
First blood pressure systolic, mean	130 mm Hg
First blood pressure diastolic, mean	75 mm Hg

Adapted from Page et al.²²⁷

Dissection Aortique

Pression artérielle

- 50 % patients avec HTA
- 71 % type B TAS > 150 mmHg vs 36% type A
- 20% Hypo TA ou choc
- TA peut être difficile à prendre
- Idéalement aux 4 membres

Journal of the American Heart Association

Society of Cardiovascular Anesthesiologists

Cardiovascular Anesthesiology Section Editor: Charles W. Hogue, Jr.
 Perioperative Echocardiography and Cardiovascular Education Section Editor: Martin J. London
 Hemostasis and Transfusion Medicine Section Editor: Jerrold H. Levy

2010 ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM Guidelines for the Diagnosis and Management of Patients with Thoracic Aortic Disease: Executive Summary

A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, American Association for Thoracic Surgery, American College of Radiology, American Stroke Association, Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, Society of Interventional Radiology, Society of Thoracic Surgeons, and Society for Vascular Medicine

Dissection Aortique

Évaluation

Évaluation du risque

Class I

a. High-risk conditions and historical features^{47,127,223,261} (Level of Evidence: B):

- Marfan syndrome, Loeys-Dietz syndrome, vascular Ehlers-Danlos syndrome, Turner syndrome, or other connective tissue disease.
- Patients with mutations in genes known to predispose to thoracic aortic aneurysms and dissection, such as *FBN1*, *TGFBR1*, *TGFBR2*, *ACTA2*, and *MYH11*.
- Family history of aortic dissection or thoracic aortic aneurysm.
- Known aortic valve disease.
- Recent aortic manipulation (surgical or catheter-based).
- Known thoracic aortic aneurysm.

Dissection Aortique

Évaluation

Évaluation du risque

Class I

b. High-risk chest, back, or abdominal pain features^{37,47,215,223,261,264,268} (Level of Evidence: B):

- Pain that is abrupt or instantaneous in onset.
- Pain that is severe in intensity.
- Pain that has a ripping, tearing, stabbing, or sharp quality.

c. High-risk examination features^{37,47,253,257,261,324} (Level of Evidence: B):

- Pulse deficit.
- Systolic blood pressure limb differential greater than 20 mm Hg.
- Focal neurologic deficit.
- Murmur of aortic regurgitation (new).

Dissection Aortique

Évaluation

Évaluation du risque

Class I

2. Patients presenting with sudden onset of severe chest, back, and/or abdominal pain, particularly those less than 40 years of age, should be questioned about a history and examined for physical features of Marfan syndrome, Loeys-Dietz syndrome, vascular Ehlers-Danlos syndrome, Turner syndrome, or other connective tissue disorder associated with thoracic aortic disease.²²³ (Level of Evidence: B)

3. Patients presenting with sudden onset of severe chest, back, and/or abdominal pain should be questioned about a history of aortic pathology in immediate family members as there is a strong familial component to acute thoracic aortic disease.²²³ (Level of Evidence: B)

Dissection Aortique

Évaluation

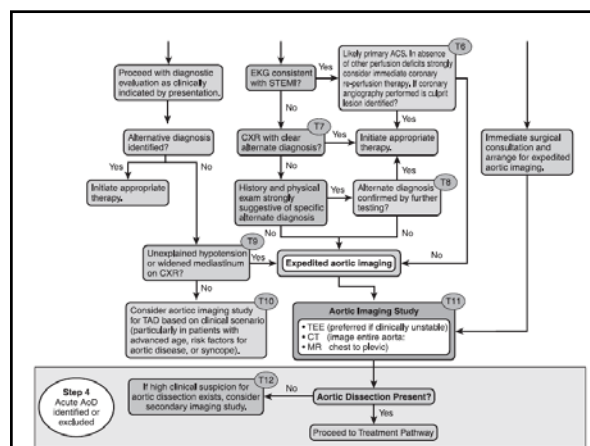
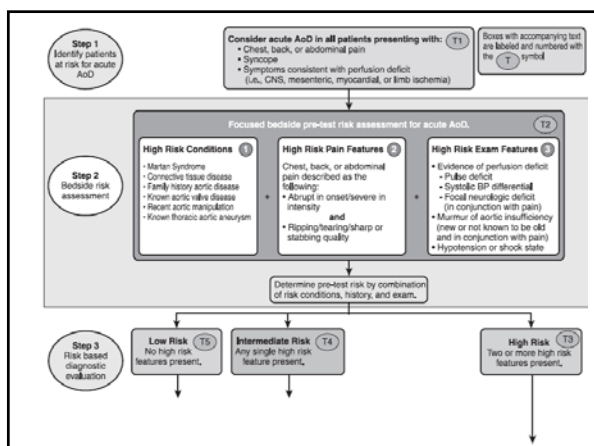
Évaluation du risque

Class I

4. Patients presenting with sudden onset of severe chest, back, and/or abdominal pain should be questioned about recent aortic manipulation (surgical or catheter-based) or a known history of aortic valvular disease, as these factors predispose to acute aortic dissection. (Level of Evidence: C)

5. In patients with suspected or confirmed aortic dissection who have experienced a syncope episode, a focused examination should be performed to identify associated neurologic injury or the presence of pericardial tamponade (see Section 8.1.6). (Level of Evidence: C)

6. All patients presenting with acute neurologic complaints should be questioned about the presence of chest, back, and/or abdominal pain and checked for peripheral pulse deficits as patients with dissection-related neurologic pathology are less likely to report thoracic pain than the typical aortic dissection patient²⁵³ (see Section 8.1.7). (Level of Evidence: C)



Dissection Aortique

Pression artérielle

Table 5

Initial medications for blood pressure control in aortic dissection

Medication Class (Agent)	Starting Dose	Infusion Regimen	Cautions for Use
β-Blockers			
1. Esmolol	Bolus 500 µg/kg	50–200 µg/kg/min	COPD or high-risk bronchospasm;
2. Labetalol	Bolus 20 mg every 5–10 min to 80–300 mg total	1–2 mg/min	consider selective β-blocker such as metoprolol or atenolol
Sodium Nitroprusside		0.5–3.0 µg/kg/min	Concomitant use of β-blocker to avoid reflex tachycardia, possible cyanide toxicity with prolonged use

Dissection Aortique

Complications

- Cardiaques
 - Régurgitation aortique aigue type A (41-76%)
 - 1) dilatation de la racine Ao et expansion de la fausse lumière



Dissection Aortique

Complications

- Cardiaques
 - Régurgitation aortique aigue type A (41-76%)
 - 2) dissection ad la racine Ao avec prolapsus valvulaire
 - 3) obstruction valvulaire en diastole par un segment du flap

Dissection Aortique

Complications

- Cardiaques
 - IM rare 19% à l'ECG
 - 7% dans 9 études 988 pts
 - Défaillance et choc 6%
 - 1) Ins Ao
 - 2) IM
 - 3) Tamponnade

Dissection Aortique

Complications

- Cardiaques
 - Effusion et Tamponnade 8 à 10%
 - 1) transsudation de la fausse lumière vers le sac péricardique 1/3 Pts
 - 2) rupture de la dissection Ao directement dans le péricarde
- Mauvais pronostique ☹

Dissection Aortique

Complications

- Neurologiques ad 17 % 1300 pts (type A et B)
 - Hypo T
 - Mal perfusion
 - Thrombo-embolisme distal
 - Compression

MECHANISM AND LOCATION	MANIFESTATIONS
Brain:	
Extension of dissection to aortic arch vessels	Transient ischemic attack/Stroke Transient global amnesia
Reduced cerebral perfusion due to hypotension	Hypoxic encephalopathy Homer syndrome
Nerve compression by enlarging false lumen	Seizure Coma
Spinal Cord:	
Spinal cord ischemia due to spinal artery obstruction	Paraparesis Paraplegia Anterior spinal cord syndrome Brown-Sequard syndrome Progressive myelopathy Transient spinal cord ischemia
Peripheral Nerve:	
Obstruction of vasa nervorum	Ischemic neuropathy (paraparesis, polyneuropathy, mononeuropathy)
Compression of a nerve by enlarging false lumen	Ischemic plexopathy Nerve compression syndrome

Reproduced with permission from Gaul C, Dietrich W, Erbguth F.J. Neurological symptoms in acute aortic dissection: a challenge for neurologists. Cerebrovasc Dis 26:1, 2008.

Dissection Aortique

Complications

- Pulmonaires
 - Épanchement pleural ad 16 %
 - Compression de l'artère pulmonaire
 - Fistule aorto-pulmonaire
 - Hémoptysies

Dissection Aortique

Complications

- GI
 - Ischémie mésentérique (plus fréquente)
 - Hémorragie
- Occlusion artérielle aiguë
 - dommage irréversible 4-6 hrs

- Occlusion artérielle aiguë
- 5 P
- Embolie ou thrombus
- Classification (Rutherford)

Catégorie	Clinique			Doppler	
	pronostic	Déficit sensoriel	Déficit moteur	Artériel	Veineux
I. Viable					
	Pas de menace	Aucun	Aucun	Audible	Audible
II. Menacé					
a. Modérément	Sauvetage si traitement rapide	Minime (orteils) ou absent	Aucun	Inaudible	Inaudible
b. Immédiatement	Sauvetage si revascularisation	Dépassement les orteils avec douleur au repos	modérée	Inaudible	Inaudible
III. Irréversible	Amputation majeure ou séquelles neurologiques permanentes inévitables	Anesthésie profonde	paralyse	Inaudible	Inaudible

Dissection Aortique

Table 11. End-Organ Complications of Acute Aortic Dissection

Type	End-Organ Complication
Cardiovascular	Aortic insufficiency
	Syncope
	Pericardial tamponade
	Myocardial ischemia or infarction
Neurologic	Congestive heart failure
	Ischemic stroke or transient ischemic attack
	Peripheral neuropathy
	Paraplegia/paraparesis
	Spinal ischemia
Pulmonary	Pleural effusion
	Aortopulmonary fistula with hemorrhage
Gastrointestinal	Mesenteric ischemia or infarction
	Aortoenteric fistula with hemorrhage
Renal	Renal failure
Extremities	Renal ischemia or infarction
	Limb ischemia

Dissection Aortique

Indications chirurgicales

- Compromis de l'aorte ascendante (A, I et II)
- Formation d'un anévrisme
- Rupture imminente
- Ischémie
- Réponse inapproprié au Rx

Dissection Aortique

Indications chirurgicales

- Dysfonction valvulaire
- Tamponnade
- Occlusion ou rupture vasculaire

Choix de l'approche chirurgicale

TEVAR vs chirurgie ouverte

- Pas d'étude randomisée comparant les 2
- Pas d'étude comparant M et M entre patients âgés et plus jeunes

TEVAR vs chirurgie ouverte

- Mortalité élevée pour les dissections aiguës de type B
- Similaire pour tx médical optimal ou chirurgical standard
- TEVAR: risque de mortalité et de paralysie plus faible à court terme?
- TEVAR: complications augmentées à long terme? (migration, infection, anévrisme, nouvelle dissection, etc)

TEVAR vs chirurgie ouverte Recommandation:

- TEVAR lorsque le risque opératoire est clairement plus bas que le risque de l'approche chirurgicale ouverte ou que celui de la thérapie médicale optimale

Svensson et al. Expert consensus document on the treatment of descending thoracic aortic disease using endovascular stent-grafts. Ann Thorac Surg. 2008

Table 3. Early Outcomes for Open Versus Endovascular Repair

Variable	Open Repair (n = 24)	TEVAR (n = 49)	p Value
In-hospital or 30-day mortality	4 (16.7%)	5 (10.2%)	0.46
Stroke	3 (12.5%)	3 (6.1%)	0.38
Need for dialysis	4 (16.7%)	2 (4.1%)	0.08
Spinal cord ischemia	1 (4.2%)	3 (6.1%)	1.0
Need for tracheostomy	4 (16.7%)	3 (6.1%)	0.21
Composite early outcome	7 (29.2%)	9 (18.4%)	0.30
Median length of stay (days)	28.1	17.5	0.05
Median duration of mechanical ventilation (h)	27	5	<0.001
Median blood product use (units of red cells, fresh-frozen plasma, or platelets)	6	0	<0.001

TEVAR = thoracic aortic endovascular repair.

Wilkinson et al. Early Open and Endovascular Thoracic Aortic Repair for Complicated Type B Aortic Dissection, Ann Thor Surg, 2013

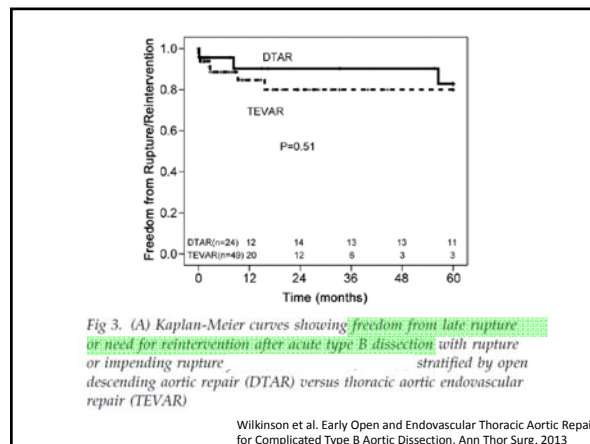
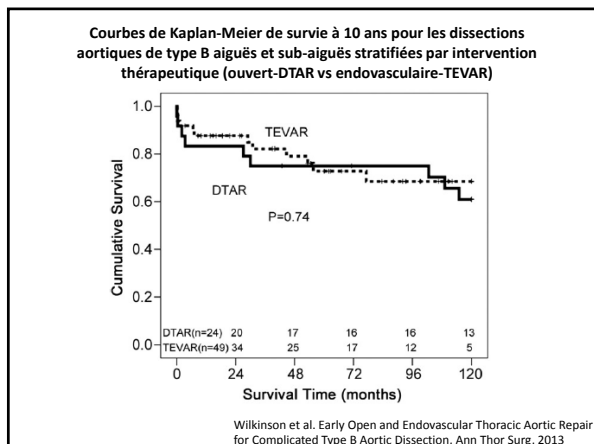
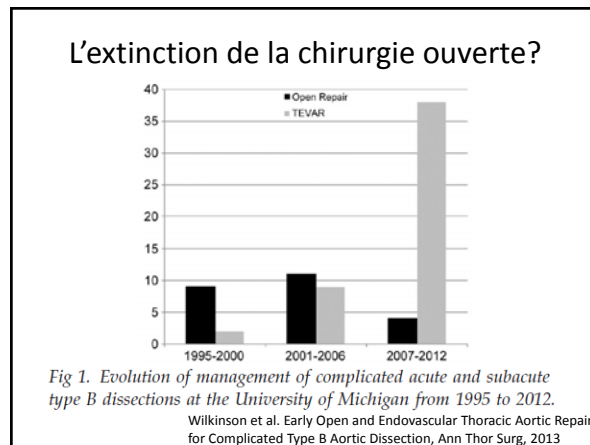


Table 15. Summary of Society of Thoracic Surgeons Recommendations for Thoracic Stent Graft Insertion

Entity/Subgroup	Classification	Level of Evidence
Penetrating ulcer/intramural hematoma		
Asymptomatic	III	C
Symptomatic	IIa	C
Acute traumatic		
Acute traumatic	I	B
Chronic traumatic	IIa	C
Acute Type B dissection		
Acute Type B dissection Ischemia	I	A
Chronic dissection		
Degenerative descending >5.5 cm, comorbidity	IIa	B
>5.5 cm, no comorbidity	IIb	C
<5.5 cm	III	C
Arch		
Reasonable open risk	III	A
Severe comorbidity	IIb	C
Thoracoabdominal/severe comorbidity	IIb	C

Reprinted from Svensson et al.²⁷¹



Prise en charge per opératoire

- Objectifs généraux per op**
- Contrôle HD entre la phase pré op à intra op
 - Mesurer et prévenir l'atteinte des organes
 - Traiter les comorbidités
 - Contrôler le saignement

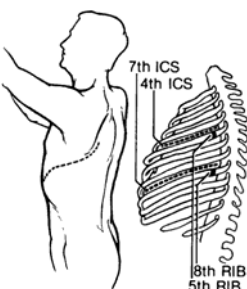
Monitoring/Installation

- Site approprié (Salle hybride)
- Accès veineux pour transfusions
- Réchauffe soluté/ sac pression
- Canule artérielle radiale droite
- Canule artérielle fémorale pour monitoring pression perfusion durant clampage aortique
- ETO (IIa/B)
- Swan

Monitoring/Installation

- SSEP et MEP (IIa/B)
- Drain lombaire
- TDL pour chirurgie ouverte
- Produits sanguins en réserve
- Anti fibrinolytiques (IIa/C)
- Auto transfuseurs
- Équipe chirurgicale en backup

Positionnement pour chirurgie ouverte



- Une incision musculocutanée et 2 incisions intercostales 4^e et 7^e espaces
- DLD avec rotation du bassin pour accès aux vaisseaux fémoraux
- Limiter risque de blessures

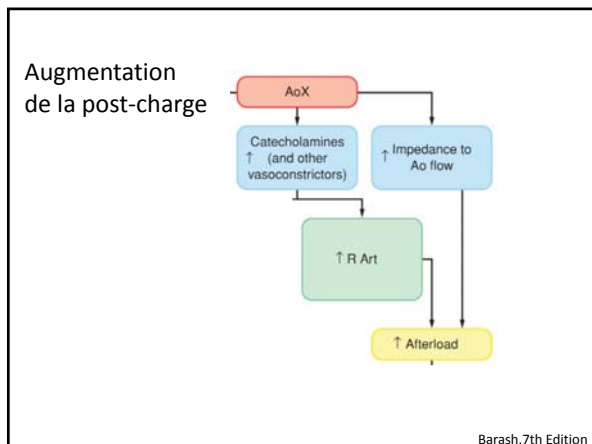
Induction

- AG / Régionale / Locale et sédation ont été utilisées
- Poursuivre le traitement médical optimal, réduire dP/dT
- Viser FC < 60 bpm
- Viser TAS < 120 mmHg ou (le + bas toléré)
- Selon la situation: hypovolémie/choc
- Estomac plein

Changements hémodynamiques propres à la chirurgie ouverte

Changements hémodynamiques avec le AoX

- ↑ Post- charge VG
- ↑ Pré-charge
- ↑ ou ↔ ou ↓ Fonction VG / contractilité / CO



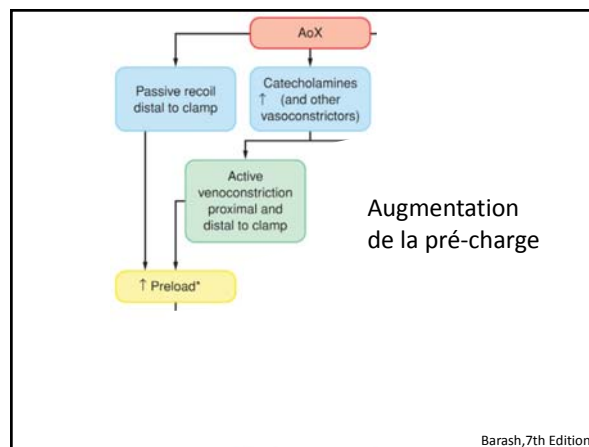
↑ Post-Charge dépend du niveau de l'occlusion

Table 1.

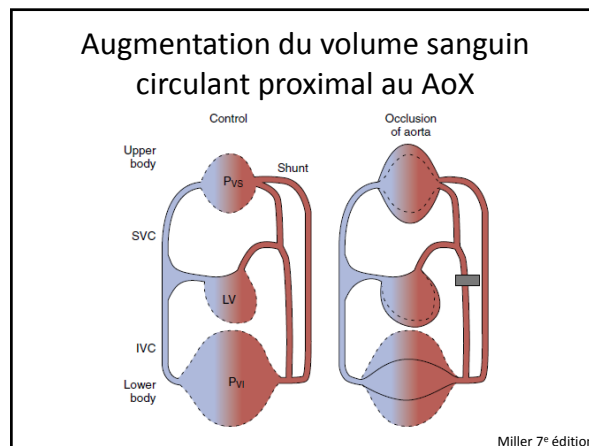
Cardiovascular Variable	Percentage (%) Change in Occlusion			
	Descending Thoracic Aorta	Supraoelical	Suprarenal-Infracoelical	Infrarenal
Mean arterial blood pressure	35 - 84 ↑	64 ↑	5 - 10 ↑	2 ↑
Pulmonary capillary wedge pressure	90 - 150 ↑	38 ↑	10 ↑	0
CVP	35 ↑			
Cardiac index	29 ↓		10 - 33 ↓	
End-diastolic area		28 ↑	2 ↑	9 ↑
End-systolic area		69 ↑	10 ↑	11 ↑
Ejection fraction		38 ↓	-10 ↓	-3 ↓
Patients with wall motion abnormalities		92 ↑	33 ↑	0

Levin A. Refresher Course: The cardiovascular effects of aortic clamping and unclamping. S Afr J Anaesthesiol Analg 2010;16(2)

- ↑ Pré-charge
- 2 raisons:
 - Théorie de la redistribution des volumes
 - L'augmentation de la pré-charge induite par l'augmentation de la post-charge (after-load mismatch)



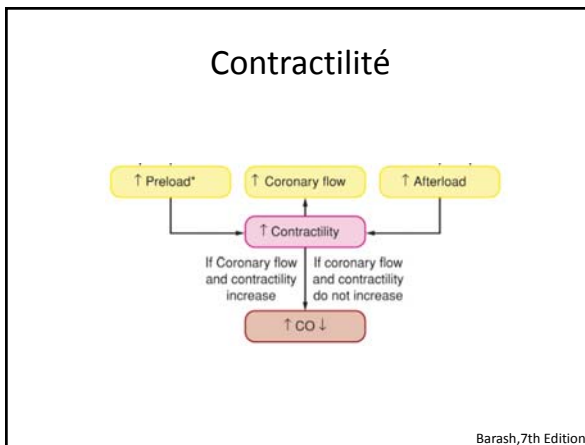
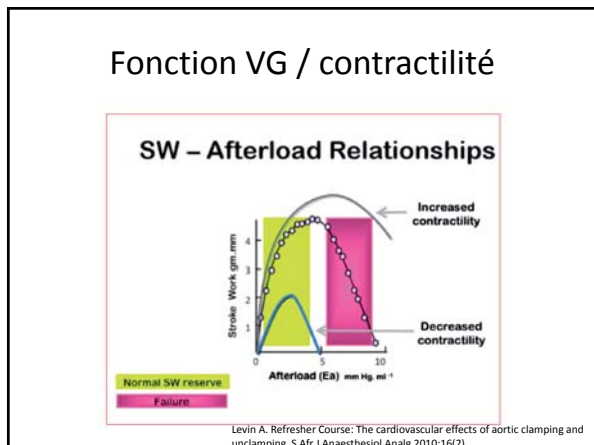
- Redistribution des volumes
- Rôle central de la vascularisation splanchnique très compliante
 - 25% du vol sanguin dont 800 mL peuvent être « autotransfusé » en quelques secondes
 - Recul (Recoil) passif de la vasculature distale
 - Sécrétion Épi/NE augmente tonus vasculaire veineux





↑ Pressions de charge

- Cœur « normal » : étude animale, pas de changement ou ↑ du CO
- Cœur MCAS: ↓ CO chez les humains
 - ↑ pression transmurale (ischémie subendocardique)
 - distension des cavités
 - ischémie coronarienne
 - activation des barorécepteurs → ↓ FC, ↓ contractilité



Clampage - Thérapies

Therapeutic Interventions

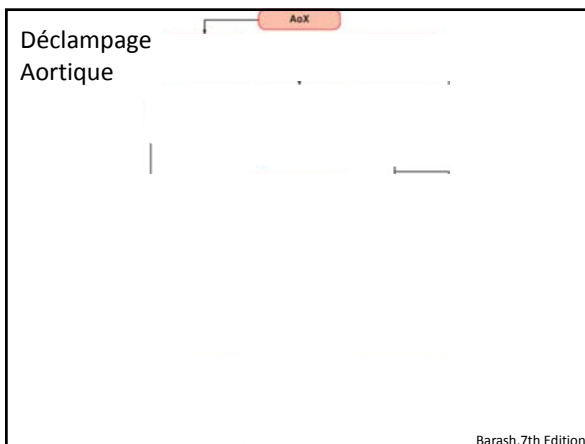
Afterload reduction

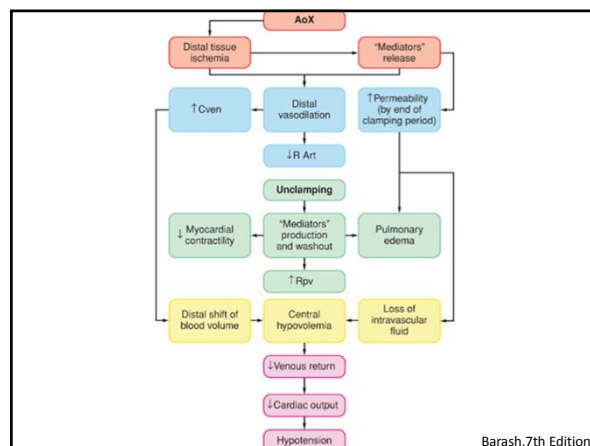
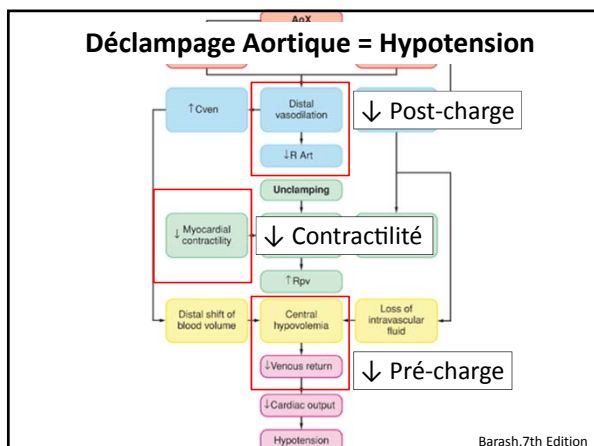
- Sodium nitroprusside
- Inhaled anesthetics
- Amrinone
- Shunts and aorta-to-femoral bypass

Preload reduction

- Nitroglycerin
- Controlled phlebotomy
- Atrial-to-femoral bypass

Miller 7^e édition





Déclantage - Thérapies

Therapeutic Interventions

- ↓ Inhaled anesthetics
- ↓ Vasodilators
- ↑ Fluid administration
- ↑ Vasoconstrictor drugs
- Reapply cross-clamp for severe hypotension
- Consider mannitol
- Consider sodium bicarbonate

Miller 7^e édition

- ### Contrôle HD lors du déploiement de l'endoprothèse
- Arche aortique distale et l'aorte descendante proximale sont + problématiques
 - Contrôle optimal et FC et TAM:
 - Pharmacologique
 - Vasodilateurs
 - B-bloqueurs
 - Adénosine 0.5-1.5 mg/kg pour asystolie de 20-30 sec

- ### Contrôle HD lors du déploiement de l'endoprothèse
- Électrique
 - « Pacing » ventriculaire rapide : 160-200 bpm pour TAM 20-30 mmHg
 - Pad externe de défib en place si induction de tachyrythmie (mais < 1%)
 - Mécanique
 - Occlusion de la VCI par ballon

Complications neuro

Table 3. Risk Factors for Stroke and Spinal Cord Ischemia

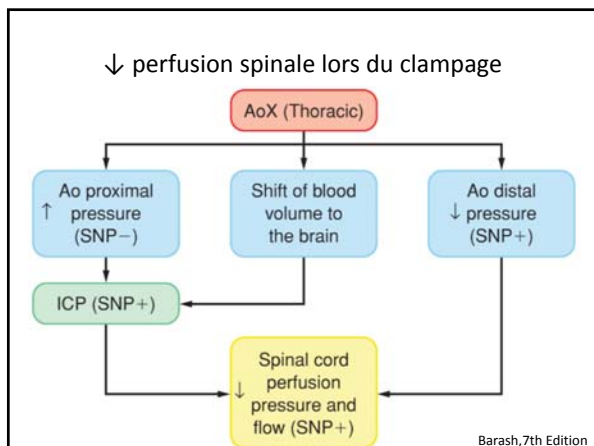
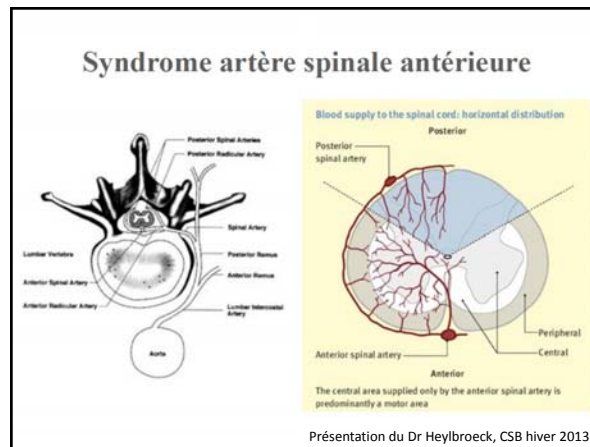
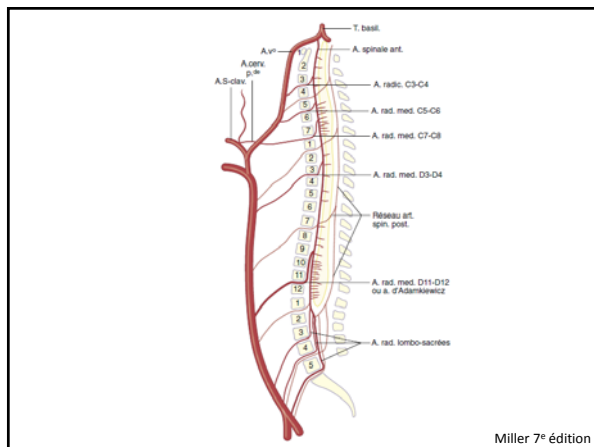
	Risk Factors
Stroke	Ascending aortic and aortic valve repair Left subclavian coverage without bypass Aortic arch wire/catheter manipulation
Incidence en chir ouverte 4-9%	Female gender Prolonged operative procedure
Spinal cord ischemia	Prolonged operative procedure
Incidence 0-8%	Perioperative blood loss Open thoracoabdominal aortic repair Intraoperative blood loss Extent of descending aorta covered by endograft Renal failure History of previous aortic repair

Jazaeri et al. Endovascular approaches and perioperative considerations in acute aortic dissection, Seminars in cardiothoracic and vascular anesthesia 2011

ACV péri op

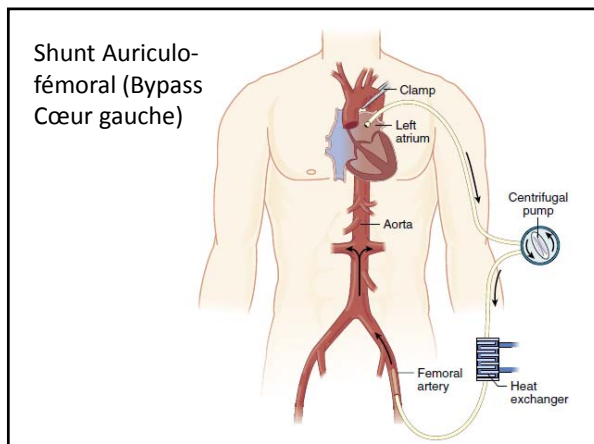
- TEVAR: Micro-embolies (le + fréquent)
- Chirurgie ouverte: Multifactoriel
- Monitoring futur: Doppler trans cranien?

Anatomie pertinente de la vascularisation de la moelle épinière



Ischémie de la moelle épinière Avenues thérapeutiques/prévention

- Maintenir et préserver le flot collatéral
 - TAM > 70 mmHg
 - Drainage LCR
 - Pour maintenir «PIC» < 10 mmHg
 - «PPC» > 65 mmHg?
- Maintenir pression distale? / Shunt vasculaire
- Papavérine en infusion épidurale?



Ischémie de la moelle épinière Avenues thérapeutiques/prévention

- Diminuer le métabolisme cellulaire
 - Hypothermie modérée
 - Agents anesthésiques
 - Irrigation épidurale solution hypothermique
- Diminuer l'effet de l'ischémie
 - Glucocorticoïdes?
 - Mannitol?
- Ajuster traitement en fonction du monitoring de SSEP ou MEP?

Circulation
Journal of the American Heart Association

American Heart Association

Society of Cardiovascular Anesthesiologists

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SPECIAL ARTICLE

2010 ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM Guidelines for the Diagnosis and Management of Patients with Thoracic Aortic Disease: Executive Summary

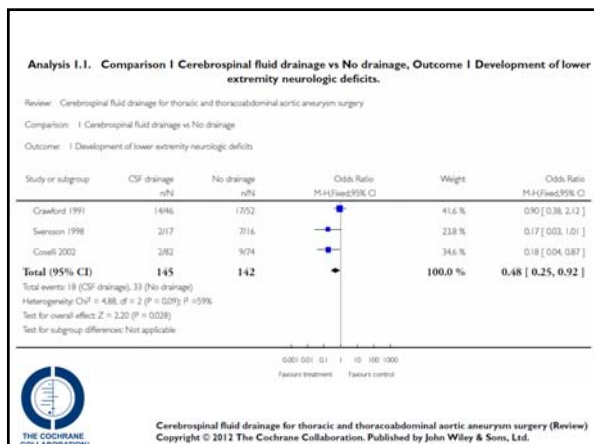
A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, American Association for Thoracic Surgery, American College of Radiology, American Stroke Association, Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, Society of Interventional Radiology, Society of Thoracic Surgeons, and Society for Vascular Medicine

Drainage lombaire - Classe I/B

14.5.2. Recommendations for Spinal Cord Protection During Descending Aortic Open Surgical and Endovascular Repairs

Class I

1. Cerebrospinal fluid drainage is recommended as a spinal cord protective strategy in open and endovascular thoracic aortic repair for patients at high risk of spinal cord ischemic injury.^{522,523,713} (Level of Evidence: B)



CSFD has been shown to benefit for Types I and II TAAAs, but the results cannot be extrapolated to the repair of less extensive aneurysms. There is currently no clinical evidence that show that CSFD prevents deficits during the repair of Type III or IV TAAAs.

Cases of spinal cord dysfunction following endovascular repair have also been reported. The role of CSFD in such a setting remains to be determined.

Optimisation de la pression de perfusion – Classe IIa/B

Class IIa

1. Spinal cord perfusion pressure optimization using techniques, such as proximal aortic pressure maintenance and distal aortic perfusion, is reasonable as an integral part of the surgical, anesthetic, and perfusion strategy in open and endovascular thoracic aortic repair patients at high risk of spinal cord ischemic injury. Institutional experience is an important factor in selecting these techniques.^{380,382,714,715} (Level of Evidence: B)

Pas d'étude randomisée

Hypothermie modérée – Classe IIa/B

Class IIa

2. Moderate systemic hypothermia is reasonable for protection of the spinal cord during open repairs of the descending thoracic aorta.⁵²⁵ (Level of Evidence: B)

Table 33.2 Principal SC ischemia prevention strategies during open repair of thoracic aortic aneurysm

Target	Strategy
1. Minimize SC ischemia time	a. Reduce aortic clamping times b. Sequential aortic clamping c. Distal aortic perfusion
2. Preserve SC blood supply	a. Revascularization of "critical" intercostal arteries b. Preserve subclavian, hypogastric arteries
3. Increase tolerance to ischemia	a. Mild systemic hypothermia b. Deep hypothermic circulatory arrest c. Selective epidural cooling d. Pharmacologic neuroprotection
4. Optimization of SC perfusion	a. Controlled hypertension b. Lumbar CSF drainage c. Avoid steal phenomenon
5. Early detection of SC ischemia	a. Intraoperative measurement of motor evoked potential, somatosensory evoked potential b. Biochemical tests of SC injury c. Early neurological assessment status

Chiesa R, Melissano G et Zangrillo A. Thoraco-Abdominal Aorta: Surgical and Anesthetic Management, Springer 2011, p. 415

Prévention de l'IRA

- Jusqu'à 25% d'IRA en post op
- Jusqu'à 8% hémodialyse
- FR: Âge, IRC, temps ischémie, transfusions, instabilité HD, athérosclérose
- Modalités:
 - Hydratation et mannitol (IIb/C)
 - Perfusion sélective des artères rénales (IIb/C)
 - Mannitol, furosémide et dopamine seuls (III/B)

Complications post-op

- SCA (1-5%)
- IC (1-5%)
- Infections (1-5%)
- AVC (2-8%)
- Ré intervention pour saignement (1-6%)
- Insuffisance respiratoire (5-15%)
- Arythmies (1-5%)
- Paralysie (2-4% aorte thoracique, 3-10% thoraco-abdo), surtout déficit moteur
- Atteinte du N récurrent laryngé

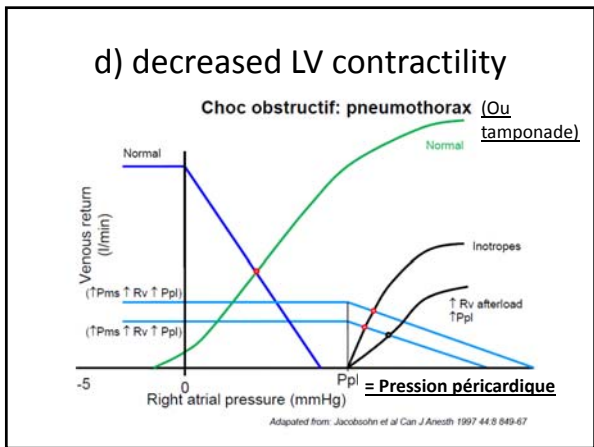
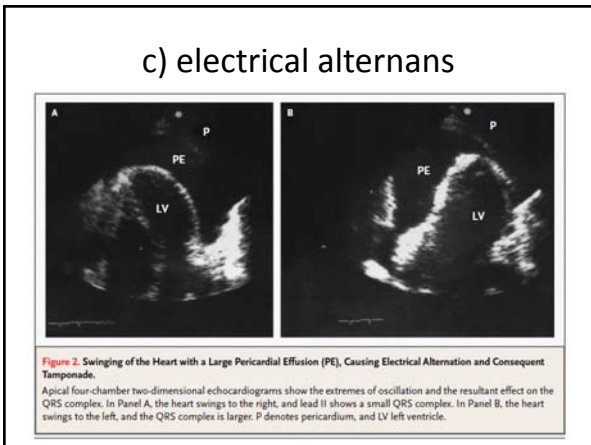
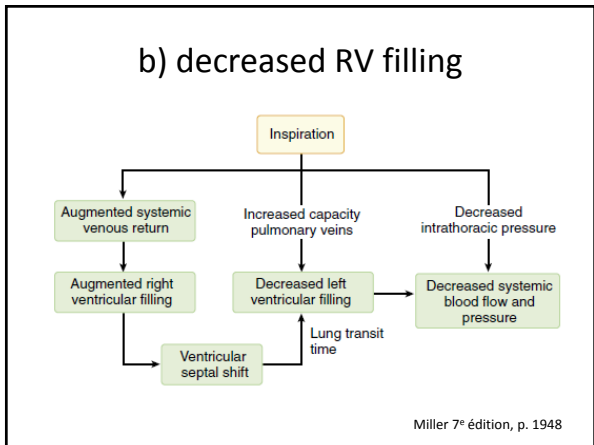
Complications post-op

- Ischémie viscères (1-10%)
- Insuffisance respiratoire (jusqu'à 20%)
 - 2° AoX
 - Large thoracotomie
 - Trauma chir
 - Atteinte N phrénique
- IRA

Questions QCM

Question # 23) Which is seen in cardiac tamponade, except:

- a) decreased LV, RV pressures
- b) decreased RV filling
- c) electrical alternans
- d) decreased LV contractility
- e) hypotension, increased heart rate



- ### e) hypotension, increased heart rate
- Lorsque les mécanismes compensateurs sont épuisés
 - Habituellement, haut tonus adrénergique = tachycardie
 - Triade de Beck: Hypotension, ↑ TVC, bruits cardiaques lointains

a) decreased LV, RV pressures

- PCWP and LV, RV, and RA diastolic pressure increase to equalize with pressure in the pericardium

Miller 7^e édition, p. 1948

Question # 23) Which is seen in cardiac tamponade, except:

a) decreased LV, RV pressures

- b) decreased RV filling
- c) electrical alternans
- d) decreased LV contractility
- e) hypotension, increased heart rate

All increase the threshold of cardiac pacemaker capturing EXCEPT:

- 1) Hypokalemia
- 2) Myocardial ischemia
- 3) Hypothermia
- 4) Acidosis

Myocardial stimulation threshold

Energie minimal requise pour stimuler le myocarde

Relation entre l'électrode et les cellules de Purkinje

Plusieurs facteurs physiologiques et pharmacologiques impliqués

« exit block »

1) Hypokalemia

Conduction lente

Hyperpolarisation de la membrane

Augmentation du seuil excitatoire

Augmentation de la pente de dépolarisation diastolique

Surcharge de Ca

Inhibition de la pompe Na K.

2) Myocardial ischemia

- Hypoxie → diminution du potentiel de repos associé à l'inactivation du transport du Sodium donc réponse au stimuli électrique diminue
- Perte du K intracellulaire
- Ischémie produit diminution du période réfractaire

3) Hypothermia
 32°C to 34°C
 augmentation de l'adenosine triphosphate
 plus de stabilité a/n de la membrane mitochondriale
 Optimisation du flot microvasculaire cardiaque

3) Hypothermia
 At temperatures below 28°C, sinoatrial pacing becomes erratic and ventricular irritability increases.
 Fibrillation usually occurs between 25°C and 30°C, and electrical defibrillation is generally ineffective at these temperatures.

4) Acidosis
 Hyper K > 7 mmol/L
 Diminution du potentiel de membrane en repos
 Diminution de la dépolarisation (Phase 0)
 Potentiel d'action plus court

Effect	Drugs	Other factors
Increase pacing threshold	Bretylium, encainide, flecainide, moricizine, propafenone, sotalol	Myocardial ischemia and infarction; progression of cardiomyopathy; hyperkalemia; severe acidosis or alkalosis; hypoxemia; after ICD shocks or external cardioversion or defibrillation
Possibly increase pacing threshold	β Blockers, lidocaine, procainamide, quinidine, verapamil	Myxedema; hyperglycemia
Possibly decrease pacing threshold	Atropine, catecholamines, glucocorticoids	Pheochromocytoma; hyperthyroid or other hypermetabolic states
No proven effect on pacing threshold	Amiodarone; anesthetic drugs, both inhalation and intravenous	

ICD = internal cardioverter-defibrillator.

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Cardiac Rhythm Management Devices (Part II)
Perioperative Management
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