Recommendations and Guidelines for Preoperative Evaluation of the Surgical Patient with Emphasis on the Cardiac Patient for Non-cardiac Surgery



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Preoperative preparation of the patient for non-cardiac surgery may be complex. The variety of presenting conditions may be difficult to define prior to surgery:

- What tests should be ordered?
- When is a long-standing condition in satisfactory control, or should some additional study or medication be added prior to operation?
- What are the risks of anesthesia and of the operation for the patient?

The following collection of information from many sources is designed to be a quick reference for anyone who is involved in the preparation of the patient for non-cardiac surgery. These are proposed guidelines and in **no way** should supersede good clinical evaluation and assessment.

The following information has been reviewed by Richard R. Miles, M.D., Cardiology; Myrna C. Newland, M.D., Anesthesiology; B. Timothy Baxter, M.D., Vascular Surgery; and Frank O. Hayworth, M.D., Anesthesiology. Compilation of the following references was through the efforts of Barbara J. Sink, PA-C, Anesthesia Preoperative Evaluation Unit; Kathi M. Healey, R.N., M.S.N., Cardiology; and computing support from Stephen J. Froscheiser, B.S., Anesthesia Preoperative Evaluation Unit.

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#### Conditions for Which Preoperative Evaluation Is Strongly Recommended Prior to the Day of Surgery

#### General

- Medical condition inhibiting ability to engage in normal daily activity
- Medical conditions necessitating continual assistance or monitoring at home within the past 6 months
- Admission within the past 2 months for acute or exacerbation of chronic condition

# Cardiocirculatory

- History of angina, coronary artery disease, myocardial infarction
- Symptomatic arrhythmias
- Poorly controlled hypertension (diastolic >110, systolic >160)
- History of congestive heart failure

# Respiratory

- Asthma/COPD requiring chronic medication or with acute exacerbation and progression within past 6 months
- History of major and/or lower airway tumor or obstruction
- Upper and/or lower airway tumor or obstruction
- History of chronic respiratory distress requiring home ventilator assistance or monitoring

# Endocrine

- Non-diet controlled diabetes (insulin or oral hypoglycemic agents)
- Adrenal disorders
- Active thyroid disease

# Neuromuscular

- History of seizure disorder or other significant CNS disease (e.g., multiple sclerosis)
- History of myopathy or other muscle disorders

# Hepatic

• Any active hepatobiliary disease or compromise

# Musculoskeletal

- Kyphosis and/or scoliosis causing functional compromise
- Temporomandibular joint disorder
- Cervical or thoracic spine injury

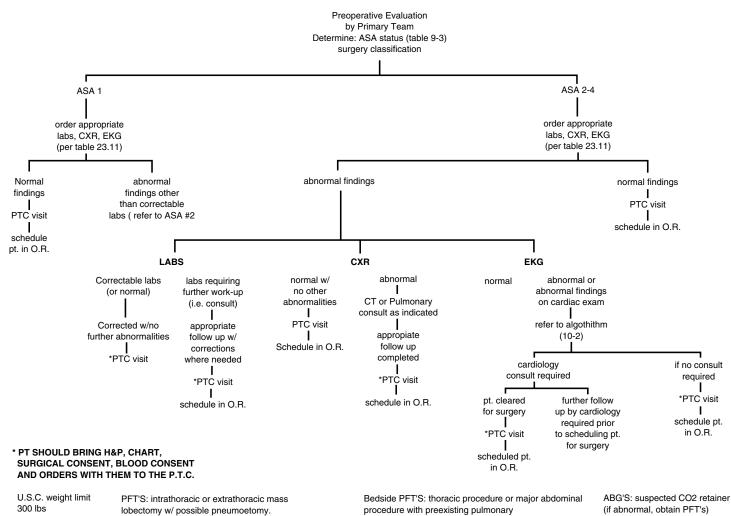
# Oncology

- Patients receiving chemotherapy
- Other oncology process with significant physiologic residual or compromise

#### Gastrointestinal

- Massive obesity (>140% ideal body weight)
- Hiatal hernia
- Symptomatic gastroesophageal reflex

Clinical Anesthesia Updates. *Preanesthesia Evaluation of the Surgical Patient*, vol. 6, #2, p. 5. L. Reuven Pasternak, M.D., M.P.H.



disease (moderate-severe).

Page 7777 if over 300#

# (Guide to Estimation of Physiological Trespass)

# Category 1

Minimal risk to the patient independent of anesthesia

Minimally invasive procedures with little or no blood loss

Often done in office setting with the operating room used principally for anesthesia & monitoring

#### Includes:

Breast biopsy

Removal of minor skin or subcutaneous lesions

Myringotomy tubes

Hysteroscopy

Cystoscopy

Vasectomy

Circumcision

Fiberoptic bronchoscopy

#### Excludes:

Open exposure of internal body organs

Repair of vascular or neurologic structures

Placement of prosthetic devices

Entry into abdomen, thorax, neck, cranium, or extremities

Postoperative monitored care setting (ICU, ACU)

Open exposure of abdomen, thorax, neck, cranium

Resection of major body organs

# Category 2

Minimal to moderately invasive procedure Blood loss less than 500 cc Mild risk to patient independent of anesthesia

**Surgical Classification System** 

**Includes:** Diagnostic laparoscopy Dilation & curettage Fallopian tubal ligation Arthroscopy Inguinal hernia repair Laparoscopic lysis of adhesions Tonsillectomy/adenoidectomy Umbilical hernia repair Septoplasty/rhinoplasty Percutaneous lung biopsy Laparoscopic cholecystectomy Extensive superficial procedures **Excludes:** Open exposure of internal body organs Repair of vascular or neurologic structures Placement of prosthetic devices Postoperative monitored care Major vascular repair (e.g., aortofemoral bypass) Planned postoperative monitored care setting (ICU, ACU)

# Category 3

Moderately to significantly invasive procedure

Blood loss potential 500–1,500 cc

Moderate risk to patient independent of anesthesia

# **Classification System** Surgical

# Includes:

Thyroidectomy

Hysterectomy

Myomectomy

Cystectomy

Cholecystectomy

Laminectomy

Hip/knee replacement

Nephrectomy

Major laparoscopic procedures

Resection, reconstructive surgery of the digestive tract

# **Excludes:**

Open thoracic or intracranial procedure

Major procedure on the oropharynx

Major vascular, skeletal, neurologic repair

# Category 4

**Includes:** 

Highly invasive procedure

Blood loss greater than 1,500 cc

Major risk to patient independent of anesthesia

# **Surgical Classification System**

Major orthopedic-spinal reconstruction Major reconstruction of the gastrointestinal tract Major genitourinary surgery (e.g., radical retropubic prostatectomy Major vascular repair without postoperative ICU stay

# Category 5

Highly invasive procedure

Blood loss greater than 1,500 cc

Critical risk to patient independent of anesthesia

Usual postoperative ICU stay with invasive monitoring

# Includes

Cardiothoracic procedure

Intracranial procedure

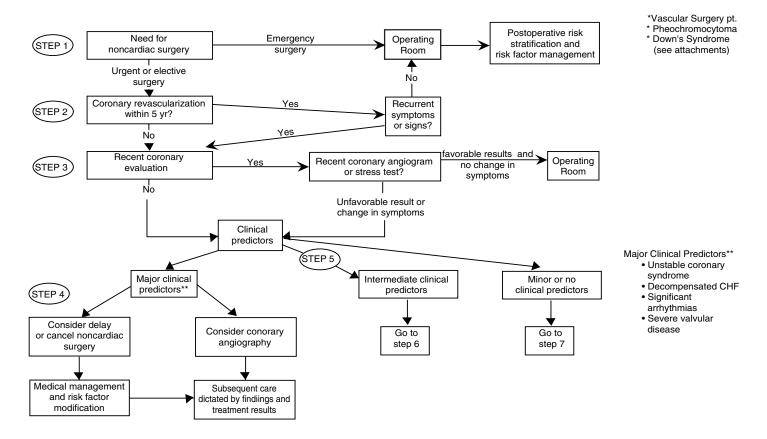
Preanesthesia Evaluation of the Surgical Patient, Vol. 6, No. 2, Pg. 4, Table 2.

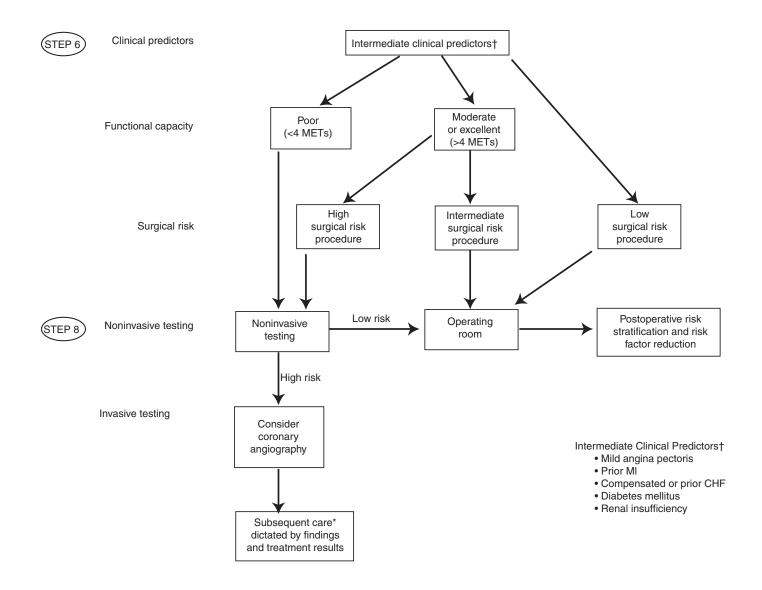
# Physical Classification of the American Society of Anesthesiologists (ASA)

Status	Disease State		
ASA Class 1	No organic, physiologic, biochemical, or psychiatric disturbance.		
ASA Class 2	Mild to moderate systemic disturbance that may or may not be related to the reason for surgery		
	Examples: Heart disease that only slightly limits physical activity, essential hypertension, diabetes mellitus, anemia, extremes of age, morbid obesity, chronic bronchitis		
ASA Class 3	Severe systemic disturbance that may or may not be related to the reason for surgery, ( <i>does limit activity</i> )		
	Examples: Heart disease that limits activity, poorly con- trolled essential hypertension, diabetes mellitus with vascular complications, chronic pulmonary disease that limits activity, angina pectoris, history of prior myocardial infarction		
ASA Class 4	Severe systemic disturbance that is life-threatening with or without surgery		
	Examples: Congestive heart failure, persistent angina pectoris, advanced pulmonary, renal, or hepatic dysfunction		
ASA Class 5	Moribund patient who has little chance of survival but is submitted to surgery as a last resort (resuscitative effort)		
	Examples: Uncontrolled hemorrhage as from a ruptured abdominal aneurysm, cerebral trauma, pulmonary embolus.		
Emergency Operation (E)			
	Any patient in whom an emergency operation is required		
	Example: An otherwise healthy 30-year-old woman who requires a dilation and curettage for moderate but persistent hemorrhage (ASA Class 1 E)		

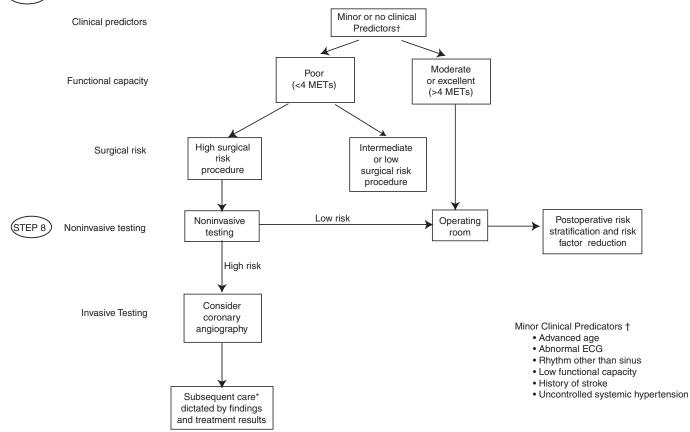
From information in American Society of Anesthesiologists

#### SPECIAL ARTICLE ACC/AHA TASK FORCE ON PRACTICE GUIDELINES ACC/AHA TASK FORCE PERIOPERATIVE CARDIOVASCULAR EVALUATION GUIDELINES





#### STEP 7



Stepwise approach to preoperative cardiac assessment. Steps are discussed in text. \*Subsequent care may include cancellation or delay of surgery, coronary revascularization followed by noncardiac surgery, or intensified care.

#### **Cardiovascular Evaluation for Noncardiac Surgery**

Name:	Vitals:	
Cardiac Meds:		
Surgery Risk:		
High	Intermediate	Low
(Emergency surgery;	(Carotid endarterec-	(Endoscopic; super-
Aortic & major vascu-	tomy; head & neck;	ficial procedure;
lar; PV surgery; proce-	intraperitoneal; intra-	cataract; breast
dures & blood loss or	thoracic; orthopaedic;	surgery)
large fluid shifts)	prostate)	
Existing Cond:		
8	Angina Stable/Uns	stable
(Compensated/decompe	_	
MI	Arrhythmias	>5 PVC/min
CABG ≥5 yrs	≤5 yrs	AICD
	repair/replacement	
AAA w-w/o repair		IHSS
_	BBB/Sick Sinus	
<b>Risk Factors:</b>		
Age	Family Hx.	
Sex	•	
Obesity	Hypercholesterem	ia
Smoking		ons:
<b>~</b>	DM	COPD
	PVD	TIA/CVA

#### **Poor General Med. Condition**

- 1. Electrolyte abn. (K<sup>+</sup>  $\leq$  3.0 meq/l or HCO<sub>3</sub>  $\leq$  meq/l)
- 2. Renal insufficiency (BUN  $\geq$ 50 mg/dl or creat.  $\geq$ 3.0 mg/dl)
- 3. Abnormal ABG's (PO<sub>2</sub>  $\leq$  60 mmHg or PCO<sub>2</sub>  $\geq$  50 mmHg)
- 4. Abnormal liver status
- 5. Chronically bedridden
- Meds: (possible cardiac toxicity)

# Symptoms/findings:

dyspnea	palpitations	rales
orthopnea	syncopal episodes	peripheral edema
chest pain	lightheadedness	cardiomegaly
pulmonary edema/	tortuous or calcified	abnormal EKG
infiltrates	aorta on CXR	in past—yes

# **Duke Activity Status Index:**

1-4 METS(Eating, dressing, walking around house, dishwashing)
4-10 METS(Climbing stairs-1 flight, walking level ground 6.4 km/hr, run-
ning short distance, game of golf)
≥10 METS(Swimming, singles tennis, football)
MET: metabolic equivalent. 1 MET = $3.5 \text{ mL of } O_2/Kg/min.$

#### **Results:**

EKG	ЕСНО	CATH
STRESS	CXR	

1. Have you had any chest pain?	yes/no
2. Have you experienced breathlessness on exertion?	yes/no
3. Have you experienced breathlessness lying flat?	yes/no
4. Has any form of heart disease ever been diagnosed?	yes/no
5. Have you had rheumatic fever?	yes/no
6. Have you ever been found to have a heart murmur?	yes/no

Paterson K. Retal: The preoperative electrocardiogram: an assessment. *Scot Med J* 28:116–118, 1983.

Response to: Recommendations and Guidelines for Preoperative Evaluations

This is a very inclusive document which covers the basics of anesthesia preoperative evaluation. The crux of this handout is the algorithm which outlines the preoperative cardiac assessment. Although I generally agree with this outline, there were a few places which may result in unnecessary preoperative noninvasive testing:

- 1. Under Step 6 in patients with moderate or excellent functional capacity undergoing high-risk surgical procedures. I am assuming that the high-risk procedures may include lower extremity bypass, aortic aneurysm repair, and carotid endarterectomy. Noninvasive testing will invariably be positive in many of these patients. There is, to my knowledge, no good data to support a role for coronary revascularization in a patient with moderate or excellent functional capacity. I would suggest at this decision point that a second option would be to undergo an operation with invasive preoperative monitoring and optimization.
- 2. Under Step 7 in the high surgical risk procedure group with minor clinical predictors, I would also suggest that a decision be made prior to noninvasive testing to consider a surgical procedure with invasive preoperative monitoring. These decisions should be made between the surgeon and the patient with the understanding of what the risk entails.

An article published in the Journal of the American Medical Association, which used computer modeling to assist the outcome of vascular surgery in patients who were moderately or mildly symptomatic, demonstrated an increased mortality in patients who underwent a preoperative evaluation. This was related to the fact that coronary revascularization, because of its own inherent risks, does not lower the overall operative mortality. Therefore, I would submit that we may be doing patients a disservice in these categories by not giving them the option of going directly to surgery. I think the data from the Portland group published in the Journal of Vascular Surgery, in which none of these patients with intermediate or minor clinical predictors were evaluated, and the overall operative mortality was <2%, clearly shows that it is unnecessary to subject these patients to noninvasive testing and possible coronary angiography. Especially considering the cost of this and the probability that the overall recommendation would simply be to use invasive monitoring.

Sincerely,

B.T. But

B. Timothy Baxter, M.D. Associate Professor Department of Surgery

# **Preoperative Physical Examination**

# Vital Signs:

Current values and range while hospitalized

# Height, Weight:

For calculation of drug dosages and pump flows

# Airway:

Anatomic features that could make mask ventilation or intubation difficult *Neck:* 

Jugular venous distention (CHF)

Carotid bruit (cerebrovascular disease)

Landmarks for jugular vein cannulation

# Heart:

Murmurs characteristic of valve lesions

 $S_3$  (increased LVEDP)

 $S_4^{'}$  (decreased compliance)

Click (MVP) or rub (pericarditis)

Lateral PMI displacement (cardiomegaly)

Precordial heave, lift (hypertrophy, wall motion abnormality)

# Lungs:

Rales (CHF) Rhonchi, wheezes (COPD)

# Vasculature:

Sites for venous and arterial access

Peripheral pulses

# Abdomen:

Pulsatile liver (CHF, tricuspid regurgitation)

# Extremities:

Peripheral edema (CHF)

# Nervous System:

Motor or sensory deficits

Clinical Anesthesia, 2nd ed. Barash, Cullen, Stoelting. Chapter 36, pp. 1035-1037.

# Preoperative Findings Suggestive of Ventricular Dysfunction

# History:

History of MI, intermittent or chronic CHF

Symptoms of CHF: fatigue, DOE, orthopnea, PND, ankle swelling

# Physical Examination:

Hypotension/tachycardia (severe CHF)

Prominent neck veins, laterally displaced apical impulse, S<sub>3</sub>, S<sub>4</sub>, rales, pitting edema, pulsatile liver, ascites (tricuspid regurgitation)

# Electrocardiogram:

Ischemia/infarction, rhythm, or conduction abnormalities

# Chest X-ray:

Cardiomegaly, pulmonary vascular congestion/pulmonary edema, pleural effusion, Kerley B lines

# Cardiac Testing:

Cath data—LVEDP >18, EF <0.4, Cl <2.0 l/min<sup>-1</sup>/min<sup>-2</sup>

Echocardiography—low EF, multiple regional wall motion abnormalities

Venticulography—low EF, multiple areas of hypokinesis, akinesis, or dyskinesis

Clinical Anesthesia, 2nd ed. Barash, Cullen, Stoelting. Chapter 36, pp. 1035-1037.

# **Grading of Cardiac Murmurs**

- I. Faintest audible; can be heard only with special effort
- II. Faint, but easily audible
- III. Moderately loud
- IV. Loud; associated with a thrill
- V. Very loud; associated with a thrill. May be heard with a stethoscope off chest
- VI. Maximum loudness; associated with a thrill; heard without a stethoscope

Practical Guide to the Care of the Medical Patient, 2nd ed. Fred F. Ferri, M.D. Chapter 3, p. 18

MURMUR	LOCATION	CHARACTER	DIFFERENTIAL
<b>Still's</b> –benign pediatric Murmur	LLSB to apex	Coarse, vibrating early systolic; $S_2$ splits and closes normally, murmur diminishes with sitting or deep inspiration	VSD, MVP, IHHS
<b>Venous hum</b> –benign pediatric murmur	Neck, USB apex just above the clavicle either medial to the sternocleidomastoid or between its insertions, more on right side	Continuous, gone when supine	PDA, AVMs
<b>MVP</b> (Mitral Valve Prolapse) (Barlow's syn- drome), associated with various inherited con- nective tissue diseases including Ehlers-Danlos, Marfan's, Osteogenesis imperfecta, von Willebrand's	Heard best at apex	Midsystolic click (92%), Crescendo mid-to-late systolic murmur (85%) that moves toward S <sub>1</sub> with standing or other decrease in venous return <b>Sx:</b> Palpitations (PACs, PVC's, premature junctional) <b>Cmplc:</b> Sudden death rarely, PVC, TIA/CVA <b>EKG:</b> normal 76%, may show "inferior and/or, apical ischemia" or PVCs	
Aortic Stenosis- most common in elderly now. Rarely (10%) due to RHD, most is calcific aortic stenosis on bicus- pid or previously normal valve	Loud ejection murmur maxi- mum at base and transmitted into neck vessels, often asso- ciated with a thrill or ejection click; no MR unlike in IHSS; ejection click (split first heart sound) radiates into carotids The ejection murmur may be soft, distant in patients with low cardiac output, obesity and/or COPD Strong apical impulse Narrowing of pulse pressure –later stage may have a necrotic notch on carotid upstroke Significant if area <0.8cm <sup>2</sup> /M <sup>2</sup> or mean gradient ≥ 35mmHg	Loud rough systolic diamond shaped murmur Sx: Often asx, fatigue, exertional dyspnea, Angina, CHF, syncope,dysphagia Si: BP normal; narrow pulse pressure; Cmplc: Angina, sudden death, GI-angiodysplasia with GI bleed, ischemia, Vtach/fib CHF, SBE, heart blocks EKG: normal, or LVH and strain, ST-T wave changes CXR: May show 4—chamber enlargement, valvular calcifications usually only with sig- nificant disease, LVH, poststenotic dilation of ascending aorta, pulmonary congestion	R/O bicuspid aortic valve Accentuation: Valsalva release Sudden squatting Passive leg raising Decrease: Handgrip Valsalva Standing
Aortic Insufficiency, severe rarely due to RHD, usually due to trauma, syphilis, dissecting aneurysm, endocarditis, SLE, RA, ankylosing spondylitis	$S_3$ heard over the apex, LSB at 3 <sup>rd</sup> or 4 <sup>th</sup> intercostal space Displacement of cardiac impulse downward and to the patient's left the aortic regurgitant murmur is difficult to hear if the heart rate is greater than 90 bpm	Decrescendo, blowing diastolic murmur, early systolic apical ejection murmur <b>Sx:</b> Gradual CHF Austin Flint murmur (low pitched apical dia- stolic rumble heard in severe AR), syncope, chest pain, dyspnea on exertion <b>SI:</b> widened pulse pressure, bounding pulses, head "bobbing" with each systole, "water hammer: or collapsing pulse "Corri- gan's pulse) at the wrist, capillary pulsations (Quincke's pulse) base of nail beds, "double Duroziez" a "to and fro" murmur over femoral arteries <b>EKG:</b> LVH <b>CXR:</b> LVH, aortic dilation	Accentuation: Sudden squatting Isometric handgrip Sitting up and leaning forward

MURMUR	LOCATION	CHARACTER	DIFFERENTIAL
Mitral Stenosis, most common in females age 20-40, always present in RHD to some degree,	Significant if valve diameter <1.2cm <sup>2</sup> History of and/or high risk for peripheral arterial embolus especially if Afib is present	mid-diastolic murmur with presystolic ac- centuation even in Afib; opening snap; loud S <sub>1</sub> , rumbling, Right ventricular "lift" over lower sternum <b>Si:</b> Exertional dyspnea , orthropnea, hemop- tysis due to shunts from pulmonary veins to bronchial veins, PND Atrial fibrillation <b>Cmplc:</b> CHF, SBE, emboli, pulmonary edema with exertion <b>EKG:</b> Afib, RVH, Rt Axis, LAE <b>CXR:</b> large left atrium, pulmonary artery, and right ventricle and may show valvular calcifications	Accentuation: Exercise Left lateral position Isometric handgrip Coughing After Valsalva stranding Squatting
Mitral Regurgitation, most common in females age 20-40, 70% with chronic MR in 1960s was due to RHD, 30% due to chordae rupture from MI, endocarditis and trauma	Holosystolic murmur at apex with radiation to base or left axilla and Left posterior intra- scapular area Hyperdynamic apex often with palpable left ventricular lift and apical thrill Apical early-to –middiastolic rumble In chronic mitral regurgita- tion the murmur maybe heard midial to left scapula secondary to severe left atrial enlargement	Sx: Exertional dyspnea, orthopnea, fatigue, Frank CHF, hemoptysis (caused by pulmonary hypertension) EKG: LAE, LVH, Atrial fibrillation CXR: LAE, LVE, possible pulmonary congestion	Accentuation: Sudden squatting Isometric handgrip Decrease: Valsalva Standing
<b>Tricuspid Stenosis,</b> usually accompanies mitral or aortic disease but occurs in only 2-4% of patients with RHD Can also be seen with carcinoid sydrome as an isolated finding	RV area	Diastolic, scratchy Increase during moving inspirations Jugular venous distension with failure to collapse on inspiration	Accentuation: Inspiration Passive leg raising Right lateral decubitis Decrease: Expiration
Tricuspid Insufficiency, usually accompanies mitral or aortic disease but occurs in only 2-4% patients with RHD May be present with any cause of severe pulmonary hypertension or carcinoid syndrome	Best at LLSB, occasionally best at epigastrium, at the RSB or if the RV is very large, over the mid-left thorax at the site of the usual LBV apex usually accompanied by RVS <sub>3</sub>	Pansystolic that increases with inspiration at the LLSB, (or wherever a palpable RV lift is felt-Carvallo sign)	Accentuation: Inspiration Passive leg raising By exercise By pressure over or just below the liver Decrease: Expiration

MURMUR	LOCATION	CHARACTER	DIFFERENTIAL
VSD	LLSB, Apical Frequently left chest will be mor prominent than right with larger VSDs	Loud harsh pansystolic murmur at lower left sternal border, widely transmitted over precordium. Apical diastolic rumble if large shunt loud P <sub>2</sub> if pulmonary hypertension Causes a left-to-right shunt, which in end stage disease reverses to right-to-left shunt (Eisenmenger's syndrome) with clubbing and cyanosis <b>Sx:</b> Usually asx; can develop dyspnea, fatigue, and pulmonary infections <b>Cmplc;</b> SBE, CHF, pulmonary hypertension, with Eisenmenger's 2/3 die in pregnancy <b>EKG:</b> normal unless huge left-to-right caus- ing LVH; RVH suggests fixed pulmonary hypertension and left atrial enlargement <b>CXR:</b> usually normal, large defects causing LVH and prominent pulmonary vasculature and large left atrium. Small aortic knob	
Pulmonic Stenosis Systolic murmur	ULSB, neck and back 2 <sup>nd</sup> Left interspace	P <sub>2</sub> diminished; loud ejection murmur at upper left sternal border neck and back ; ejection click, decreases with inspiration RV lift is gradient is significant <b>Sx:</b> fatigue dyspnea, cyanosis, CHF <b>Si:</b> CHF including hepatomegaly in children <b>Cmplc:</b> SBE, CHF <b>EKG:</b> normal, or LVH +strain; P pulmonale <b>CXR:</b> PA dilatation, RVH, diminished pulmonary vasculature	Accentuation: Valsalva release Decrease: Expiration
Pulmonary Regurgitation		Slight delay after the $P_2$ before any murmur is heard, even if it starts with the $P_2$ , the murmur tends to be short and rough, decrescendo	
Graham-Steell murmur- high pressure in the pulmonary artery	LSB > RSB	Diastolic decrescendo murmur heard in severe pulmonary hypertension, secondary to pulmonary regurg High pitched, often increases with inspira- tion	
IHSS (Idiopthic hypertro- phic subaortic stenosis) Genetic positive family history in 1/3 Abnormal Intraventricular septum produces dynamic aortic root obstruction and subaortic stenosis; mitral regugitation is due to an- terior mitral leaflet being distorted by the septum Admistration of inotropic drugs may cause/worsen shock and/or cardiac arrhythmias Loud systolic LSB mur- mur after inotropes given SAM (systolic ante- rior motion of the mitral leaflet)	Intolerent of decreased blood volume, & venous return, as it increases murmur intensity and outflow tract gradient	Diphasic arterial systolic pulse, best palpated in the carotid arteries Aortic murmur without radiation into neck <b>Si:</b> LVH by palpation and double PMI <b>Sx:</b> dyspnea, syncope chest pain , angina that starts when exercise stops, palpitation Cmplc:Sudden death by Vtach/fib,endocarditis Afib <b>EKG:</b> LVH, big Qs in V <sub>1-2</sub> (hypertrophied septum) Holter for malignant PVCs. Non- specific EKG changes CXR: LVH	R/O: "athlete's heart" w LV wall thickness always <16mm, rarely > 13mm, which overlaps w IHSS in men but not in women Accentuation: Valsalva strain op- posite of A.S. Standing Exercise Etoh Decrease: Handgrip Squatting Leg elevation

MURMUR	LOCATION	CHARACTER	DIFFERENTIAL
Coarctation of the aorta Associated with PDA, bicuspid aortic valve	Left upper back or pulmonic area	Systolic murmur Sx: CHF, leg pains, & fatigue, headaches- rarely in childhood; CHF in 3 <sup>rd</sup> -4 <sup>th</sup> decades Si: hypertension in arms, normal or low BP in legs; decreased/delayed femoral pulses compared to radial pulses Cmplc: SBE, intracranial bleeding, hyperten- sive encephalopathy; ruptured/dissected aorta; hypertensive cardiovascular disease Risk of aortic dissection remains even after repair EKG: normal, or LVH; RVH, suggests a PDA beyond the coarc CXR: normal heart, and pulmonary vascula- ture; coarc visible on plain chest occasionally "notch" in aortic root shadow; rib notching in older patients	
PDA (Patent Ductus Arteriosus)	Left base	"Machinery" murmur or Gibson murmur or "to & fro" murmur L-to-R shunt, which in end-stage disease reverses to R-to-L shunt (Eisenmenger's syndrome) with clubbing and cyanosis; bounding pulses indicate wide pulse pressure; pink fingers, blue toes with Eisenmenger's <b>Cmplc:</b> SBE, CHF, pulmonary hypertension; with Eisenmenger's, 2/3 die in pregnancy <b>EKG:</b> normal; occasionally LVH: if has RVH, suggest fixed pulmonary hypertension <b>CXR:</b> Normal, with small PDA; or w larger one, LVH, enlarged aortic knob with dilatation of the proximal relative to the distal aorta, and a large LA with prominent vasculature	R/O : coronary AV fistula, Ruptured sinus of Valsalva, Coarctation of aorta AS/AR
ASD(Atrial Septal Defects) Primum: AV canal de- fects including low ASD, high VSD, cleft mitral and/or tricuspid valves or a common AV valve Increased risk in Down's Syndrome	Apical	Harsh systolic murmur , pansystolic from MR (33%), TI or VSD (75%) P <sub>2</sub> increased; Fixed Split S <sub>2</sub> ; <b>Si:</b> cyanosis mild or absent, CHF, RVH <b>Sx:</b> dyspnea, fatigue, CHF, pulmonary infection <b>Cmplc:</b> CHF, pulmonary hypertension, SVT arrhythmias, SBE rarely, Increased incidence of rheumatic fever <b>EKG:</b> L axis, pRBBB, R and LVH, counter- clockwise rotation <b>CXR:</b> Small aortic knob; RVH and increase pulmonary flow, if MR, then large left atrium and LV; "gooseneck deformity" of LV out- flow tract on angiography; serrated MV leaf	

MURMUR	LOCATION	CHARACTER	DIFFERENTIAL
MURMUR ASD: Secundum: Mid or upper ASD allows left to right shunt	LOCATION Upper left stermal border Lower left sternal border	Mild systolic murmur mid-diastolic rumble form high flow S <sub>2</sub> wide and fixed; RVH <b>Sx:</b> rarely in infancy or childhood ; later dyspnea w CHF and pulmonary HT, palpita- tions if arrhythmias	<b>DIFFERENTIAL</b> Increased risk of atrial arrhythmias
		Cmplc: CHF and Pulmonary HT late <b>EKG:</b> axis normal or RAD, partial and full RBBB, RVH and strain; clockwise rotation <b>CXR:</b> small aortic knob, RVH+ increased pulmonary flow; no LA enlargement unlike patent ductus or VSD	

Reviewed and edited by: Daniel Mathers, M.D., FACC Cardiology, UNMC

References

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- 2. Onion, D.K., M.D., MPH, FACP. (1998). *The Little Black Book of Primary Care–Pearls and References. (3rd ed.)* pp. 90-100, 704). Massachusetts: Blackwell Science, Inc.
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# **READING AN EKG**

RATE: 300 / (# Large squares from R to R) **RHYTHM:** Is every QRS preceded by P-wave? Is the rhythm regular? MEAN QRS VECTOR: Normal: -30 to +90 (degrees) LAD: < - 30 RAD: >+90 P-WAVE VECTOR: +30 to +60 T-WAVE VECTOR: Within 45 of QRS INTERVALS (normals): sec (ms) (Note: 1 small square = 0.04 sec) PR interval: 0.12-0.20 (120-200) ORS duration: < 0.12 (<120) QT interval: Varies with HR **INTERVAL** RATE 125 <0.25 Sec <0.35 Sec 75 45 <0.45 Sec **QRS NOMENCLATURE** Q-wave = first negative deflection before positive R-wave = any positive deflection

S-wave = first negative after positive

#### NORMAL EKG:

P-waves upright in I, II, V2-V6 T-waves upright in I, II, V3-V6 inverted in aVR variable in III, aVL, aVF, V1, V2 Small Q-wave normal in I, aVL Deep Q-wave (QS) normal in aVR, and occasionally seen in III, V1, V2

#### **BASIC EKG ABNORMALITIES**

#### **PR INTERVAL:**

<0.12 seconds: -normal in tachycardia -junctional (nodal) rhythm -"pre-excitation": Wolff- Parkinson-White syndrome ("delta-waves" prolong the QRS) >0.20 seconds (1st degree AV block): -focal fibrosis -digitalis -ischemic heart disease -rheumatic heart disease -hyperkalemia

#### **QRS DURATION: >0.12 SECONDS**

-Bundle branch blocks (QRS > or = 0.12) -Intraventricular conduction delay (IVCD) -LVH -Hyperkalemia -Procainamide, quinidine -Wolf-Parkinson-White syndrome

#### **QT INTERVAL:**

Long QT:

-cardiac depressants (i.e., quinidine) -tricyclic antidepressants -ischemic heart disease -hypokalemia, hypocalcemia, alkalosis -bundle branch block -stroke, coma -ventricular hypertrophy Shortened QT: -hypercalcemia -digitalis

#### **P-WAVE ABNORMALITIES:**

Tall peaked P-waves (amplitude > or = 3 mV) -usually largest in lead II -suggests Right Atrial Abnormality (RAA)(enlargement) -often seen in COPD Broad, notched P-waves > or = 0.12 sec -suggests Left Atrial Abnormality (LAA) (enlargement) -often seen in Mitral Valve Disease Biphasic P-wave in lead VI -may be normal -initial deflection > terminal deflection suggests RAA -terminal deflection > initial deflection suggest LAA

#### **QRS COMPLEX:**

Low Amplitude -obesity -COPD -effusions -- pleural or pericardial -old age -- especially after MI's -hypothyroidism -pneumothorax -primary cardiomyopathy

Tall QRS

-ventricular hypertrophy -bundle branch block -normal for age <35

#### **Poor R-wave progression**

-lead placement -clockwise rotation (normal variant) -anteroseptal MI -LVH, RVH -LBBB, LAFB, IVCD -COPD Q-waves (see MYOCARDIAL INFARCTION)

-must be > 0.04 sec and/or at least 1/3 the amplitude of the R-wave to be significant

#### **ST SEGMENT:**

Elevated ST segment

-acute MI (usually focal)

-pericarditis (diffuse)

-ventricular aneurysm (persistent)

-atypical angina (Prinzmetal)

-may be normal variant (early repolarization)

Depressed ST segment

-ischemia/angina pectoris

-digitalis ("scooped out" ST's)

-ventricular hypertrophy-if down sloping called "strain"

-bundle branch block

-hypokalemia/hypomagnesemia

#### **T-WAVE CHANGES:**

Tall peaked T-waves (5 small boxes in limb leads or 10 small boxes in chest leads)

-hyperkalemia

-hyperacute MI

-normal variant in young

Inverted T-waves

-anything causing ST-depression

-digitalis

-pericarditis-begins upright and flips

-normal variant-persistent juvenile

pattern in 10% blacks and children

U-Waves

-from repolarization of papillary muscle -seen most in precordial leads -hypokalemia/hypomagnesemia

-most often normal variant

#### LEFT AXIS DEVIATION: (-30 or >)

-LVH

-Left anterior fascicular block

-LBBB

-Inferior MI

#### **RIGHT AXIS DEVIATION:** (+90 or >)

--MI (lateral)

-RVH

-RBBB

-Left posterior fascicular block (rare)

-COPD

-Acute PE

#### **COMMON CRITERIA FOR EKG DIAGNOSES**

#### RIGHT ATRIAL HYPERTROPHY

-"Peaked" P-waves > 2.5 mV in II, III, or aVF -Biphasic P-wave in lead VI with positive > negative

#### LEFT ATRIAL HYPERTROPHY

-Long P-wave > 0.12 sec in any lead (primarily in lead II) -Biphasic P-wave in lead VI with negative > positive -Double peaked P-wave in I, II, aVL, V4-V6

#### LEFT VENTRICULAR HYPERTROPHY (criteria not valid if patient is younger than 35 years old)

-Axis > -15 degrees (nonspecific)
-S in VI or V2 + R in V5 or V6 > 35 mV
-R in V5 or V6 > 26 mV
-R in aVL > 12 mm
-"Strain": ST depression, often with flipped T's in I, II, aVL, V5, V6

#### RIGHT VENTRICULAR HYPERTROPHY

-R > S in V1 and S > R in V6 -Axis > +110 degrees -"Strain": ST depression, often with flipped T's in V1-V4

#### RIGHT BUNDLE BRANCH BLOCK (RBBB)

-Prolonged QRS > or = 0.12 sec

-Axis normal or rightward

(if left axis deviation present, then must have both RBBB and left anterior fascicular block)

-rSR' pattern in V1, V2 "mutant rabbit ears" -Terminal (last 0.04 sec) S-wave in V5, V6 -ST depression and T-wave inversion in V1, V2

#### LEFT BUNDLE BRANCH BLOCK (LBBB)

-Prolonged QRS > or = 0.12 sec
-Axis normal or leftward
-Poor R-wave progression. May have rS or large Q wave in V1, V2, V3, and V4
-Terminal QRS forces (last 0.04 sec) positive in V5 and V6 (pure positive QRS complex in V6)
-T-wave inversion, ST depression & elevation in most leads. (Often exaggerated)

#### LEFT ANTERIOR FASCICULAR BLOCK (Hemiblock)

-Axis more negative than -45 degrees -No other cause of axis deviation present -Normal QRS duration (.10-.11) -Small Q in I, small R in III (Q1R3 pattern)

#### LEFT POSTERIOR FASCICULAR BLOCK (Hemiblock)

-Axis more positive than +110 degrees -No other cause of axis deviation present -Normal QRS duration -Small Q in III, small R in I (Q3R1 pattern)

#### MYOCARDIAL INFARCTION

Progression of changes

-hyperacute (min-hrs): ST evaluation and high "peaked" T-waves
-acute MI (hrs): ST drops but still elevated, T-wave inversion
-Q-waves develop in hours to days
-recent MI (weeks-months): ST returns baseline, T-waves inverted for

months to years, and Q-waves remain

-old MI (months-years): Q-waves

#### Significant Q waves: (must meet one of two criteria)

1) Q wave must be 1/4 (1/3) the size of the R wave to be considered significant or

2) Q wave is .04 seconds wide (one small box) or greater to be considered significant

#### **Definitions of Transmural (Q-wave) Infarctions**

-septal	V1-V2
-anterior	V2 -V3, V4
-anteroseptal	V1 - V3, V4
-high lateral	I, aVL
-anterolateral	V5 - V6, I, aVL
-extensive anterior	V1 - V5, V6
-inferior	II, III, aVF
-inferolateral	II, III, aVF, V5 - V6
-posterior	R-wave V1 - V2 with ST depression
-right ventricular	rV3 -rV4 with ST depression

#### NOTE:

-EKG changes in only 80% with MI -Inferior MI's commonly result in a BBB

-Q-waves disappear in 20% of patients who had MI

#### PERICARDITIS

-Diffuse ST elevation (except aVR)

-No reciprocal ST depression

-As pericarditis subsides, ST returns to baseline and T-waves invert

#### WOLFF-PARKINSON-WHITE SYNDROME (WPW)

-Is considered the Great Mimic. Tends to mimic many other ECG conditions. Is fairly uncommon (2 per 1000) but occurs frequently enough to cause problems for the unwary. -Short PR interval

-QRS widening

-Presence of delta waves

-Patients with WPW are highly susceptible to certain cardiac arrhythmias. If suspect WPW, do not use digoxin, verapamil or diltiazem.

#### DIGITALIS

-Digitalis effect: (the degree of changes has no consistent relation to the amount of digitalis admin.)

-"scooped" out ST depression

-biphasic T-wave (may show decreased amplitude)

-shortening of QT

-prolonged PR

Digitalis toxicity:

-all the above, plus

-excitatory effects: Digitalis toxicity is known to be capable of producing almost all types of cardiac arrhythmias, except atrial flutter and BBB.

(i.e., PVC's, PAT with block), V-Tach, V-Fib, etc.)

-suppressant effects:

-sinus bradycardia, SA block, AV blocks

QUINIDINE (similar effects with other-antiarrhythmics)

Therapeutic effects

-Prolonged QT interval

-ST depression

-T-waves depressed, widened, notched, inverted

-Prominent  $\overline{U}$  waves

#### Toxic effects

-QRS prolongation

-Various degrees of AV blocks or marked sinus bradycardia, sinus arrest or SA blocks

-Various Ventricular rhythms and sudden death

#### HYPERKALEMIA

-K < 7.5 mEq/L:

-decreased amplitude of P-waves
-wide QRS (Intraventricular conduction defect)
-Tall, narrow, and peaked T-waves
-K > 7.5 mEq/L:
-Absence of P-waves
-"sine wave" R-S-T pattern (sinoventricular rhythm)

#### HYPOKALEMIA

-ST depression. Decreased T-wave amplitude (or inversion),

-Prominent U-waves and P-waves

-Prolongation of the QRS duration

-Prolonged QT interval

#### HYPERCALCEMIA

-Short QT interval (short ST)

HYPOCALCEMIA

-Prolonged QT interval (long ST)

*Prepared by* Darwin Brown, PA-C and Charles Seelig, MD. 3/2003 update

# CLINICAL ENTITIES THAT HAVE BEEN ASSOCIATED WITH ELEVATED C-TROPONIN-1 LEVELS

**Myocarditis** Cardiomyopathy Congestive heart Failure Sepsis Pulmonary Embolism Rhabdomyolysis **Chest Contusion** Emboli caused by endocarditis Mural Thrombi Prosthetic valves Neoplasms Inflammatory processes, including viral infections such as with coxsackie B Radiation-induced coronary stenosis Congenital abnormalities in a coronary artery Hurler's syndrome Homocystinuria Rheumatoid arthritis Systemic lupus erythematosus Marathon runners Cocaine abusers MI Angina Cardiac Surgery Specimens from patient who have received preparations of mouse monoclonal antibodies for diagnosis or therapy may contain human anti-mouse antibodies

#### References

- 1. Quillen, D.M., M.D., (2003). Troponin-I Elevation in Noncardiac Conditions. *Medscape Primary Care*. 11/13/2002. Available at: http://www.medscape.com/viewarticle/444137
- Abbott AXSYM System. (2001) Troponin-1, List No. 3C29, 69=5606/R5 (Package Insert), Abbott Laboratories, Diagnostics Division, Abbott Park, Illinois.

"The impact of congestive heart failure in perioperative morbidity and mortality is often minimized. Currently 2.3\* million Americans have CHF with 400,000 added each year. The mortality rate for CHF is between 15 to 60% with 200,000 deaths each year."

Barash. Perioperative evaluations of the cardiac patient for noncardiac surgery. *Can J Anesth.* 1991/38:4/PPR/34-R/39

\* 5 million. 2006 American College of Cardiology

http://www.acc.org/media/patient/chd/chf/htm

# Criteria for NYHA Functional Classification for CHF Patients\* Functional Capacity

#### Class I

No limitation of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, or dyspnea.

#### Class II

Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in fatigue, palpitation, or dyspnea.

#### Class III

Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity causes fatigue, palpitation, or dyspnea.

#### Class IV

Unable to carry on any physical activity without discomfort. Symptoms of cardiac insufficiency may be present even at rest. If any physical activity is undertaken, discomfort is increased.

\*The Criteria Committee of the New York Heart Association: Nomenclature and Criteria for Diagnosis of the Heart and Great Vessels, 8th ed. Boston: Little Brown & Co., 1979.

# Pathology-Unstable Angina

Clinical Definition:

- 1. New onset of angina occurring at rest or low levels of exercise. or
- 2. A sudden increase in frequency and severity of previously stable angina. or
- 3. Recurrent rest angina or an episode of prolonged ischemia pain without subsequent evidence of myocardial necrosis.

Apparently, the event initiating all acute ischemia syndromes is the development of plaque fissuring, fracture, ulceration, or rupture.

Unstable angina is to be differentiated from Prinzmetal's angina, in that in the former, more severe underlying coronary artery disease is present, and vasospasm is just one (generally within a continuum) of the pathophysiological mechanisms promoting recurrent ischemia. In unstable angina, pain can be associated with ST segment elevation on the ECG (as in Prinzmetal's), but exercise capacity would be preserved because of high-grade underlying coronary artery stenosis in one or more coronary vessels.

# **Natriuretic Peptides in Congestive Heart Failure**

Three major natriuretic peptides are under study for use in the neurohumoral profiling in congestive heart failure. Atrial (A-type) natriuretic peptide (ANP) and BNP which are of myocardial cell origin and C-type natriuretic peptides which are of endothelial origin.<sup>3</sup> BNP is secreted from the ventricles in response to volume expansion and pressure overload; some BNP is released from the atrial tissue. ANP major storage sites include both the atria and ventricles.

BNP levels are being used to assess clinical status and predict prognosis of patients in congestive heart failure. BNP levels correlate with hemodynamic parameters such as right atrial pressure (RAP), PCWP, and left ventricular end diastolic pressures. Studies have compared BNP measurements with echocardiogram as a reference diagnostic standard in appropriate patients with New York Heart Association classes I-IV heart failure.<sup>1</sup>

NYHA CLASS	Mean ± SD (ng/L)	True Normal <80
Ι	240±290	1-300
II	390±370	300-600
III	640±450	600-900
IV	820±440	>900-1000

**BPN** concentrations with increasing CHF severity

(UNMC Daniel Mathers, M.D., FACC)

#### **Interpretation of Levels of BNP:**

- A. No CHF: 9 pg/ml (Median BNP)
- B. CHF
  - 1. CHF Diagnosis: >100 pg/ml (standard cutoff)
  - 2. NYHA Class I CHF: Median BNP 83 pg/ml (49-137)
  - 3. NYHA Class II CHF: Median BNP 235 pg/ml (137-391)
  - 4. NYHA Class III CHF: Median BNP 459 pg/ml (200-871)
  - 5. NYHA Class IV CHF: Median BNP 1119 pg/ml (>728)<sub>2\*</sub>

Causes of increased BNP level are congestive heart failure, left ventricular hypertrophy, cardiac inflammation (myocarditis, cardiac allograft rejection), Kawasaki disease, primary pulmonary hypertension, renal failure, ascitic cirrhosis, primary hyperaldosteronism, Cushing syndrome<sup>2\*</sup>, Pulmonary embolism, increased C-Reactive Protein and septic shock.

Patients with lung disease may have higher levels of BNP than patients without lung disease in part because many patients with end stage pulmonary disorders have concomitant RV dysfunction another BNP trigger.<sup>3\*</sup>

1

2

*B-type natriuretic peptide (BNP, for CHF {Mar 2004; 121-6}* <u>http://www.jr2.ox.ac.uk/bandolier/band121/b121-6.html</u>

Family Practice notebook.com, *a Family Medicine Resource* <u>http://www.fpnotebook.com/CV140.htm</u>

looksmart, Maisel, Alan S., Measuring BNP levels in the diagnosis and treatment of CHF: an evolving tool proves invaluable when time is of the essence http://www.looksmart.com/ http://www.findarticles.com/ Journal of Critical Illness>Nov, 2002

Reviewed and edited by: Daniel Mathers, M.D., FACC Cardiology *UNMC* 

#### Syndromes with Associated Cardiovascular Involvement

Wilson, et al. Congenital Heart Disease, Harrison's Principles of Internal Medicine, 12th ed., chapt. 186, pp. 924–925.

Syndrome	Major cardiovascular manifestations	Major noncardiac abnormalities		
HERITABLE AND POSSIBLY HERITABLE				
Ellis-van Creveld	Single atrium or atrial septal defect	Chondrodystrophic dwarfism, nail dysphasia, poly- dactyly		
TAR (thrombocytopenia-absent radius)	Atrial septal defect, tetralogy of Fallot	Radial aplasia or hypoplasia, thrombocytopenia		
Holt-Oram	Atrial septal defect (other defects common)	Skeletal upper limb defect, hypoplasia of clavicles		
Kartagener	Dextrocardia	Situs inversus, sinusitis, bronchiectasis		
Laurence-Moon-Biedl	Variable defects	Retinal pigmentation, obesity, polydactyly		
Noonan	Pulmonary valve dysplasia, cardiomyopa- thy (usually hypertrophic)	Webbed neck, pectus excavatum, cryptorchidism		
Tuberous sclerosis	Rhabdomyoma, cardiomyopathy	Phakomatosis, bone lesions, hamartomatous skin lesions		
Multiple lentigines (leopard) syndrome	Pulmonic stenosis	Basal cell nevi, broad facies, rib anomalies		
Rubenstein-Taybi	Patent ductus arteriosus (others)	Broad thumbs and toes, hypoplastic maxilla, slanted palpebral fissures		
Familial deafness	Arrhythmias, sudden death	Sensorineural deafness		
Osler-Rendu-Weber	Arteriovenoius fistulas (lung, liver, mucous membranes)	Multiple telangiectasia		
Apert	Ventricular septal defect	Craniosynostosis, midfacial hypoplasia, syndactyly		
Incontinentia pigmenti	Patent ductus arteriosus	Irregular pigmented skin lesions, patchy alopecia, hypodontia		
Alagille (arteriohepatic dyspla- sia)	Peripheral pulmonic stenosis, pulmonic stenosis	Biliary hypoplasia, vertebral anomalies, prominent forehead, deep-set eyes		
DiGeorge	Interrupted aortic arch, tetralogy of Fallot, truncus arteriosus	Thymic hypoplasia or aplasia, parathyroid aplasia or hypoplasia, ear anomalies		
Friedreich's ataxia	Cardiomyopathy and conduction defects	Ataxia, speech defect, degeneration of spinal cord dorsal columns		
Muscular dystrophy	Cardiomyopathy	Pseudohypertrophy of calf muscles, weakness of trunk and proximal limb muscles		
Cystic fibrosis	Cor pulmonale	Pancreatic insufficiency, malabsorption, chronic lung disease		
Sickle cell anemia	Cardiomyopathy, mitral regurgitation	Hemoglobin SS		
Conradi-Hünermann	Ventricular septal defect, patent ductus arteriosus	Asymmetric limb shortness, early punctate mineraliza- tion, large skin pores		

Cockayne	Accelerated atherosclerosis	Cachectic dwarfism, retinal pigment abnormalities, photosensitivity dermatitis
Progeria	Accelerated atherosclerosis	Premature aging, alopecia, atrophy of subcutaneous fat, skeletal hypoplasia
Connective Tissue Disorders		
Cutis laxa	Peripheral pulmonic stenosis	Generalized disruption of elastic fibers, diminished skin resilience, hernias
Ehlers-Danlos	Arterial dilatation and rupture, mitral regurgitation	Hyperextensible joints, hyperelastic and friable skin
Marfan	Aortic dilatation, aortic and mitral incom- petence	Gracile habitus, arachnodactyly with hyperextensibil- ity, lens subluxation
Osteogenesis imperfecta	Aortic incompetence	Fragile bones, blue sclera
Pseudoxanthoma elasticum	Peripheral and coronary arterial disease	Degeneration of elastic fibers in skin, retinal angioid streaks
INBORN ERRORS OF METABOLISM		
Pompe's disease	Glycogen storage disease of heart	Acid maltase deficiency, muscular weakness
Homocystinuria	Aortic and pulmonary arterial dilatation, intravascular thrombosis	Cystathionine synthetase deficiency, lens subluxation, osteoporosis
Mucopolysaccharidosis: Hurler, Hunter	Multivalvular and coronary and great artery disease, cardiomyopathy	<ul> <li>Hurler: Deficiency of α-L-iduronidase, corneal clouding, coarse features, growth and mental retardation.</li> <li>Hunter: Deficiency of L-iduranosulfate sulfatase, coarse facies, clear cornea, growth and mental retardation</li> </ul>
Morquio, Scheie, Maroteaux- Lamy	Aortic incompetence	Morquio: Deficiency of <i>N</i> -acetylhexosamine sulfate sulfatase, cloudy cornea, normal intelligence, severe bony changes involving vertebrae and epiphyses.
		Scheie: Deficiency of α-iduronidase, cloudy cornea, normal intelligence, peculiar facies.
		Maroteaux-Lamy: Deficiency of arylfulfatase B, cloudy cornea, osseous changes, normal intel- ligence
CHROMOSOMAL ABNORMALITIES		
Trisomy 21 (Down's syndrome)	Endocardial cushion defect, atrial or ventricular septal defect, tetralogy of Fallot	Hypotonia, hyperextensible joints, mongoloid facies, mental retardation
Trisomy 13 (D)	Ventricular septal defect, patent ductus arteriosus, double-outlet right ventricle	Single midline intracerebral ventricle with midfacial defects, polydactyly, nail changes, mental retarda- tion

Trisomy 18 (E)	Congenital polyvalvular dysplasia, ven- tricular septal defect, patient ductus arteriosus	Clenched hand, short sternum, low-arch dermal-ridge pattern on fingertips, mental retardation
Cri-du-chat (short-arm deletion-5)	Ventricular septal defect	Cat cry, microcephaly, antimongoloid slant of palpe- bral fissures, mental retardation
XO (Turner)	Coarctation of aorta, bicuspid aortic valve	Short female, broad chest, lymphedema, webbed neck
XXXY and XXXXX	Patient ductus arteriosus	XXXY: hypogenitalism, mental retardation, radial- ulnar synostosis XXXX: small hands, incurving of fifth fingers, mental retardation
Sporadic Disorders		
VATER association	Ventricular septal defect	Vertebral anomalies, anal atresia, tracheoesophageal fistula, radial and renal anomalies
CHARGE association	Tetralogy of Fallot (other defects common)	Colobomas, choanal atresia, mental and growth defi- ciency, genital and ear anomalies
Williams	Supravalvular aortic stenosis, peripheral pulmonic stenosis	Mental deficiency, "elfin" facies, loquacious personal- ity, hoarse voice
Cornelia de Lange	Ventricular septal defect	Micromelia, synophyrs, mental and growth deficiency
Shprintzen (velocardiofacial)	Ventricular septal defect, tetralogy of Fal- lot, right aortic arch	Cleft palate, prominent nose, slender hands, learning disability
TERATOGENIC DISORDERS		
Rubella	Patent ductus arteriosus, pulmonic valvular and/or arterial stenosis, atrial septal defect	Cataracts, deafness, microcephaly
Alcohol-induced	Ventricular septal defect (other defects)	Microcephaly, growth and mental deficiency, short palpebral fissures, smooth philtrum, thin upper lip
Phenytoin-induced	Pulmonic stenosis, aortic stenosis, coarcta- tion, patient ductus arteriosus	Hypertelorism, growth and mental deficiency, short phalanges, bowed upper lip
Thalidomide-induced	Variable	Phocomelia
Lithium-induced	Ebstein's anomaly, tricuspid atresia	None

#### **Down's Syndrome**

Common Surgical Procedures:

- cardiac malformations
- congenital duodenal obstructions
- strabismus
- congenital cataract or glaucoma
- kyphoscoliosis
- dental care

#### Congenital Heart Disease (40–50% DS)

- tetralogy of Fallot, 8%
- cardiac cushion defect, 40–50%
- atrioventricularis communis
- persistent PDA, 12%

DS patients may have:

- immunological deficiency
- adrenal response to ACTH may be subnormal
- thyroid hypofunction
- leukemia
- epilepsy or senile dementia

#### Skeletal abnormalities associated with DS

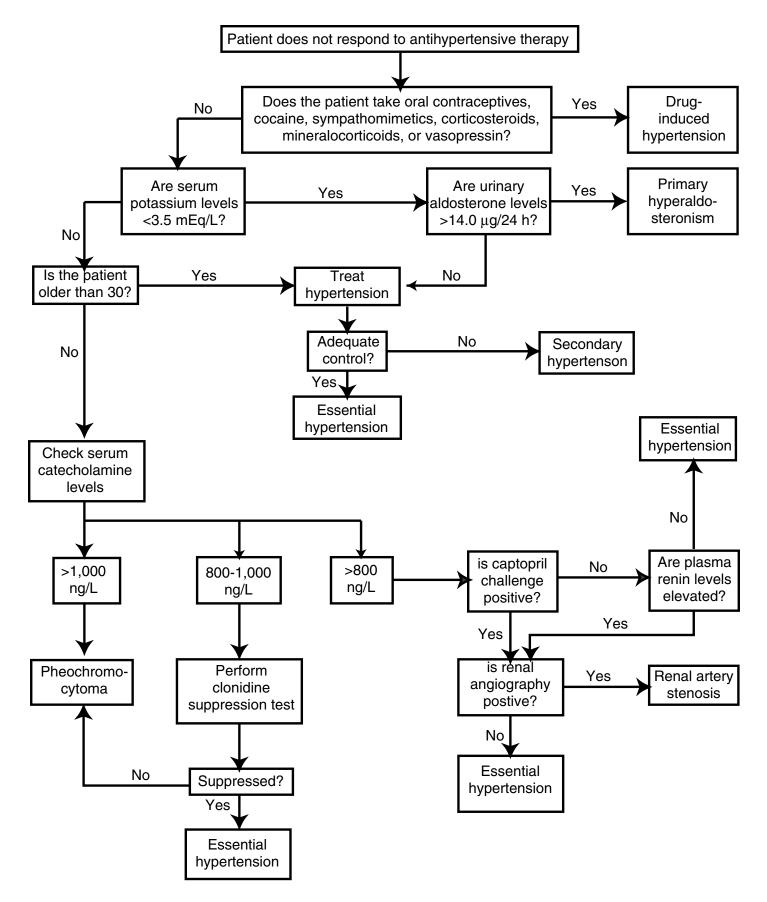
- scoliosis
- 15–20% instability of the atlantoaxial joints (asymptomatic in the majority of patients)
- hypoplastic facial bones
- maxilla and mandible are small, resulting in protruding tongue—unusually large
- high arched palate
- abnormal dentition: numbers, shape, location
- narrow nasopharynx
- tonsils and adenoids that are large
- larynx may be small
- increased incidence of subglottic stenosis
- increased frequency of laryngospasm
- chronic upper airway obstruction which may lead to arterial hypoxemia
- microcephaly

#### The endotracheal tube required may need to be smaller than predicted by age or height.

Pre-op evaluation should include:

- lateral neck in flexion and extension to assess atlantoaxial instability
- ECHO
  - patient with known cardiac lesion or audible murmur >20 years old who has not had an ECHO within the last 3–5 yr (results of ECHO must be available at time of pre-op workup)
- SBE coverage
- Consider need for possible
  - -steroids
  - -thyroid studies

Robert K. Stoelting, Stephen F. Dierdort, *Anesthesia and Co-Existing Disease*, 3rd ed. New York: Churchill Livingston Inc. 1993, p. 602.



Modified with permission from Healely PM, Jacobson EJ, Common medical diagnoses: An algorithmic approach. Philadelphia, Pa: WB Saunders Company. 1994

Figure 1 Does the patient have truly resistant hypertension?

Family Practice Recertification Vol. 19, No. 1, January 1997

### The Five H's: Hypertension, Hypermetabolism, Hyperhydrosis, Headache, & Hyperglycemia

In 5% of patients with hypertension there is a secondary cause such as renovascular hypertension, primary aldosteronism, and pheochromocytoma. Approximately 36,000 patients in the U.S., or from 0.09% to 2.2% of all patients with sustained hypertension, have a pheochromocytoma:

- •90% are found in the adrenal medulla (R > L), and about 10% bilaterally.
- a small percentage are found in extra-adrenal sites (especially in children).
- primarily found within the ages of 30–50 years old, with approximately 1/3 of the cases occurring in children (males > females). 10% family history for pheochromocytoma.
- associated with multiple endocrine neoplasia (MEN).

#### Table 1. Manifestations of Multiple Endocrine Neoplasia (MEN)

Syndrome	Manifestations
MEN type IIa (Sipple Syndrome)	Medullary thyroid cancer Parathyroid adenoma Pheochromocytoma
MEN type IIB	Medullary thyroid cancer Mucosal adenomas Marfan appearance Pheochromocytoma
von Hippel-Lindau Syndrome	Hemangioblastoma involving the central nervous system Pheochromocytoma

#### Table 2. Features of Pheochromocytoma

Severe headache Palpitations Weakness, weight loss Tachycardia (rarely brady) Hypertension, sustained or paroxysmal Association conditions such as neurofibromatosis, thyroid swelling Episodic sweating Anxiety, tremulousness Pallor (rarely, flushing) Orthostatic hypotension Paradoxical blood pressure response to drugs, anesthesia

More than 95% of patients have HA's, hyperhydrosis, and palpitations FLUSHING IS RARE in pheochromocytoma.

### Table 3. Differential Diagnosis of pheo's

Anxiety, psychosis Paroxysmal cardiac arrhythmias Autonomic insufficiency Hyperdynamic circulatory states Migraine, cluster headaches Coronary artery disease, heart disease Labile essential hypertension autonomic hyperreflexia in quadriplegic persons Hypertension induced by drugs or drug/food interaction Hyperthyroidism Diabetes Hypoglycemia Eclampsia Neuroblastoma Lead intoxication Porphyria Carcinoid Hypertension from sudden drug withdrawal

#### **Triggering Factors**

Exercise, vigorous abdominal palpations, gravid uterus, bladder distention, constipation, trauma, drugs: tricyclic antidepressants, phenothiazines, histamines, glucagon, epinephrine,  $\beta$ -blockers, angiotensin II analogs, metoclopramide, nicotine.

**Nearly 50% of deaths** due to unsuspected pheochromocytomas occur during anesthesia and surgery or parturition. Time of significant intra-operative concerns: 1) during intubation, 2) during manipulation of tumor, 3) after ligation of the tumor's venous drainage.

**Work-ups:** metanephrine levels; VMA; clonidine suppression test; CXR to include oblique, CT scan, adrenal scintigraphy, arteriography, and venous sampling.

Patients should be treated 7–10 days prior to procedures with drugs such as phenoxybenzamine or metryzosine and generally given IV fluids 2–3 days prior to surgery.

C. Venkata, S. Ram, M.D. Hypertension—When to suspect underlying pheochromocytoma or aldosteronism. *Consultant*. Jan. 1996, pp. 147–153.

### **Ischemic Heart Disease**

Approximately one third of the 25 million patients in the U.S. who are undergoing surgery each year are at high risk for ischemic heart disease (IHD). Since a patient may be asymptomatic despite 50–70% stenosis of a major coronary artery, acute myocardial infarction and sudden death may be the first indication of IHD (more than 50% of victims of sudden death have unexpected IHD).

#### **Risk Status on Presence of CHD Risk Factors Other than LDL-cholesterol**

#### **Positive Risk Factors**

#### 1. Age

Male ≥45 years

Female  $\geq$  55 years or premature menopause without estrogen replacement therapy

#### 2. Family history of premature CHD

Definite myocardial infarction or sudden death before 55 years of age in father or other male first degree relative, or before 65 years of age in mother or other female first degree relative.

#### 3. Current cigarette smoking

It is estimated that cigarette smoking is responsible for 30% of the 500,000 annual fatalities attributed to IHD. The addition of the risk factor of cigarette smoking is equivalent to increasing plasma cholesterol concentration 50-100 mg/dl.

"According the American Cancer Society, within just 20 minutes of your last cigarette, your blood pressure and pulse rate drop to normal levels. Within 48 hours, your ability to smell and taste are enhanced. After 2 to 12 weeks, circulation improves and lung function increases by up to 30%. Breathing becomes noticeably easier. Within a year, the risk of heart disease drops to half that of a current smoker. At the 5-year mark, the death rate from lung cancer for the average former pack-a-day smoker decreases by almost 50%. By 10 years, the death rate from lung cancer is similar to that of nonsmokers. At 15 years, the risk of heart disease is the same as that of a nonsmoker."

#### 4. Hypertension

 $\geq$ 140/90 mm Hg or on antihypertensive medications

"Commonly, hypertension on admission to the hospital is regarded as a "normal" response to the stress of hospital admission. However, this group of patients may represent an untreated or inadequately managed subset of hypertensive patients. A study examining this problem revealed that these patients generated the highest mean arterial BP (MAP) in response to laryngoscopy and intubation. In addition, myocardial ischemia was observed and 75% of the patients in this group required vasodilator therapy. In contrast, those patients with normal admission BP, or adequately treated hypertension, had an uneventful per-induction course.

In the perioperative period, uncontrolled or poorly controlled hypertension is associated with an increased incidence of ischemia, myocardial infarction, dysrhythmias, and stroke. Adequate preoperative treatment is associated with a reduced incidence of serious cardiovascular complications."

	Blood pressure (mm Hg)		
Category	Systolic	Diastolic	
Normal*	<130	<85	
High normal	130–139	85–89	
Hypertension†			
Stage 1 (mild)	140–159	90–99	
Stage 2 (moderate)	160–179	100–109	
Stage 3 (severe)	180-209	110–119	
Stage 4 (very severe)	>210	>120	

### **Classifying Blood Pressure in Adults**

\*Regarding cardiovascular risk, optimal BP is a systolic measurement of <120 mm Hg and a diastolic measurement of <80 mm Hg. Unusually low readings should, however, be evaluated for clinical significance.

<sup>†</sup>Based on the average of 2 or more readings that have been taken at each of two or more visits after initial screening

NOTE: *Adult* refers to persons 18 years and older. Also, this classification is limited to persons who are neither taking antihypertensive drugs nor acutely ill. To determine BP status when systolic and diastolic BP fall into different categories, assign the patient to the category in which the higher measurement falls (e.g., 160/ mm Hg is stage 2; 180–120 mm Hg is stage 4). Isolated systolic hypertension (ISH) is defined as systolic BP of >140 mm Hg and diastolic BP <90 mm Hg (e.g., 170/85 mm Hg is classified as stage 2 ISH).

In addition to classifying stages of hypertension based on average BP levels, the clinician should specify the presence or absence of target-organ disease and other risk factors. A patient who has diabetes, BP of 142/94 mm Hg, and left ventricular hypertrophy, for example, should be classified as stage 1 hypertension with target-organ disease (left ventricular hypertrophy) and with an additional major risk factor (diabetes). This specificity is important for calculating risk and for management.

Stage 1 Hypertension: Treatment Considerations and Realities. JAAPA, January 1996, pp. 28-38.

### **Causes of Secondary Hypertension**

- I. Renal disease
  - A. Parenchymal disease
    - 1. Chronic pyelonephritis
    - 2. Glomerulonephritis (acute and chronic)
    - 3. Nephrolithiasis
    - 4. Polycystic kidney disease
  - B. Renal artery stenosis (renovascular hypertension)
  - C. Renin-producing tumors
- II. Endocrinologic diseases
  - A. Primary hyperaldosteronism
  - B. Cushing's syndrome or disease
  - C. Pheochromocytoma
  - D. Hyperthyroidism
  - E. Congenital or hereditary adrenogenital syndromes
  - F. Myxedema
  - G. Acromegaly
  - H. Hyperparathyroidism
- III. Coarctation of the aorta
- IV. Substance abuse
  - A. Cocaine
  - B. Alcohol
  - C. Other stimulants (amphetamines)
- V. Drugs
  - A. Oral contraceptive agents
  - B. Phenylpropanolamine phenylephrine
- VI. Miscellaneous
  - A. Elevated intracranial pressure (acute)
  - B. Fever
  - C. Pregnancy
  - D. Acute stress or anxiety
  - E. Other

The estimated decrease in the risk of myocardial infarction is 2-3% for every reduction of 1 mm Hg in the diastolic blood pressure.

#### 5. Low HDL cholesterol <35 mg/dl Total cholesterol >200 mg/dl

About one third of the adults in the U.S. have hypercholesterolemia. It is estimated that maintaining plasma cholesterol below 200 mm/dl would decrease the incidence of IHD 30–50% in persons younger than 65 years of age. Alternatively, a 1% decrease in a patient's total plasma cholesterol concentration yields a 2-3% reduction in the risk of IHD.

6. Diabetes mellitus

Prominent risk factor for development of IHD

*Negative Risk Factors* High HDL-cholesterol (≥60 mg/dl)

#### "A postoperative MI is a lethal lesion with a >50% mortality rate."

Robert K. Stoelting, Stephen F. Dierdor, <u>Anesthesia and Co-existing Disease</u>, 3rd ed. New York: Churchill Livingston, Inc., 1993, p. 602 Paul Barach, M.D., Preoperative evaluation of the cardiac patient for non-cardiac surgery. *Can J Anesth*, 1991.

"Left ventricular ejection fraction,...as determined by MUGA Scan, has been shown to be an important predictor of survival in patients with myocardial infarction and congestive heart failure" (who are undergoing non-cardiac surgery).

LVEF	Mortality Rate
>55%	2.2%
35-54%	5.4%
<35%	19.5%

For LVEF of <50% or >70%, the cardiac complication rate is 58%, as compared to 12% in those with normal ejection fractions.

*Preoperative Medicine*, 2nd ed. Goldman, Brown. The Surgical Patient with Congestive Heart Failure, Howard J. Eiser.

# I. ECHOCARDIOGRAPHY

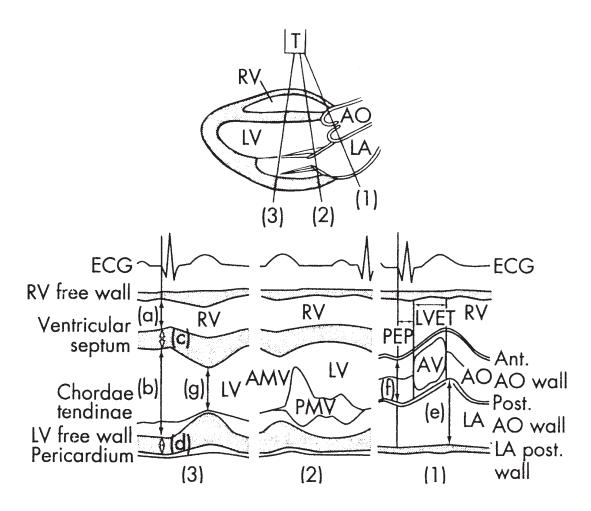
# A. M-Mode Echocardiography

The M-mode echo, which provides an "ice pick" view of the heart, is important in the evaluation of certain cardiac conditions and functions, particularly in the measurement of dimensions and timing. An M-mode echo through three important structures of the left side of the heart is illustrated in Fig 2-1.

- 1. Applications of the M-mode echo
  - a. Measurement of the dimensions of cardiac chambers and vessels and the thickness of the ventricular septum and free walls.
  - b. LV systolic function (e.g., fractional shortening, ejection fraction).
  - c. Study of the motion of valves (e.g., MVP, MS, pulmonary hypertension) and the interventricular septum.
  - d. Detection of pericardial fluid.
- 2. Normal M-mode echo values (See Appendix, Tables A-3, A-4)
- 3. LV Function (see Table A-4)
  - a. Fractional shortening (FS) of the LV is as follows:

$$FS(\%) = \frac{Dd - Ds}{Dd} \times 100,$$

where Dd is end-diastolic dimension and Ds is endsystolic dimension.



#### FIG. 2-1.

Cross-sectional view of the left side of the heart along the long axis *(top)* through which ice pick views of the M-mode echo recordings are made *(bottom). a,* RV dimension. *b,* LV diastolic dimension (Dd). *c,* thickness of ventricular septum. *d,* thickness of posterior free wall. *e,* LA dimension. *f,* aortic dimension. *g,* LV systolic dimension (Ds). *AMV,* anterior mitral valve. *PMV,* posterior mitral valve. *LVET,* LV ejection time. *PEP,* pre-ejection period. (From Park MK: *Pediatric Cardiology for Practitioners,* ed 3. St. Louis, Mosby, 1995.)

Normal FS is 36% (28% to 44%, 95% CI). b. Ejection fraction (EF) is obtained by this formula:

EF (%) = 
$$\frac{(Dd)^3 - (Ds)^3}{(Dd)^3} \times 100.$$

Normal EF is 74% (64% to 83%, 95% CI). c. Systolic time intervals

# **Prophylaxis Guidelines—Endocarditis** Erythromycin for Bacterial Endocarditis Prophylaxis: Then and Now

• **In 1994,** the American Heart Association issued recommendations for antibiotic prophylaxis for patients who are prone to bacterial endocarditis. For amoxicillin/peni-ciillin-allergic patients, the Heart Association recommended:

Erythromycin ethylsuccinate 800 mg or erythromycin stearate 1.0 g orally 2 hours before a procedure; then on-half the dose 6 hours after the initial dose. OR Clindamycin 300 mg orally 1 hour before a procedure and 150 mg 6 hours after initial dose.

**Problem #1:** There was confusion over the Heart Associations's recommendations. The Association said to ues 1 g erythromycin stearate, or if you're using the \ethylsuccinate salt, they say to give 800 mg. Pharmacists generally agree that 250 mg of tearate is roughly equivalent to 500 mg of the ethylsuccinate. So, 1 g of stearate is roughly equivalent to 1600 mg of ethylsuccinate... not 800 mg as recommended by the Heart Association. While these doses of erythromycin aren't considered equivalent, they provide adequate antibiotic concentrations.

Problem #2: Erythromycin can cause GI upset.

• **In 1997,** the Heart Association issued new guidelines. Erythromycin is no longer recommended for the amoxicillin/penicillin-allergic patient, Instead, the Heart Association recommends:

A single dose of clindamycin 600 mg, azithromycin 500 mg, clarithromycin 500 mg, cephalexin 2 g or cefadroxil 2 g for adults.

But if the patient and physician are comfortable using the old erythromycin regimen, they can continue to do so; but the new regimen is considered effective and has fewer side effects.

To help you keep track of who shold receive prophylaxis for bacterial endocarditis, what procedures are risky and what regimens are recommended, we have attached some tables reprinted with permission from the American Heart Association.

# Cardiac Conditions For Which Prophylaxis Is Or Is Not Recommended

### **Endocarditis Prophylaxis Recommended**

#### **High Risk Category**

Prosthetic cardiac valves, including bioprosthetic and homograft valves Previous bacterial endocarditis

Complex cyanotic congenital heart disease (e.g. single ventricle states, transposition of the great arteries, tetralogy of Fallot)

Surgically constructed systemic-pulmonary shunts or conduits

#### **Moderate Risk Category**

Most other congenital cardiac malformations (other than above and below) Acquired valvar dysfunction (e.g. rheumatic heard disease) Hypertrophic cardiomyopathy

Mitral valve prolapse with valvar regurgitation and/or thickened leaflets

# Endocarditis Prophylaxis Not Recommended

#### Negligible Risk Category (No Greater than the General Population)

Isolated secundum atrial septal defect

Surgical repair of atrial septal defect, ventricular septal defect, or patent ductus arteriosus (without residual beyond 6 mo)

Previous coronary artery bypass graft surgery

Mitral valve prolapse without valvar regurgitaion

Physiologic, functional, or innocent heart murmurs

Previous Kawasaki disease without valvar dysfunction

Previous rheumatic fever without valvar dysfunction

Cardiac pacemaker (intravascular and epicardial) and implanted defibrillators

# Dental Procedures For Which Prophylaxis Is Or Is Not Recommended

## **Endocarditis Prophylaxis Recommended**

\*Dental extractions

Periodontal procedures including surger, scaling and root planing, probing, recall maintenance

Dental implant placement and reimplantation of avulsed teeth

Endodontic (root canal) instrumentaion or surgery only beyond the apex

Subgingival placement of antiobiotic fibers/strips

Initial placement of orthodontic bands but not brackets

Intraligamentary local anesthetic injections

Prophylactic cleaning of teeth or implants where bleeding is anticipated

## **Endocarditis Prophylaxis Not Recommended**

Restorative dentistry<sup>^</sup> (operative and prosthodontic) with/without retraction cord# Local anesthetic infections (nonintraligamentary) Intracanal endodontic treatment; post placement and buildup Placement of rubber dams Postoperative suture removal Placement of removable prosthodontic/orthodontic appliances Taking of oral impressions Taking of oral radiographs Fluoride treatments Orthodontic appliance adjustment Shedding of primary teeth

\* Prophylaxis is recommended for patients with high and moderate risk cardiac conditions.

<sup>^</sup> This includes restoration of decayed teeth (filling cavities) and replacement of missing teeth.

#Clinical judgement may indicate antibiotic use in selected circumstances that may create significant bleeding.

# Other Procedures For Which Prophylaxis Is Or Is Not Recommended

## **Endocarditis Prophylaxis Recommended**

**Respirator Tract** 

Tonsillectomy and/or adenoidectomy

Surgical operations that involve respiratory mucosa

Bronchoscopy with a rigid bronchoscope

Genitourinary Tract

Prostatic surgery

Cystoscopy

Urethral dilation

Gastrointestinal Tract\*

Sclerotherapy for esophageal varices

Esophageal stricture dilation

Endoscopic retrgrade cholangiography with billiary obstruction

Billiary tract surgery

Surgical operations that involve intestinal mucosa

## **Endocarditis Prophylaxis Not Recommended**

**Respiratory Tract** 

Endotracheal intubation

Bronchosopy with flexible bronchoscope, with or without biopsy#

Tympanostomy tube insertion

Gastrointestinal Tract

Transophageal echocardiography#

Endoscopy with or without gastrointestinal biopsy#

Genitourinary Tract
Vaginal hysterectomy#
Vaginal delivery#
Cesarean section
In uninfect5ed tisue:
urethral catheterization
Uterine dilatation and curettage
therapeutic abortion
sterilazation procedures
insertion or removal of intrauterine devices
Other
Cardiac catheterization, including balloon angioplasty
Implantation of cardiac pacemakers, implanted defibrillators, and coronary stents
Incision of biopsy of surgically scrubbed skin
Circumcision

\* Prophylaxis is recommended for high-risk patients; optional for medium-risk patients. # Prophylaxis is optional for high-risk patients.

#### Prophylactic Regimens For Dental, Oral, Respiratory Tract, Or Esophageal Procedures (No Follow-Up Dose Recommended)

	· · · · ·	I /
Situation	Agent	Regimen#
Standard general prophylaxis	Amoxicillin	Adults: 2.0 g; Children: 50 mg/kg PO 1 hour before procedure
Unable to take oral medications	Ampicillin	Adults: 2.0 g IM or IV; Children: 50 mg/kg IM or IV within 30 minutes before procedure
Penicillin-allergic		Adults: 600 mg; Children: 20 mg/kg PO 1 hour before procedure Adults: 2.0 g; Children: 50 mg/kg PO 1 hour before procedure Adults: 500 mg; Children: 15 mg/kg PO 1 hour before precedure
Penicillin-allergic and unable to take oral medicatrions	Clarithromycin Clindamycin OR Cefazolin*	Adults: 600 mg; Children: 20 mg/kg IV within 30 minutes before procedure Adults: 1.0 g; Children: 25 mg/kg IM or IV within 30 minutes before procedure
	Cerazoilli	Adults. 1.0 g, Children. 25 mg/kg hvi of 1V within 50 minutes before procedure

# Total children's dose should not exceed adult dose.

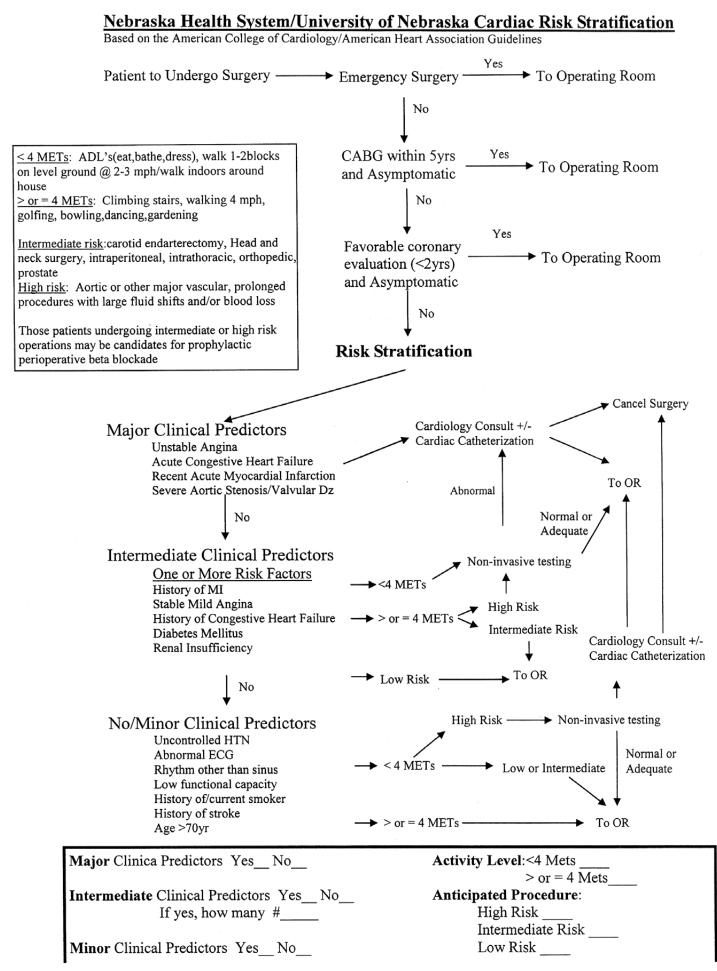
\* Cephalosporins shold not be used in individuals with immediate type hypersensitivity reaction (urticaria, angioedema, or anaphylaxis) to penicillins.

#### Prophylactic Regimens For Genitourinary/Gastrointestinal (Excluding Esophageal) Procedures

Situation	Agent(s)*	Regimen#
High-risk patients	Ampicillin plus Gentamicin	Adults: ampicillin 2.0 g IM/IV plus gentamicin 1.5 mg/kg (not to exceed 120 mg) withing 30 min of starting the procedure. Six hours later, ampicillin 1 g IM/IV or amoxicillin 1 g PO.
		Children: ampicillin 50 mg/kg IM or IV (not to exceed 2.0 gm) plus gentamicin 1.5 mg/kg within 30 minutes of starting the procedure. Six hours later, ampicillin 25 mg/kg IM/IV or amoxicillin 25 mg/kg PO.
High-risk patients allergic to ampicillin/amoxicillin	Vancomycin plus Gentamicin	Adults: vancomycin 1.0 g IV over 1-2 hours plus gentamicin 1.5 mg/kg IV/IM (not to exceed 120 mg). Complete injection/infusion within 30 minutes of starting the procedure.
		Children: vancomycin, 20 mg per kg IV over on to two hours, plus gentamicin, 1.5 mg per kg IV or IM; injection or infusion should be completed within 30 minutes of starting the procedure.*
Moderate-risk patients	Amoxicillin OR Ampicillin	Adults: amoxicillin 2.0 gm PO 1 hour before preocedure, OR Ampicillin 2.0 gm IM/IV within 30 minutes of starting the procedure
		Children: amoxicillin 50 mg per kg orally one hour before the procedure, OR ampicillin, 50 mg per kg IM or IV within 30 minutes of starting the procedure
Moderate-risk patients allergic to ampicillin/amoxicillin	Vancomycin	Adults: vancomycin 1.0 gm IV over 1-2 hours. Complete infusion within 30 minutes of starting the procedure. Children: vancomycin 20 mg/kg IV over 1-2 hours. Complete infusion within 30 minutes of starting the procedure.
		minutes of starting the procedure.

# Total children's dose should not exceed adult dose.

\* No second dose of vancomycin or gentamicin is recommended.





#### The Nebraska Medical Center: Prophylactic Perioperative Beta Blockade (PPBB) Protocol and Cardiac Risk Stratification

The following guidelines are basic recommendations and are not intended as absolute or standard requirements. Practice guidelines should be modified based on clinical needs and individual practice to ensure highest quality of care.

- All patients scheduled for elective non-cardiac surgery (intermediate and high risk\*) requiring general or regional anesthesia and a hospital stay qualify
  - o Emergency surgery and hemodynamically unstable patients assessed on an individual basis
- PPBB should be considered for patients with
  - o Known coronary artery disease
  - o Atherosclerotic vascular disease
  - o Diabetes
  - o Any two of the following: Age>65, HTN, hyperlipidemia, current smoker
- PPBB may be contraindicated in the following
  - o Sensitivity to Beta Blockers
  - o Congestive Heart Failure (LVEF <30%)
  - o Active Bronchospasm
  - o Second or Third degree heart block
  - o Systolic BP < 100mmHg or HR <50 bpm
  - o C-Section
- Drug Choice: Atenolol or Metoprolol may be used. If the patient is previously on another beta blocker, it is unnecessary to change to a Beta-1 selective drug. However, the dosage should be adjusted to keep HR<70bpm perioperatively.
- PPBB initiation: Target HR: >50 and <70 bpm
  - o **Preoperatively in clinic:** if HR >60 and SBP>100mmHg, begin metoprolol (25-50mg PO q 12h) or atenolol (50-100mg PO qd) several days before surgery.
  - o **Immediately Preop:** If HR>70 and SBP>100mmHg, metoprolol 2.5-5.0mg IV can be given every 10 minutes to maintain HR<70 and SBP>100mmHg
  - o **During surgery:** if HR >70 and SBP>100mmHg, metoprolol 2.5-5.0mg IV can be given every 10 minutes to maintain HR<70 and SBP>100mmHg. Alternatively, esmolol infusion may be titrated to maintain HR<70.
  - o **PACU or ICU:** If HR>70 and SBP>100mmHg, metoprolol 2.5-5.0mg IV can be given every 10 minutes to maintain HR<70 and SBP>100mmHg. Consider utilizing standard orders.
  - o **Postoperative:** Utilize standard orders for both IV and PO dosing.
  - o Discharge: Oral Beta Blocker therapy should be continued for at least 7 days postoperatively.
    - Goal HR<70 and SBP>100
    - Patients with a history of coronary artery disease may benefit from indefinite Beta-Blocker therapy.

\*Intermediate risk: Carotid endarterectomy, Head and neck surgery, intraperitoneal, intrathoracic, orthopedic, prostatectomy

High risk: Aortic or other major vascular, prolonged procedures with large fluid shifts and/or blood loss

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		R less than 70 bpm			
	NOTE: Hold beta	blocker at any time if HR less that	n 50 bpm or SBP less than 90 mmH	<b>D</b>	
	NOTE: Hold beta blocker at any time if HR less than 50 bpm or SBP less than 90 mmHg ECG monitoring required for giving IV beta-blocker				
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and other second second		our after every oral dose of b-blocke nin after every IV dose of b-blocke			
an Alakan kang	<b>DOSING PO:</b> If NPO, patient may still take beta blocker PO with sip of H2O				
	Atenolol mg PO q day Max dose 200 mg/day				
·····	Metoprolol	mg PO q 12 hours Max dos	se 400 mg/day		
	DOSING IV: Dru	ig to be administered by slow IV p	ush		
	Metoprolol	mg IV q 6 h (Usual Dose- 5-1.	5mg)		
	DISCHARGEPLAN	NING:			
a constant of the second	<ul> <li>Continue beta blocker PO for 7 days following surgery</li> <li>Continue beta blocker until seen by primary care physician</li> </ul>				
-	DOSINGPARAMI	CTERS;			
ay a carao e e e e e e	HR	IV	РО		
	less than 50	Hold all doses and notify MD	Hold all doses and notify MD		
	50-55	Decrease next dose by 50%	Decrease next dose by 50%		
	55-70	No change	No change		
ACCULATION AND A DESCRIPTION			-		
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oonaa ohaan ka araba dha araba ahaa aa arabka aa ahaa dha dhaanaa aanaa ahaa dha	70-100 greater than 100	Double next dose	Double next dose		

 PERIOPERATIVE
 PROPHYLACTIC
 BETA
 BLOCKER
 PROTOCOL

 White Copy - Medical Record
 Yellow Copy - Pharmacy
 Pink Copy - Patient

ORD-MR-0426 (Rev. 2/05)

ORDERS

### PROPHYLACTIC PERIOPERATIVE BETA BLOCKER PROTOCOL

ATTACH PATIENT STICKER HERE

Please complete after **each surgical procedure** and return, along with the usual self-assessment quality assurance sheet, to **Anesthesia Administration**.

Question 1 – Does the patient meet the criteria for perioperative beta blocker use?

Question 2 – If yes, did the patient receive oral beta blockers pre-operatively? □ Yes – Go to Q4 □ No – Go to Q3

Question 3 – If not, did the patient receive intravenous beta blockers in the immediate perioperative period <u>and</u> intraoperatively? □ Yes – Go to Q4 □ No – Go to Q4

**Question 4-** Were there any adverse events specifically related to the use of beta blockers?

□ Yes

🗆 No

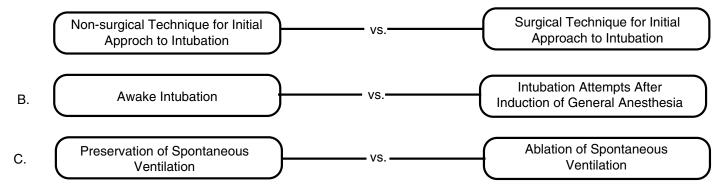
\*\*\* Anesthesia Administration: Please forward to Stephanie Gould at ZIP: 7465.

#### **Rapid Sequence Intubation**

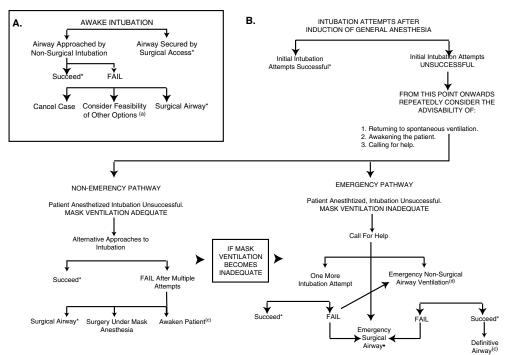
Difficult airway algorithm developed by ASA Task Force on Guidelines for Difficult Airway Management Reprinted with permission from American Society of Anesthesiologists

#### **DIFFICULT AIRWAY ALGORITHM**

- 1.Assess the likelihood and clinical impact of basic management problems:
  - A. Difficult Intubation
  - **B. Difficult Ventilation**
  - C. Difficulty with Patient Cooperation or Consent
- 2. Consider the relative merits and feasibility of basic management choices:



3. Develop primary and alternative strategies:



#### \* CONFIRM INTUBATION WITH EXHALED C0,

(a) Other options include (but are not limited to): surgery under mask anesthesia, surgery under local anesthesia infiltration or regional nerve blockade, or intubation attempts after induction of general anesthesia.

(b) Alternative approaches to difficult intubation include (but are not limited to): use of different laryngoscope blades, awake intubation, blind oral or nasal intubation, fiber-optic intubation, intubating stylet or tube changer, light wand, retrograde intubation, and surgical airway access.

(c) See awake intubation.

(d) Options for emergency nonsurgical airway ventilation include (but are not limited to): transtracheal jet ventilation, laryngeal mask ventilation, or esophageal-tracheal combitube ventilation. (e) Options for establishing a definitive airway include (but are not limited to): returning to awake state with spontaneous ventilation, tracheotomy, or endotracheal intubation.

#### Selected Pathologic States That Influence Airway Management

Pathologic State	Difficulty
Infectious epiglottis	Laryngoscopy may worsen obstruction.
Abscess (submandibular, retropharyngeal, Ludwig's angina)	Distortion of airway renders mask ventilation or intubation extremely difficult.
Croup, bronchitis, pneumonia (current or recent)	Airway irritability with tendency for cough, laryngospasm, broncho- spasm
Papillomatosis	Airway obstruction
Tetanus	Trismus renders oral intubation impossible.
Traumatic foreign body	Airway obstruction
Cervical spine injury	Neck manipulation may traumatize spinal cord.
Basilar skull fracture	Basal intubation attempts may result in intracranial tube placement.
Maxillary/mandibular injury	Airway obstruction, difficult mask ventilation, and intubation; crico- thyroidotomy may be necessary with combined injuries.
Laryngeal fracture	Airway obstruction may worsen during instrumentation. Endotracheal tube may be misplaced outside larynx and may worsen the injury.
Laryngeal edema (postintubation)	Irritable airway, narrowed laryngeal inlet
Soft tissue, neck injury (edema, bleeding, emphysema)	Anatomic obstruction of airway Airway obstruction
Neoplastic upper airway tumors (harynx, larynx)	Inspiratory obstruction with spontaneous ventilation
Lower airway tumors (trachea, bronchi, mediastinum)	Airway obstruction may not be relieved by tracheal intubation. Lower airway distorted
Radiation therapy	Fibrosis may distort airway or make manipulations difficult.
Inflammatory rheumatoid arthritis	Mandibular hypoplasia, temporomandibular joint arthritis, immobile cervical spine, laryngeal rotation, cricoarytenoid arthritis all make intubation difficult and hazardous.
Ankylosing spondylitis	Fusion of cervical spine may render direct laryngoscopy impossible.
Temporomandibular joint syndrome True ankylosis "False" ankylosis (burn, trauma, radiation, temporal craniotomy)	Severe impairment of mouth opening

Scleroderma	Tight skin and temporomandibular joint involvement make mouth opening difficult.
Sarcoidosis	Airway obstruction (lymphoid tissue)
Angioedema	Obstructive swelling renders ventilation and intubation difficult.
Endocrine/metabolic acromegaly	Large tongue, bony overgrowths
Diabetes mellitus	May have reduced mobility of atlanto-occipital joint
Hypothyroidism	Large tongue; abnormal soft tissue (myxedema) make ventilation and intubation difficult.
Thyromegaly	Goiter may produce extrinsic airway compression or deviation.
Obesity	Upper airway obstruction with loss of consciousness. Tissue mass makes successful mask ventilation unlikely.

Adapted from: Stone, DJ, Gai, TJ. Airway management. In Miller, R.D., ed. *Anesthesia*, vol. 2, New York: Churchill Livingstone: 1994; (4):1407.

### **Conditions Associated with Increased Risk of Aspiration**

Full stomach (<8 hour fast), or vague history of intake

Gastrointestinal pathology (intestinal obstruction/inflammation; gastric paresis secondary to drugs, infection, uremia, diabetes mellitus)

Obesity

Pregnancy

Trauma

Esophageal disorders, for example, reflux, motility disorders

Adapted from: Stone, DJ, Gai, TJ. Airway management. In Miller, RD, ed. Anesthesia, vol. 2, New York: Churchill Livingstone; 1994:(4), 1403–1435.

Reprinted in Coggin, WJ. Rapid sequence intubation. *Physician Assistant*, January 1996, pp. 53–62.

# **Protocol for Management of Surgical Patients**

### **Preoperative Nothing by Mouth (NPO) Orders**

- 1. Adults receiving general, regional, or monitored anesthesia care will be NPO after midnight preceding surgery.
- 2. Guidelines for NPO status in children:

age	solids	clear liquids
<6 mos.	4°	2°
6-36 mos.	6°	3°
3-6 yr.	8°	3°

6 years or older:

NPO after midnight or at least 8 hours prior to arrival time.

3. Patients receiving local anesthesia will follow any NPO instructions given by their surgeon. If no orders are given, the patient will be instructed to remain NPO after midnight.

### PULMONARY FUNCTION TESTS

### Physiological assessments

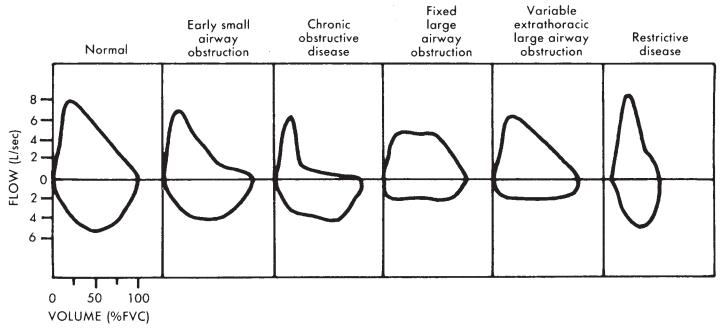
Physiological assessment tests are performed according to accepted protocols and the results are expressed as percents of established normals. Therapeutic bronchodilators should be withheld if possible before testing. Fig. 28-4 compares normal obstructive and restrictive ventilatory patterns. Table 28-1 summarizes pulmonary function testing abnormalities in common disorders.

- 1. The presence of obstructive disorders (e.g., bronchitis, asthma) is evidenced by reduced airflows:
  - a. FEV<sub>1</sub>/FVC: % predicted
    >80 normal
    65-79 mild obstructive disease
    50-64 moderate obstructive disease
    <50 severe obstructive disease</li>
  - b. FEF 25-75: indicates small airways
  - c. Peak expiratory flow rate (PEFR): reflects mostly large airways
  - d. FEF 50, 25: indicate small airways (flow-volume)
  - e. Flow-volume tracing may also indicate extrathoracic obstruction, particularly in the inspiratory phase (Fig. 28-5 compares flow-volume curves in various disease states)
  - f. Patients with COPD may have a mixture of reversible and irreversible airway obstruction
    - (1) Improvement in  $FEV_1 > 15\%$  after inhaled bronchodilator demonstrates reversibility
    - (2) Negative response does not exclude reversibility, however, since patients can still have improvement in pulmonary function on long-term bronchodilator therapy
- 2. Increased residual volume (RV) on spirometry suggests hyperinflation (COPD, asthma); decreased RV occurs in restrictive disease (e.g., interstitial fibrosis) (Fig. 28-6 compares spirometric patterns in obstructive and restrictive disease)
- 3. Presence of restriction is manifested by a decrease in lung volumes, particularly VC and TLC; it occurs with neuromuscular, skeletal, pleural, and interstitial lung disorders
- 4. Diffusing capacity is reduced with interstitial disease, thickness of or loss of air exchange surface, and decreased ventilation or perfusion; useful as an early sign of emphysema or predictor of oxygen desaturation or poor exercise tolerance

Disorder	FVC	FEV <sub>1</sub>	FEV <sub>1</sub> /FVC	RV	TLC	Diffusing Capacity
Asthma	$\downarrow$	$\downarrow$	$\downarrow$	↑	N/ ↑	N
COPD	N/ ↓	¥	$\downarrow$	Î.	N/ ↑	$N/\downarrow$
Kyphoscoliosis	$\downarrow$	$\downarrow$	N/ ↑	N/↓	$\downarrow$	Ν
Interstitial fibrosis	$\downarrow$	$\downarrow$	N/ ↑	$\downarrow$	$\downarrow$	$\downarrow$

### Table 28-1 PFT abnormalities in common disorders

KEY:  $\downarrow$ , Decreased;  $\uparrow$ , increased; N, normal; FVC, forced vital capacity; FEV<sub>1</sub>, forced expiratory volume in 1 sec; RV, residual volume; TLC, total lung capacity





Flow-volume curves of restrictive disease and various types of obstructive diseases compared with normal curves. FVC is functional vital capacity.

1. Fred F. Ferri, M.D. Practical Guide to the Care of the Medical Patient (2nd ed.) Mosby-Year Book, Inc., p.508-512

# The Assessment of Dyspnea

Grade 0	No dyspnea while walking at a normal pace
Grade I	"I am able to walk as far as I like provided I take my time."
Grade II	Specific street block limitations – "I have to stop for a while after one or two blocks."
Grade III	Dyspnea on mild exertion – "I have to stop and rest while going from the kitchen to the bathroom."

Grade IV Dyspnea at rest

Marienau, M.E.S., CRNA, MS, & Back, C.I., CR, RRT, (1998). Preoperative Evaluation of the Pulmonary Patient Undergoing Non Pulmonary Surgery. *Journal of Peri Anesthesic Nursing*. 13 (6), 340-348

#### 

	RECOMMENDED GUIDELINES FOR SURGICAL PREOP SURGICAL PROCEDURES CLINICAL DISORDERS CONDITIONS/DRUGS																													
		SU	RGICA	L PRO	OCED	URES						CLINI	CALE	DISOR	DERS								c	ONDI	TIONS	/DRUG	3S			$\square$
<ul> <li>+ needs         <ul> <li>- does not need</li> <li>ASA 3.4 workup within</li> <li>7-10 days</li> <li>pregnancy screen</li> <li>within 24 hours</li> </ul> </li> </ul>	Cardiac/Thoracic	Vascular	Intraperit/(Major)Abd	Anticipated >2u Estimated Blood Loss	Intracranial	Ortho Prosthesis	TURP	Cardiovascular (HTN, CHF, CVA, CAD)	Pulmonary Disease	Chemotherapy/Malignancy	Hepatic Disease	Renal Disease	Diabetes	Seizure Disorder	HIV	Autoimmune/Lupus	Unstable Thyroid	Parathyroid	Anticoag/Bleeding Disorder	≥ 20 pk/yr Smoking	ETOH/Drug Abuse	Diuretics, Fluid or Lyte Loss	Suspected Pregnancy	Radiation	Adrenal Disorder	Digoxin	Obesity	SSRI (Antidepressant)	Age > 50	Age > 60
TEST																														
EKG	+	+	+	+	+	+	+	+	+	+	+	+	+		+	+ '	+	+		+	+	±		+	+	+	+		+	+
CBC & PLT	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+		+	+	±	+	+		+			
BASIC METABOLIC (GLUCOSE INCLUDED)	+	+	+	+	+	+	+	+		+	+	+	+	+	+	+	+	+			+	+		+	+	+	+	+		+
LFT'S			±							±	+			±	+						+			±	+		±			
CALCIUM											+							+												
PT/PTT	+	+		+	+	+			±	±	+	+				+			+		+			±			±			
UA(CULTURE)						+							-																	
CXR	+	+	+	±	±	+	±	+	+	+	+	+	+		±	+	+			+	+			+	±	+	+			
HORMONE LEVEL																	+	+												
BLEED TIME																			±											
PREGNANCY																							+							
CARDIAC/SEIZURE/ PULMONARY DRUG LEVELS					±			±	±					±											+	+				
CLOT	Depe	nds on si	urgery/An	nount of t	blood loss	anticipat	ed																							
PFT	±								±											±				±			±			
SLEEP STUDY																											±			
COMPLETE C-SPINE: RHEUM	ETE C-SPINE: RHEUMATOID ARTHRITIS + DOWNS SYNDROME + RECENT NECK INJURY +																													

Adapted from: Thomas M. Halaszynski; Richard Juda; David G. Silverman, "Optimizing postoperative outcomes with efficient preoperative assessment and management," Critical Care Medicine, Volume 32, Number 4,(April 2004), S80

Anesthesia Third Edition. Churchill Livingston Inc., 1990. New York. Chapter 23: "Preoperative Evaluation" Michael F. Roizen. pp. 762-765

Diagnostic	Hospital charge to patient	Pro-fee charge to patient	Total charg to patient
EKG	\$247.60	\$25.00	\$272.60
Adult echocardiogram dop, color 2D	1,325.87	373.00	1,698.87
Adult transesophageal			
echocardiogram	1,509.73	521.00	2,030.73
Treadmill	827.33	152.00	979.33
Stress echo	1,762.18	429.00	2,191.18
Lab Test	Inpatient (\$)	Outpa	tient (\$)
CBC w/auto diff	104.29	)	48.31
	69.99		
	93.84		
Hemoglobin & Hematocrit			
Plt. Count automated			
Basic Metabolic Panel	230.89	)	46.09
Lytes BUN/Creat ]	167.17	7	31.87
BUN/Creat.	BUN: 57.13/C: 81.89	BUN: 22.	37/C: 22.37
	57.13		
Complete Metabolic			
Hepatic Function Panel			
Mg+		2	
Ca++			
Calcium Ionized			
Glucose (Quant.)	57.13	3	22.37
	76.11		
	76.33		
	132.74		
T4, Free			
UA (without micro)			
UA with Microscopic			
hCG serum			
hCG urine or serum (qual.)			
DII	45.71		
RH		3	
AB (only)			
Type & Cross per unit	IUI.33	) <sup>**</sup>	63.32* 164.60
Blood Gas w/O <sub>2</sub> Sat	1//.3 125 77	l 7	104.09 125 77
Blood Gas, Arterial CXR pa/lat			

\*for two units this would double. Also, if the AB is positive or the type and crossmatch were incompatable, there would be additional charges

### New HCFA Chemistry Panels Compared With Previously Offered Panels At Clarkson and University Hospitals

#### **Electrolytes**

Phamis: LYTE1 Sodium Potassium Chloride Carbon Dioxide Anion Gap Inpatient Cost: 167.17

Outpatient Cost: 31.87

#### **Basic Metabolic Panel**

Phamis: BMET Glucose BUN Calcium Creatinine Sodium Postassium Chloride Carbon Dioxide BUN/CR Anion Gap Calc. Osmolality Inpatient Cost: 230.89 Outpatient Cost: 46.09

#### **Comprehensive Metabolic Panel**

Phamis: CMET Glucose **BUN** Creatinine **BUN/CR** Sodium Potassium CO<sub>2</sub> and ALT Chloride Calc. Osmolality Calcium Total Protein Albumin AST ALK Phos. **Total Billirubin** Inpatient Cost: 352.97

Outpatient Cost: 73.16

### New HCFA Chemistry Panels Compared With Previously Offered Panels At Clarkson and University Hospitals

#### **Hepatic Function Panel**

1	
Meditech: HEPFUNC	Phamis: HFP
Albumin	
AST	
ALT	
ALK Phos.	
Total Bilirubin	
Direct Bilirubin	
Inpatient Cost: 194.72	Outpatient Cost: 51.27

#### **Lipid Panel**

L	
Meditech: LIPTID1	Phamis: LIPID
Cholesterol	
Triglyceride	
HDL	
CHOL/HDL	
LDL (calculated)	
VLDL (calculated)	
Inpatient Cost: 125.17	Outpatient Cost: 73.03

Please note: The Comprehensive Metabolic Panel and the Hepatic Function Panel may not be ordered simultaneously on a patient due to the duplication of panel components.

- 1. *Type & Screen:* unarmbanded specimen type: ABO/Rh, antibody screen
- 2. *Type & Possible: armbanded specimen*. Type: ABO/Rh, antibody screen. If screen is (+), then the antibody is identified and 2 Ag–units are crossmatched.
- 3. *Clot to Hold: armbanded specimens*. Specimen is processed and placed in storage in case of need for products.
- 4. *Type & Cross: armbanded specimen*. Type: ABO/Rh, antibody screen. If screen is (–), the # of units needed is crossmatched. If screen is (+), antibody identification & units "Ag–" are crossmatched.

### FOR #s 1–4...72 HOURS.

5. *Extended Crossmatch*. This is valid if the patient has not been transfused with any blood products for 120 days or pregnant within 120 days. An armbanded specimen that is valid for 10 days from the date of draw. ABO/Rh, antibody screen & antibody identification if necessary, the day before surgery the units are crossmatched.

### Normal Ranges for Lab

### **PT Prothrombin Time**

11-13.8 seconds

### PTT Partial Thromboplastin Time

22–37 seconds

	Male	Female
WBC X 10 <sup>3</sup> /MM <sup>3</sup>	4–11	4–11
RBC	4.40–5.80	3.80-5.20
HGB	13–17	11.5–15.5
HCT%	37–51	35–46
MCV	82–98	89–92
МСН	27–33	27–33
MCHC%	32–36	32–36
RDW%	10.5–14.5	10.5–14.5

CBC

### **Differential Count (adults)**

	%	Absolute # X 10 <sup>3</sup> /MM <sup>3</sup>
Segs	43–74	
Bands	0–10	<1.0
Neutrophils	45–75	1.8–7.5
Lymphocytes	15–45	1.0–3.4
Monocytes	1–2	0.1–0.8
Eosinophils	0–6	≤0.4
Basophils	0–2	≤0.2

### **Renal Panel**

	Reference Range	Critical Limits
Chloride	95–110 mEq/L	<70, >120 mEq/L
CO2	22–30 mEq/l	<10, >40 mEq/L
Creatinine	0.9–1.3 mg/dl (adult male)	>10 mg/dl
	0.6–1.1 mg/dl (adult female)	
Potassium	3.5–4.7 mEq/L	<2.5, >6.5 mEq/L
Sodium	134–145 mEq/L	<115, >160 mEq/L
Urea nitrogen	5–22 mg/dl	>100 mg/dl
Anion gap	4–15	

#### T4, Thyroxine, Total (adult) 4.5–11.5 ug/dl

#### TSH (Thyroid Stimulating Hormone) 0.4–5.0 uIU/ml

Critical >100 uIU/ml

#### **Chemistry Profile**

•		<b>Critical Limits</b>
Albumin	3.6–5.0g/dl	
Alkaline phosphatase	40–143 IU/L	
	(adults ≥20 years)	
AST (SGOT)	0–46 IU/L	
Bilirubin (total)	0–1.2 mg/dl	>15 mg/dl
Calcium	8.4–10.4 mg/dl	<6.0, >13.0 mg/dl
Cholesterol	<200 mg/dl	
Creatinine	0.9–1.3 mg/dl (adult male)	>10 mg/dl
	0.6–1.1 mg/dl (adult female)	
Glucose	60–110 mg/dl	<45, >500 mg/dl
LDH	100–250 IU/L	>1000 IU/L
Phosphorus	2.5–4.5 mg/dl	<1.2, >9.0 mg/dl
Protein, total	6.3–8.3 g/dl	
Urea, nitrogen	5–22 mg/dl	>100 mg/dl
Uric Acid	3.3 mg/dl–8.6 mg/dl male	>15.0 mg/dl
	2.5–7.5 mg/dl	

Certain medications need to be stopped prior to surgery. If you are taking any of the following medications, please notify your physician to see what alternative medication you may be able to take, or if it is safe to discontinue the medication. Some medications may not be stopped abruptly, but may need to be weaned - CHECK WITH YOUR PRIMARY CARE PHYSICIAN. DO NOT STOP OTHER PRESCRIBED MEDS, i.e. blood pressure medication, thyroid meds, etc.

#### Aspirin or aspirin containing products (Stop 2 weeks prior to surgery)

Alka Seltzer Anacin Ascriptin Aspergum Bayer Bufferin Ecotrin Easprin Empirin Excedrin Generic Aspirin Measurin Midol Synalgos Zorprin

#### IMPORTANT NOTE IN BOXED AREA FOR ALL PATIENTS ON BLOOD THINNERS

If you have had an MI, stroke/TIA (ministroke), CAD, angioplasty with/without stent placement, By-pass surgery, Atrial fibrillation, carotid surgery, or blood clot (in the leg or lung) check with your primary care physician or cardiologist before stopping ASA or "blood thinners".

#### "Blood thinners" / Antiplatelet

Coumadin / warfarin Dipyridamole / persantine Plavix / clopidorel Pletal / cilostazol Ticlid / ticlopidine Danaparoid (Orgaran) Lovenox / enoxaprarin Fragmin / dalteparin Innohep - Tinzaparin Orgaran / danaparoid Heparin Enoxaprarin (Lovenox) Dalteparin (Fragmin) Tinzaparin

#### Anti-inflammatory/NSAIDs (Stop 2 weeks prior to surgery)

Advil Aleve (naproxen sodium) Anaprox (naproxen, naprelan, synflex) Ansaid (flurbiprofen) Cataflam (dicolfenac, voltaren, arthrotec) Clinoril (sulidac) Daypro (oxaprozin) Disalcid (salsalate, salgesic, salflex, monogesic) Feldene (piroxicam) Indocin (indomethacin, haltran) Lodine (etodolac) Meclomen Mefenamic acid (Ponstel) Motrin (ibuprofen, rufen, meipren midol naprin) Nalfon (fenoprofen) Orudis (oravail, ketoprofen) Relafen (nabumetone) Tolectin (choline Mg, trisalicylate) Toradol (acular, ketorolac)

Arthritis Medications (Stop 2 weeks prior to surge	erv)	erv	surge	to	orior	eks	we	p 2	(Stop	cations	Medic	hritis	Ar
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Cama arthritis B/C po
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Migraine/Headache Medications (Stop 2 weeks prior to surgery) Fiorinal Triaprin

#### Pain Medication (Stop 2 weeks prior to surgery)

Equagesic	Roxiprin
Norgesic	Salsalate
Percodan	Trisalate (choline Magnesium trisalicylate)
Robaxisal	

### Selective COX-2 inhibitors (Check with surgeon about stopping)

Celecoxib (Celebrex) Rofecoxib (Vioxx) - If you were told to stop your aspirin or blood thinners you should stop your Vioxx as well.

### All Diet Medications: Prescribed, Over-the-counter, Herbal

(Stop 2 weeks prior to surgery)	
Meridia	Phentermine (ionamin, adipex)
Metabolife	Tenuate

#### All Herbal Medications / teas / supplements (Stop 2 weeks prior to surgery) i.e. Echinacea, Ephedra, Ginseng, Gingko, Goldenseal, Kava Kava, St. John's Wort

#### Diet Supplements (Check with Primary Care physician about stopping) i.e., Protein Drinks Diet Drinks (SlimFast, etc.) (This does not mean TPN, Tube Feeding, Ensure, etc. - Do as directed by surgery or anesthesia team)

# Vitamins (Stop 10 days to 2 weeks prior to surgery, unless otherwise directed by surgeon)

All mega dose vitamins, Vitamin E, anti-oxidants, fish oils

# Medications for Ulcerative Colitis (check with primary care physician to see if you can stop meds)

Rowasa / Pentasa / Asacol

Sulfasalazine / azulfidine

### **Psychiatric Medications / Anxiety / Sleep Medication**

(Do not stop abruptly, check with Primary Care physician about stopping meds, medication may need to be weaned or substituted)

MAO inhibitors Serzone Trazadone (Desyrel)

\*\*\*\* STOP Alcohol products 48 hours prior to surgery. \*\*\*\*

\*\*\*\* STOP Tobacco products 24 hours prior to surgery. \*\*\*\*

\* \* \* \* STOP "street" / illicit drugs should be stopped 72 hours before surgery. \* \* \* \*

\* \* \* \* \* \* You MAY take Tylenol / Acetaminophen products for pain \* \* \* \* \* \* \*

# Recognizing the Difference Between *Drug Allergy* and *Drug Intolerance* (Adverse Drug Reactions)

Drug allergies are a special type of adverse drug reaction (ADR). The *Textbook of Adverse Drug Reactions*<sup>1</sup> defines "drug allergy" as mediated by immunological mechanisms. Allergic drug reactions are categorized as a type B (bizarre) adverse drug reaction. These reactions are totally aberrant effects that are not to be expected from the known pharmacological actions of a drug when given in the usual therapeutic doses. They are usually unpredictable and are not observed during conventional pharmacological and toxicological screening programs. Although their incidence and morbidity are usually low, their mortality may be high.

In contrast, an intolerance to a drug is categorized as a type A (augmented) adverse drug reaction. These reactions are the result of an exaggerated, but otherwise normal, pharmacological action of a drug given in the usual therapeutic doses. Examples include bradycardia with beta-blockers, hemorrhage with anticoagulants, or drowsiness with benzodiazepines. Type A reactions are largely predictable on the basis of a drug's known pharmacology. Drug therapy can often be continued with an alteration in dose or other intervention. They are usually dose-dependent and although their incidence and morbidity are often high, their mortality is generally low.

Why should we be concerned about the difference? Obviously, if a patient has a true allergy to a drug or class of drugs, we want to be aware not to expose the patient to a potentially dangerous or life-threatening situation. However, if a drug is listed as an allergy, but in actuality the patient has not demonstrated allergic symptoms but has experienced an intolerance such as nausea or gastrointestinal distress, the patient should **not** be precluded from future treatment with the drug as warranted.

*Example:* A patient comes to the emergency room with sustained chest pain and history of angina, hypertension, and coronary artery disease. The diagnosis of acute myocardial infarction is made following EKG and laboratory analysis. The treatment prescribed includes morphine. Morphine (and other narcotic analgesics to a lesser degree) is desirable for pain associated with ischemia because of its cardiovascular effects of venous pooling in the extremities causing decreased peripheral resistance. This effect results in decreases in venous return, cardiac work, and pulmonary venous pressure, thus decreasing oxygen demand by the heart.

When the patient's old chart arrives, the allergy list includes morphine. When the family is interviewed, they describe the patient's "allergy" as vomiting in response to morphine following a previous hospitalization. Morphine causes a central nervous system effect on the vomiting center to cause nausea and vomiting by depressing the vomiting center. An increase in vestibular sensitivity may also contribute to the high incidence of nausea and vomiting in ambulatory patients. By questioning the patient's family, the emergency room staff was able to conclude that the patient was not truly allergic to morphine.

If allergies are not correctly identified in PHAMIS, people won't know when to trust the information. Accurately describing an allergy vs. an intolerance will make sure patients are able to receive needed treatment when necessary and alert the health care team to actual allergic conditions which may preclude treatment with the offending drug.

1. E-S.K. Assem. Drug allergy and tests for its detection. In Davies, D.M., editor. *Textbook of adverse drug reactions*, 4th ed. New York: Oxford University Press, 1991, 689-713.

Prepared by Mary Windle, Pharm.D. Drug Information Services

# **Medication Allergies or Serious Adverse Drug Reactions**

**Drug Name:** (should this be from a list in order to allow PHAMIS to search for cross sensitivity)

#### **Description of reaction** (check all that apply):

Onset	immediate (within an hour following administration) delayed							
Severity								
	life-threatening							
	resulted in hospitalization							
	resulted in permanent disability							
	required antidote or treatment to prevent impairment							
Respiratory Symptoms								
_	shortness of breath							
	respiratory distress							

- \_\_\_\_\_ wheezing/bronchospasm
- \_\_\_\_\_ other (describe)

#### **Cardiovascular Symptoms**

- \_\_\_\_\_ shock (severe, abrupt hypotension)
- \_\_\_\_\_ hypertension
- \_\_\_\_\_ cardiovascular arrest
- \_\_\_\_\_ increased heart rate/palpitations

#### Dermatologic

- \_\_\_\_\_ urticaria/hives
- \_\_\_\_\_ itching, pruritus
- \_\_\_\_\_ contact dermatitis
- \_\_\_\_\_ other (describe

#### **Additional Symptoms**

- \_\_\_\_\_ fever
- \_\_\_\_\_ diaphoresis
- \_\_\_\_\_ generalized edema
- \_\_\_\_\_ laryngeal or facial edema (angioedema)
- \_\_\_\_\_ other (describe)

Reaction	$\mathrm{HN}_2$	CTX	IFX	CLB	BUS	L-PAM	TT	NTU	MTX	5-FU	6-MP	ara-C	ACT D	ADR	DNR	MX	VCR	VLB	BLEO	OTIM	DTIC	PCZ	DDP	CP	L-ASP	VP-16	Π
Hypersensitivity Anaphylaxis Skin rash Fever, chills	• p	• p	•	• p	• p	p p	• p	•	р + р	р р	• p	• p p	р • +	p + +	р +	• p	•	•	р р ++	• p p	• p +	р р	+ + •	р р	++ + •	р р	• p p
Dermatologic																											
Alopecia	+	++	++	•	•	•	р	р	+	+	р	р	р	++	++	р	++	+	+	р	р	р	+	р	р	р	•
Local necrosis	+	•	•		_	•	٠	•	•	р	—	р	++	++		р	++	++	•	++	р	_	•	•	•	р	•
Hyperpigmentation Nail changes	on • •	++	•	•	+	• +	р •	р •	р •	+ +	•	•	+ •	++	р +	• p	•	•	++	•	•	р •	р •	•	•	•	р +
Mucositis	+	+	•	•	•	•	•	•	++	++	•	р	++	++	++	+	•	+	+	+	•	+	•	•	•	•	•
Ocular																											
Conjunctivitis Lacrimal duct	•	•	•	•	•	•	•	•	+	+	•	+	•	+	•	р	•	•	•	•	•	•	•	•	•	•	•
fibrosis	•	•	•	•	•	•	•	•	•	+	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•
Diplopia	•	•	•	р	•	•	•	р	•	р	•	•	•	•	•	•	+	+	•	•	•	р	•	•	•	•	•
Retrobulbar				1				1		1												1					
neuritis	•	٠	٠	٠	٠	٠	٠	р	٠	•	٠	•	٠	٠	•	٠	٠	•	•	٠	٠	•	+	•	•	٠	•
Cataracts	٠	•	•	•	+		•	•	•	•	•	•	•	•	•	•	٠	•	•	•	•	•	٠	•	•	•	•
					•						-																
Reaction Cardiac	• HN <sub>2</sub>	+ CTX	+ IFX	• CLB	• BUS	• L-PAM	TT	• NTU	• MTX	• 5-FU	• 6-MP	• ara-C					. VCR	• VLB	• BLEO	• OTIM	• DTIC	• PCZ	DDP	• CP	• L-ASP	• VP-16	• HU
Reaction	• • HN2	• + CTX	• + IFX	• • CLB	d BUS	• • L-PAM	• •	• •	• p	а 5-FU	• •	• • ara-C			DNR +		d • VCR	d • VLB	• • BLEO	• b	• • DTIC	• • PCZ	d • DDP	• CP	• • L-ASP	d • VP-16	• •
Cardiac Necrosis Other cardiac Pulmonary	• • HN <sub>2</sub>	• + CTX	• + IFX	• • CLB	•	• • L-PAM	• •	• •	•	•	• • 6-MP	• • ara-C		DL	DL		•	•	• • BLEO	•	• • DTIC	• • PCZ	•	• •	• • L-ASP	•	• • HU
Cardiac Necrosis Other cardiac Pulmonary Acute infiltrate	•	+ • +	• • + IFX	• • CLB	• p	•	• • TT	• • p	• p +	•	• • •	+ • • ara-C		DL	DL		•	• p	•	• p	• • DTIC	+ • • PCZ	•	• •	• • • L-ASP	•	• • HU
Cardiac Necrosis Other cardiac Pulmonary	• • • HN <sub>2</sub>	+ + CTX	• • • + IFX	+ • • • • CLB	•	а • • L-PAM	•••	•	• p	•	• • • 6-MP	• + • • ara-C		DL	DL		•	• p	• BLEO	• p	• • • DTIC	• + • • PCZ	•	• • • •	• • • • L-ASP	•	••••••••••••••••••••••••••••••••••••••
Cardiac Necrosis Other cardiac Pulmonary Acute infiltrate Chronic fibrosis Gastrointestinal	•	+ • +	• • • + IFX	+ • • • • CLB	• p	•	• • • • TT	• • p	• p +	•	• • • • 6-MP	• + • • ara-C		DL	DL		•	• p	•	• p	• • DTIC	• + • • PCZ	•	• • •	• • • • L-ASP	•	• • • • •
Cardiac Necrosis Other cardiac Pulmonary Acute infiltrate Chronic fibrosis Gastrointestinal Nausea &	•	+ • + p	++ • • + IFX	• • • +	• p	•	• • •	• • +	• p +	•	• • •	• • • ara-C	•	DL + •	DL +	р •	• p	• p	• • DL	• p • +	++ • • • • • DTIC	• • +	•	+ • • •	а • • • <sup>L-ASP</sup>	•	•
Cardiac Necrosis Other cardiac Pulmonary Acute infiltrate Chronic fibrosis Gastrointestinal	•	+ • + p	+ • •	• • • +	• p •	• • p	•	• • p	• p +	•	•	• d • • • ara-C	•	DL + •	DL	р •	•	• p	• • DL	• p	•	d d • • • PCZ	•	• • •	• d • • • • L-ASP	•	ь • • • • • • • • • • • • • • • • • • •
Cardiac Necrosis Other cardiac Pulmonary Acute infiltrate Chronic fibrosis Gastrointestinal Nausea & vomiting	• • • ++	+ • + p	+ •	• • • +	• p •	• • p	•	• • +	• p +	•	•	• • • • • • • • • • • • • • • •	•	DL + •	DL +	р •	• p	• p •	• • DL	• p • +	•	• • •	•	d d + • •	• • <sup>-</sup> d • • • • • •	•	•
Cardiac Necrosis Other cardiac Pulmonary Acute infiltrate Chronic fibrosis Gastrointestinal Nausea & vomiting Diarrhea	• • • ++	+ • + p	+ • • +++ p	• • +	• p •	• • p	•	• • +	• p +	•	•	$\bullet \bullet \bullet d \bullet \bullet$	•	DL + •	DL +	р •	• • •	• • •	• • DL p	• p • +	•	• • •	• p • •	• • • + p	+ • • d • • • • • •	•	•
Cardiac Necrosis Other cardiac Pulmonary Acute infiltrate Chronic fibrosis Gastrointestinal Nausea & vomiting Diarrhea Constipation, ileu Pancreatitis Hepatic	• • • ++	+ • + p	++ • • •	• • +	• p •	• • p	•	• • • • •	• p + p + + • •	•	• • •	• • • •	•	DL + •	DL +	p • •	• • •	• • •	• • DL p	• p • + • +	• • • • •	• • •	• p • •	• • • • • •	+ • • d • • • • • • • • • • • • • • • •	•	•
Cardiac Necrosis Other cardiac Pulmonary Acute infiltrate Chronic fibrosis Gastrointestinal Nausea & vomiting Diarrhea Constipation, ileu Pancreatitis Hepatic Function tests Cholestatic	• • • ++	+ • + p	+ • • +++ p	• • +	• p • + • •	• • p	•	• • +	• p +	•	• • • • •	+ • • ara-C	•	DL + •	DL + • • • • • •	р •	• • •	• • •	• • DL	• p • +	•	• • •	• p • • • • • • •	• • • + p	+ • • d • • • • • • • • • • • • • • • •	•	•
Cardiac Necrosis Other cardiac Pulmonary Acute infiltrate Chronic fibrosis Gastrointestinal Nausea & vomiting Diarrhea Constipation, ileu Pancreatitis Hepatic Function tests	• • • ++	+ • + p	++ • • •	• • +	• p •	• • p	•	• • • • •	• p + p + + • •	•	• • •	• • • •	•	DL + •	DL + • • • • • •	p • •	• • •	• • •	• • DL	• p • + • +	• • • • •	• • •	• p • • • • • • •	• • • • • •	• • • • • • • • • • • • • • • • • • •	•	•
Cardiac Necrosis Other cardiac Pulmonary Acute infiltrate Chronic fibrosis Gastrointestinal Nausea & vomiting Diarrhea Constipation, ileu Pancreatitis Hepatic Function tests Cholestatic jaundice	• • • • • • • • •	+ • + p	++ • • •	• • +	• p • + • •	• • p	•	• • • • •	• p + p + + • •	•	• • • • •	• • • •	•	DL + •	DL + • • • • • •	p • •	• • •	• • •	• • DL	• p • + • +	• • • • •	• • •	• p • •	• • • • • •	+ • • • • • • • • • • • • • • • • • • •	•	•

#### Drug Toxicity Summary of the Toxic Reactions Reported to Commonly Used Chemotherapeutic Agents

Reaction	$HN_2$	CTX	IFX	CLB	BUS	L-PAM	$\operatorname{TT}$	NTU	MTX	5-FU	6-MP	ara-C	ACTD	ADR	DNR	MX	VCR	VLB	BLEO	MITO	DTIC	PCZ	DDP	CP	L-ASP	VP-16	ΠH
Renal/metabolic Toxic nephropath SIADH Urinary retention Hypomagnesemia Hypocalcemia Hypoglycemia Hyperglycemia	•	• + • •	p • •	• • • •	• • • •	• • • •	• • • •	DL • • •	, +++ • • • • • +	• • • •	• • • •	• • • •	• • • •	• • • •	• • • •	p • •	• + • •	• + • •	• • • •	+ • • •	• • • •	• • • •	DL • ++ +	•	p • • + +	• • • •	• • • •
Hematologic Marrow depression Megaloblastosis Hemolytic anemi DIC	•	• ++•	++ • •	+++ • •	• ++•	•	++ • •	•	++ + •	++ + •	++ p •	++ ++ •		• ++•	+++ • •	++ • •	• • •	+ • •	• • • +	+++ • +	+ • •	+++ • +	+ • +	DL • •	+ • +	+++ • •	+++ +++ •
Neurologic Peripheral neuropathy Cerebellar ataxia Ototoxicity Acute encephalopathy	•	• • •	• • ++	• • •	• • •	• • •	• • •	• p •	• • +	• + •	• • •	р • +	• • •	• • •	• • •	• •	DL • •	•	•	• • p	• • +	+ + •	+ • +	+ p •	• • ++	•	• •
(Neurologic—cont Stroke-like syndrome (see text for int	tinue •	•	•	•	•	•	•	• NTU	+ MTX	• 5-FU	• 6-MP	ara-C	• ACT D	• ADR	DNR	• MX	• VCR	• VLB	• BLEO	• MITO	DTIC	• PCZ	DDP	• CP	• L-ASP	• VP-16	• HU
Miscellaneous Hypertension Hypotension Raynaud's syndrome Radiation recall Hemorrhagic cystitis Hypothyroidism Pseudohypo- thyroidism Parotid pain	• • • •	• • p + •	• • • • •	• • • •	•	•	• • • •	• • • •	• • • •	• • + •	• • • •	• • • •	• • ++ • •	•	•	• p • • • •	• + • + +	• + + • • • +	• + + • •	• • • •		+ + • •	p + • •	•	• • • • •	• • • •	• • • •

**Radiation:** Radiation therapy to the mediastinum may injure the pericardium and myocardium. (dose dependent= 5% of patients with  $\geq$  4000 rads to more than 50% of their heart).... Acute pericarditis typically appears within a year of therapy and may result in tamponade. Chronic pericarditis usually causes an asymptomatic pericardial effusion presenting several years after therapy. Chronic pericarditis may resolve spontaneously or may progress to constrictive pericarditis...... Radiation injury to the myocardium can cause premature coronary artery disease. The overall incidence is low, but risk increases with higher doses, particularly with those delivered to an anterior field..... Patients with a history suggestive of myocardial ischemia who have received mediastinal irradiation **should be carefully evaluated** regardless of age. It may well be more than ten years before coronary artery disease appears. The electrocardiogram may be abnormal in many patients but **may not** predict coronary or pericardial disease. From <u>Perioperative Medicine 2<sup>nd</sup> ed.</u> Goldman, Brown. "Surgery in the Patient with Cancer" pg. 289

Abbreviations used for the drugs are as follows: nitrogen mustard, HN<sub>2</sub>; cyclophosphamide, CTX; Ifosfamide, IFX; chlorambucil, CLB; busulfan, BUS; L-phenylalanine mustard, L-PAM; thiotepa, TT; the nitroureas, NTU; methotrexate, MTX; 5-fluorouracil, 5-FU; 6-mercaptopurine, 6-MP; cytosine arabinoside, ara-C; actinomycin D, ACT D; doxorubicin, ADR; daunorubicin, DNR; mitoxantrone, MX; vincristine, VCR; vinblastine, VLB; bleomycin, BLEO; mitomycin C, MITO; Dacarbazine® (Miles), DTIC; procarbazine, PCZ; cisplatin, DDP; carboplatin CP;

L-asparaginase, L-ASP; etoposide, VP-16; and hydroxurea, HU.

The side effects to the nitrosoureas are quite similar and these agents have not been subcategorized. Several agents have been omitted: mithramycin, which causes hypocalcemia, liver toxicity, and facial flushing; and hormonal agents (androgens, estrogens, anitestrogens, progestigens, and adrenal corticosteroids), which cause uniform predictable side effects characteristic of each hormone. Experimental drugs and a few other little-used agents have been omitted.

#### Key:

- indicates a side effect has not been reported.
- p indicates a side effect is possibly associated or has been reported very rarely.
- + indicates a side effect has been observed and may, on occasion, present a clinical problem.
- ++ indicates a common and/or unusually severe side effect.
- DL indicates a dose-limiting side effect.

Chemotherapy Source Book Michael Perry, M.D., ACP, 1992 pg. 1141-1143

### Approximate Risk for Transmitting an Infectious Agent via Blood Products

Hepatitis B virus .....1 in 63,000

Hepatitis C virus ......0.03%/unit transfused (1990. With improved testing in 1992, may now be lower.)

(90% post-transfusion hepatitis

HIV infection ......\*1:450,000-1:660,000 per transfused unit of blood

The period between viral infection and its detection by tests for the presence of antibodies is approximately 22 days.

\* Higher rates may occur in areas with increased HIV prevalence.

\* HIV-1 Ag. tests (donor screening) are expected to prevent 25% of window period cases (5–10 cases/year).

#### **Other Infectious Diseases**

Bacterial sepsis Babesia Malaria Syphilis All rare; no accurate data available. Parasitic and bacterial—possibly 1:1,000,000 Chagas' disease 1:42,000 FDA reported cases, 1976–1985=26 deaths due to bacterial contamination of blood components RBC 1:500,000 PHS 1:10,000-20,000

Nonhemolytic transfusion reactions occur in approximately 1-5% of all transfusions.

The estimated risk of ABO-incompatible transfusion is 1:12,000 RBC transfusions.

The probability of a fatal hemolytic transfusion reaction is uncertain with estimates ranging from 1:500,000 to 1:800,000 (1976–1985=131 fatal ABO-incompatible transfusions reported to FDA.)

Approximately 12,000,000 units of RBCs are transfused each year in the U.S. More than 7,000,000 units of platelets are transfused each year in the U.S. Approximately 2,000,000 units of FFP are transfused each year in the U.S. Almost 1,000,000 units of cryoprecipitate are transfused each year in the U.S.

Modified from Dodd, RY: Risk of transfusion-transmitted disease declines: editorial, *N Engl J Med* 326:419, Aug. 6, 1992.

*Practice Guidelines for Blood Component Therapy*. Anesthesiology, 1996. pp. 732-747. A Report by the American Society of Anesthesiologists Task Force on Blood Component Therapy.

*Blood Supply: Transfusion Associated Risks.* General Accounting Office Feb. 1997. Report to the Ranking Minority Member, Committee on Commerce, House of Representatives.

Department	Extension	Pager
ADMITTING	9-4222	#0886
ANESTHESIA		#6006/#7777
CATH LAB	2-3391	—
CARDIOLOGY CLINIC	9-8888	—
Clinic FAX		
Cardiology FAX		
DIAGNOŠTIC CENTER		—
HEART STATION		
ECHO NURSE		
EKG TECH		
ER		
LAB		
Chemistry		
Hematology		
Microbiology		
MED RECORDS		
PASTORAL CARE		
PHARMACY		
Inpatient		
Outpatient.		
PRE-SURGICAL TESTING		
FAX		
PA Barb Sink		#2282
Anesthesiology		
PA Christy Shearer		
Dr. Hurlbert	9-4081	
PULMONARY LAB		
RADIOLOGY		
SOCIAL SERVICE		
SURGERY SCHEDULE		
THORACIC		
TRANSCRIPTION		
UNIVERSITY HOUSE		

# **Frequently Used Numbers**

# TEMPERATURE CONVERSION CHART

Degree F°	Degree $C^{\circ}$	<b>Degree F</b> °	Degree $C^{\circ}$
96.8	36.0	100.6	38.1
97.0	36.1	100.8	38.2
97.2	36.2	100.9	38.3
97.3	36.3	101.1	38.4
97.5	36.4	101.3	38.5
97.7	36.5	101.5	38.6
97.9	36.6	101.7	38.7
98.1	36.7	101.8	38.8
98.2	36.8	102.0	38.9
98.4	36.9	102.2	39.0
98.6	37.0	102.4	39.1
98.8	37.1	102.6	39.2
98.9	37.2	102.7	39.3
99.1	37.3	102.9	39.4
99.3	37.4	103.1	39.5
99.5	37.5	103.3	39.6
99.7	37.6	103.5	39.7
99.9	37.7	103.6	39.8
100.0	37.8	103.8	39.9
100.2	37.9	104.0	40.0
100.4	38.0		

• Calculation of creatinine clearance (CCr)

$$CCr (male) = \frac{(140 - age) \times wt (in kg)}{Serum creatinine} \times 72$$
$$CCR (female) = 0.85 \times CCr (male)$$

• Alveolar-arterial oxygen gradient (Aa gradient)

Aa Gradient = 
$$\left[ (713) (Flo_2) - \left( \frac{PaCO_2}{0.8} \right) \right] - Pao_2$$

Normal Aa gradient = 5-15 mm

 $Flo_2 = Fraction of inspired oxygen (normal = 0.21-1.0)$   $Paco_2 = Arterial carbon dioxide tension (normal = 35-45 mmHg)$   $Pao_2 = Arterial partial pressure oxygen (normal = 70-100 mmHg)$ Differential diagnosis of Aa gradient:

Abnormality	15% 0 <sub>2</sub>	100% 0 <sub>2</sub>
Diffusion defect Ventilation/Perfusion mismatch	Increased gradient Increased gradient	Correction of gradient Partial or complete correction of gradient
Right-to-left shunt (intracardiac or pulmonary)	Increased gradient	Increased gradient (no correction)

\* Anion gap

$$AG = Na^{+} - (CI^{-} + HCO_{3})$$

• Fractional excretion of sodium

$$FE_{Na} = \frac{U_{Na}/P_{Na}}{U_{Cr}/P_{Cr}} \times 100$$

• Serum osmolality

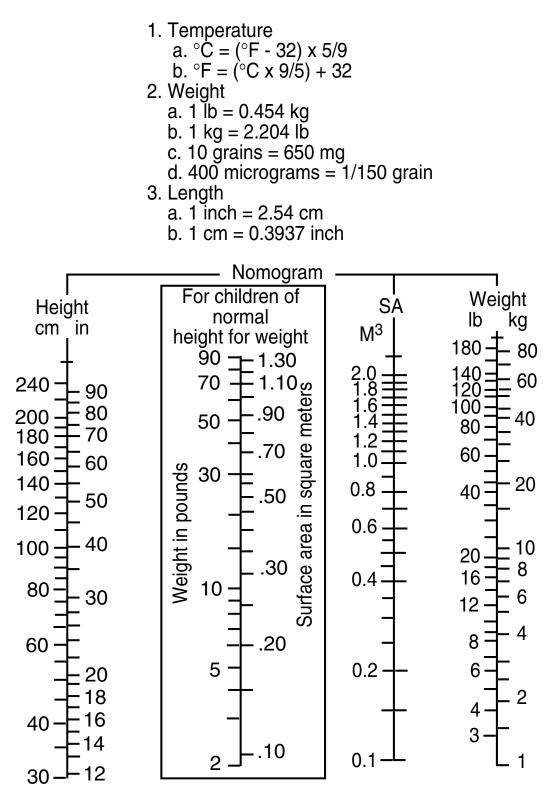
$$Osm = 2 (Na^{\dagger} + K^{\dagger}) + \frac{Glucose}{18} + \frac{BUN}{2.8}$$

Corrected sodium in hyperglycemic patients

Corrected Na<sup>+</sup> = Measured Na<sup>+</sup> + 1.6 x 
$$\frac{\text{Glucose - 140}}{100}$$

Water deficit in hypernatremic patients

Water deficit (in liters) = 0.6 x body weight (kg) x  $\frac{\text{Measured serum sodium}}{\text{Normal serum sodium}}$ 



# **BMI Metric Formula**

BMI = Weight [in kilos] divided by (Height [in meters] X Height [in meters]) or:

# **BMI Pounds/Inches Formula**

BMI = Weight [in pounds] X 704.5 divided by (Height [in inches] X Height [in inches])