The 12 Lead ECG in

Preoperative Cardiovascular Risk Assessment

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www.ECGtraining.org

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2012 – Present: Bayfront Health Dade City Cardiovascular Clinical Coordinator Stroke Coordinator Principal Investigator, "Simple Acute Coronary Syndrome (SACS Risk Stratification Score – Scientific Validation Study and Comparison to Modified TIMI and HEART ACS Risk Stratification Scores, NIH #NCT

1994 – 2012: St Joseph's Hospital, Tampa, FL Interventional Cardiovascular Technologist Cardiac Electrophysiology Technologist 12 Lead ECG Instructor, Education Department

2010: Author, Editor, "<u>12 Lead ECG Interpretation in Acute Coronary</u> <u>Syndrome with Case Studies from the Cardiac Catheterization Lab</u>," 310 page textbook marketed by Ingram Book Company.

-1982 – present, Paramedic (National Registry, Pennsylvania, Florida)

- -1982 present, AHA ACLS Instructor
- -1988 present, AHA PALS Instructor

Course Reference Sources:



From: 2014 ACC/AHA Guideline on Perioperative Cardiovascular Evaluation and Management of Patients Undergoing Noncardiac Surgery: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines

J Am Coll Cardiol. 2014;64(22):e77-e137. doi:10.1016/j.jacc.2014.07.944



Stoelting's Anesthesia and Co-Existing Disease, 5th Edition

EXPERT CONSULT - ONLINE AND PRINT By Roberta L. Hines, MD and Katherine E. Marschall, MD

Course Syllabus:

- Pre-operative Verbal History for Cardiovascular Disorders
- The Normal ECG
- Bundle Branch Blocks
- Myocardial Infarction
- Myocardial Ischemia
- Old MI
- Brugada Syndrome
- Long QT Syndrome (LQTS)
- Wolff-Parkinson-White Syndrome (WPW)
- Arrhythmogenic Right Ventricular Dysplasia (ARVD)
- Pericarditis / Myocarditis
- Hypertrophy
 - Hypertrophic Cardiomyopathy (HCM)
 - Valvular Disorders
 - Cor Pulmonale
- •Atrial Fibrillation /Flutter

Preoperative CV Diagnostic Tests:

- 12 Lead ECG
- Echocardiogram
- Stress Test
- CT / MRI
- Cardiac Catheterization

The need for PREOPERATIVE CAD Risk Factor Assessment

Does the NORMAL ECG rule out OBSTRUCTIVE Coronary Artery Disease ??





LEFT VENTRICULAR ANGIOGRAPHY EJECTION FRACTION = 69%



Dia Area = 4983.5 mm2 Dia Volume = 213.5 cc



"The WORST coronary vasculature I have seen in nearly 20 years -- an estimated 12,000 cases -- in the CATH LAB ... And this patient's 12 Lead ECG was essentially normal !!"

ACS Risk Stratification: HEART Score

History	Highly suspicious2Moderately suspicious1Slightly/not suspicious0
ECG	ST Depression
Age	65 +2 45-651 < 450
Risk Factors	3 + Risk Factors –or known CAD21 or 2 Risk Factors1No known Risk Factors0
Troponin	3X or more normal limit



From: 2014 ACC/AHA Guideline on Perioperative Cardiovascular Evaluation and Management of Patients Undergoing Noncardiac Surgery: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines

CLASS IIa

1. A validated risk-prediction tool can be useful in predicting the risk of perioperative MACE in patients undergoing noncardiac surgery (37,114,115). (Level of Evidence: B)

Regarding ACS Risk Stratification tools:

- Modified TIMI ACS Score has been "national standard" for several years
- "Modified TIMI Risk Score CANNOT be used to identify low-risk chest pain in the ED" Emerg Med J 2014 Apr; 31 (14):281-5
- "Prospective Validation of HEART Score for Chest Pain patient in the ED," Int J Cardiol 2013 Oct 3;168(3):2153-8
- Study to validate Simple Acute Coronary Syndrome (SACS) Score + comparison to HEART and Modified TIMI: <u>www.clinicaltrials.gov</u> NIH Study ID # NCT02358148

The Simple Acute Coronary Syndrome (SACS) Score

Developed in 2009 from noting correlations between patients' SYMPTOMS, ECG RESULTS, RISK FACTOR PROFILE and CARDIAC MARKERS in approximately 12,000 patients.

A scientific study to VALIDATE this score, and to compare its accuracy and reliability to the Modified TIMI and HEART Scores for predicting the presence of OBSTRUCTIVE CORONARY ARTERY DISEASE is in progress at Bayfront Health St Petersburg and Bayfront Health Dade City.

NIH Study ID # NCT02358148 www.clinicaltrials.gov

Simple Acute Coronary Syndi	rome <mark>S</mark>	core:
	POINTS ASSIGNED	TOTAL POINTS
CHECK ONE:	2	
ATYPICAL ACS - eg: EPIGASTRIC PAIN / UNUSUAL FATIGUE / WEAKNESS / DIZZINESS / DIB COLD SWEATS / NAUSEA / PALPITATIONS	1	
NONE RELEVANT	0	
ECG		
CHECK ONE: ST ELEVATION (2010 AHA/ACC Criteria) HYPERACUTE T WAVES - and/or - CONVEX ST SEGMENTS DYNAMIC ST SEGMENT and/or T WAVE CHANGES IN SERIAL EKGs ANY of ABOVE in 2 or more contiguous leads NEW or PRESUMABLY NEW LBBB	2	
 ST DEPRESSION (>0.5 mm @ J POINT) and/of INVERTED or BIPHASIC T WAVES and/of PREVIOUSLY UNDIAGNOSED Q WAVES &/or ABNORMAL R WAVE PROGRESSION (CONSISTENT WITH MYOCARDIAL INJURY /NECROSIS) (any of the above IN 2 or MORE CONTIGUOUS LEADS) 	1	
NORMAL or NON-DIAGNOSTIC EKG	0	
RISK FACTORS for CORONARY ARTERY DISEASE		
CHECK ALL THAT APPLY:	1	
SMOKING NO Hx OF CAD AGE: 65 or MORE AGE: 75 or MORE AGE: 65 or M	0	
CARDIAC MARKERS		
CHECK ONE: TROPONIN and/or CK/MB - ANY ELEVATION ABOVE YOUR INSTITUTION'S NORMAL RANGE:	1	
NORMAL	0	
TOTAL SCORE		



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Session Summary:

Session Type: Abstract Poster Session Session Title: Clinical and Hospital-Based Observational Studies Session Date and Time: Sunday Nov 8, 2015 9:00 AM - 6:45 PM Abstract Title: Validation of the Simple Acute Coronary Syndrome (SACS) Score for Identifying Obstructive Coronary Artery Disease Control Number: 20047 Presentation Number: S 3025 Board Number: 3025 Poster Presentation Time: 2:00pm - 3:15pm

HEART Score

+



?



AREAS VIEWED by 12 LEAD ECG

AVR	BASILAR SEPTAL
AVL, I	LATERAL ANTERIOR
V1, V2	ANTERIOR
	SEPTAL
POST	TERIOR (recip.)
V3, V4	ANTERIOR
V5, V6	LATERAL
II, III, A	VF INFERIOR







V1 - V4 VIEW THE ANTERIOR-SEPTAL WALL of the LEFT VENTRICLE



V1, V2 - ANTERIOR / SEPTAL V3, V4 - ANTERIOR















THE 18 LEAD ECG COVERS THE ENTIRE HEART..

CHEST LEADS V1 - V6 PLUS V4R, V5R, V6R, and WHAT EACH LEAD "SEES" . . . V7, V8, V9



To do 18 Lead ECG with 12 Lead machine – after you obtain 12 Lead, reposition CHEST LEADS to this configuration, then print !

V3

AR

V6R

V4



INDICATIONS for 18 Lead ECG include:

 - INFERIOR WALL MI
 - ST Depression in LEADS V1-V4

Quick Review Of **Essential** Waveforms

THE P WAVE

- SHOULD BE UPRIGHT, CONVEX-SHAPED DOME IN ALL LEADS EXCEPT AVR and V1
- SHOULD BE LESS THAN .2 mv (2 mm) HIGH
- SHOULD BE LESS
 THAN 100 ms (2.5mm) LONG



THE P WAVE





ATRIAL HYPERTROPHY

(SPECIFIC CRITERIA FOR ATRIAL HYPERTROPHY IS DISCUSSED IN MORE DETAIL IN THE "CHAMBER HYPERTROPHY" SECTION)
NORMAL P-R INTERVAL







QRS COMPLEX TOO WIDE WIDER THAN 120 mSEC

THINK:

- BUNDLE BRANCH BLOCK
- VENTRICULAR COMPEX (ES)
- PACED RHYTHM
- L VENTRICULAR HYPERTROPHY
- **ELECTROLYTE IMBAL.** $(\uparrow K + \downarrow C_a ++)$
- DELTA WAVE (PRE-EXCITATION)

THE "TURN SIGNAL METHOD" for identifying BUNDLE BRANCH BLOCK

USE LEAD V1 for this technique To make a **RIGHT TURN**

you push the turn signal lever **UP**....

THINK:

V1

V1

"QRS points UP = RIGHT BUNDLE BRANCH BLOCK"

To make a **LEFT TURN** you push the turn signal lever **DOWN**

THINK:

"QRS points DOWN = LEFT BUNDLE BRANCH BLOCK"

What about biphasic QRS ?



DIAGNOSING BUNDLE BRANCH BLOCK



USING LEAD V1

- QRS WIDER THAN 120 ms
- BEAT IS SUPRAVENTRICULAR IN ORIGIN
- TERMINAL PHASE OF QRS COMPLEX (LAST DEFLECTION)
 - NEGATIVE = LEFT BUNDLE BRANCH BLOCK
 - POSITIVE = RIGHT BUNDLE BRANCH BLOCK







SOME CAUSES OF LEFT BUNDLE BRANCH BLOCK (LBBB)

- CONDUCTION SYSTEM DISEASE
- OLD ANT./ SEPTAL MI (NECROSIS TO LBB)
- 🍼 CARDIOMYOPATHY
- SEVERE L.V.H. ACUTE MYOCARDITIS



SOME CAUSES OF RIGHT BUNDLE BRANCH BLOCK (RBBB)

- CONGENITAL VARIATION (IN HEALTHY HEART)
- CONDUCTION SYSTEM DISEASE
- OLD ANT./SEPTAL MI (NECROSIS TO RBB)
- PREVIOUS C.A.B.G. (RBB CUT DURING SURGERY)
- SEVERE R.V.H.
- ACUTE PULMONARY EMBOLUS
 BRUGADA SYNDROME



THE QRS COMPLEX

QRS AMPLITUDE

MAXIMUM NORMAL VALUES are difficult to define due to differences in PATIENT AGE, BODY SIZE, and FITNESS.

HOWEVER A GENERAL VALUE GUIDELINE IS: 3.0 mV (30 mm on normally calibrated EKG)

OVERALL QRS AMPLITUDE TOO HIGH: (GREATER THAN 3.0 mV / 30 mm)



VENTRICULAR HYPERTROPHY

THE QRS COMPLEX

QRS AMPLITUDE

CRITERIA FOR MINIMUM AMPLITUDE:

Abnormally LOW QRS VOLTAGE occurs when the OVERALL QRS is:

 \leq 0.5 mV IN ANY LIMB LEAD

– and –

 \leq 1.0 mV IN ANY PRECORDIAL LEAD

OVERALL QRS AMPLITUDE TOO LOW: (VERTICAL QRS SIZE)

THINK (in absence of obvious OBESITY):

- MYOCARDITIS / CONSTRICTIVE PERICARDITIS EFFUSIONS / TAMPONADE
- COPD c HYPERINFLATION
- AMYLOIDOSIS (abnormal protein accumulation in organs)
- SCLERODERMA (abnormal hardening of skin)
- HEMACHROMOTOSIS

MYXEDEMA

(excessive iron buildup in blood / organs)

(thyroid disorder)



THE MARKERS of ACUTE CORONARY SYNDROME

NORMAL ST - T WAVES

- WHEN QRS WIDTH IS NORMAL (<120 ms)

ASSESS:



- J POINT: ISOELECTRIC (or < 1 mm dev.)
- ST SEG: SLIGHT, POSITIVE INCLINATION
- T WAVE: UPRIGHT, POSITIVE -

in EVERY LEAD EXCEPT aVR !!

THE S-T SEGMENT

SHOULD HAVE A "SLIGHT POSITIVE" INCLINATION

THE S-T SEGMENT

SHOULD BE "CONCAVE" IN SHAPE . . .

THE S-T SEGMENT

AS OPPOSED TO "CONVEX" IN SHAPE

SHOULD BE "CONCAVE" IN SHAPE . . .

		NORMAL STATE OF PERFUSION	
CHANGES ASSOCIATED	C A R D I	ARTERIAL BLOCKAGES> NONE SIGNIFICANT CELLULAR OXYGENATION> NORMAL CELLULAR METABOLISM> AEROBIC CELLULAR FUNCTION> NORMAL CONTRACTION EKG: J POINT ISOELECTRIC, ST SEG "SLIGHT, POSTIVE INCLINATION, T WAVE POSITIVE	
		ISCHEMIA	1
WIIH		ARTERIAL BLOCKAGES> PARTIAL OBSTRUCTION	1
CELLULAR	C E	CELLULAR METABOLISM> AEROBIC	
PERFUSION	Ŀ	PATIENT SYMPTOMS \longrightarrow POSSIBLE, WITH EXERTION	$I \sim $
		EKG: J POINT DEPRESSED, ST SEGMENT VARIES, T WAVE VARIES	
		INFARCTION	
THE:	RF	ARTERIAL BLOCKAGES> TOTAL OBSTRUCTION CELLULAR OXYGENATION> NONE CELLULAR METABOLISM> ANAEROBIC CELL BEGINS TO	
- QRS	S I	BURN GLYCOGEN RESERVES CELLULAR FUNCTION> STOPS CONTRACTING PATIENT SYMPTOMS> TYPICAL or ATYPICAL ACS Sx	Ŷ
- J POINT	O N	EKG - INDICATIVE: J POINT ELEVATES, ST SEGMENT CONVEX, T WAVE POSITIVE, MAY EKG - RECIPROCAL: J POINT DEPRESSES, ST SEGMENT DOWNSLOPING, T WAVE INVERTE	Y ENLARGE D
- ST SEGMENT	e	NECROSIS	
- T WAVE	S T A T E S	ARTERIAL BLOCKAGES \longrightarrow TOTAL OBSTRUCTION CELLULAR OXYGENATION \rightarrow NONE CELLULAR METABOLISM \rightarrow CELL DIES WHEN GLYCOGEN RESERVES DEPLETED. CELLULAR FUNCTION \longrightarrow NONE. CELL DEAD. PATIENT SYMPTOMS \longrightarrow POSS. HYPOTENSION, DEATH	\mathcal{V}
		EKG-INDICATIVE: J POINTS, ST SEGMENTS NORMALIZE; ABNORMAL Q WAVES FORM EKG-RECIPROCAL: J POINTS, ST SEGMENTS NORMALIZE; ABNORMAL TALL R WAVES FORM	



THE T WAVE



REMEMBER, IN LEAD AVR *EVERYTHING* IS "UPSIDE-DOWN"

Leads where the T WAVE may be INVERTED:



An inverted T wave in TWO OR MORE CONTIGUOUS LEADS = potential problem (ischemia)

THE T WAVE



AMPLITUDE GUIDELINES:

- IN THE LIMB LEADS, SHOULD BE LESS THAN 1.0 mv (10 mm)
- IN THE PRECORDIAL LEADS, SHOULD BE LESS THAN 0.5 mv (5 mm)
- SHOULD NOT BE TALLER THAN R WAVE IN 2 OR MORE LEADS.



ECG CRITERIA for DIAGNOSIS of STEMI: (ST ELEVATION @ J POINT)

*LEADS V2 and V3:

- MALES ----- 2.0 mm
- FEMALES ------ 1.5 mm

ALL OTHER LEADS: 1.0 mm or more,

in TWO or more

CONTIGUOUS LEADS

* P. Rautaharju et al, "Standardization and Interpretation of the ECG," JACC 2009;(53)No.11:982-991

Reciprocal S-T Segment Depression *may* or *may not* be present during AMI.

The presence of S-T Depression on an EKG which exhibits significant S-T elevation is a fairly reliable indicator that AMI is the diagnosis.

However the *lack of Reciprocal S-T Depression* DOES NOT rule out AMI.

ACUTE MI

COMPLICATIONS TO ANTICIPATE FOR ALL MI PATIENTS :

- LETHAL DYSRHYTHMIAS
- 👱 CARDIAC ARREST
- **FAILURE OF STRUCTURE(S)** SERVED BY THE BLOCKED ARTERY





OCCLUSION of MID - LEFT ANTERIOR DESCENDING ARTERY



ANTERIOR WALL STEMI -LEFT ANTERIOR DESCENDING (LAD) ARTERY

ANTICIPATED COMPLICATIONS based on structures fed by the LAD:

 - LV PUMP FAILURE and CARDIOGENIC SHOCK (LAD feeds approx 45% of LV muscle mass)
 - PULMONARY EDEMA

(resulting from depressed LV function)

- COMPLETE HEART BLOCK with IDIO-VENTRICULAR ESCAPE Complexes (resulting from failure of Bundle of HIS and Bundle Branches)














ST Elevation in Lead AVR with STEMI ??



ST ELEVATION in Lead AVR With STEMI.... THINK *"LEFT MAIN CORONARY* ARTERY OCCLUSION !"

When LEAD AVR shows ST Elevation:

- STEMI: consider occlusion of the Left Main Coronary Artery.
 - AILEIY.
- NSTEMI and Unstable Angina consider LMCA Occlusion – or TRIPLE VESSEL DISEASE



STEMI from Left Main Coronary Artery occlusion = GLOBAL WALL MI:

- 75% Mortality Rate
- Cardiogenic Shock
- Pulmonary Edema
- Imminent Cardiac Arrest
- Standard "90 min D2B" TOO LONG !







INFERIOR WALL STEMI -RIGHT CORONARY ARTERY (RCA) - 75-80% - CIRCUMFLEX (CX) ARTERY – 10-15%

ANTICIPATED COMPLICATIONS based on structures fed by the **RCA**:

 BRADYCARDIA (RCA feeds SA Node 85-90% of patients)
RIGHT VENTRICULAR MI (RCA feeds RIGHT VENTRICLE)
1st, 2nd AND 3rd DEGREE HEART BLOCKS (Dominant artery feeds AV Node)
If POSTERIOR and/or LATERAL WALL MI present, expect

PUMP FAILURE / CARDIOGENIC SHOCK

IN EVERY CASE of **INFERIOR WALL STEMI** You must first RULE OUT **RIGHT VENTRICULAR MI** By obtaining a RIGHT SIDED **ECG BEFORE** giving any:

- NITROGLYCERIN
- Dieuretics

NITROGLYCERIN is a CLASS III CONTRINDICATION in RIGHT VENTRICULAR MI ! !*

It WILL precipitate PROFOUND HYPOTENSION !

* AHA/ACC 2013 STEMI Guidelines



To do a RIGHT - SIDED EKG . .

MOVE leads V4, V5, and V6

to the corresponding placement on the RIGHT SIDE of patient's chest . . .









A.H.A. ACLS GUIDELINES 2000/2006

PATIENTS with RIGHT BUNDLE BRANCH BLOCK --

use J-POINTS and S-T SEGMENTS in the usual manner to screen for ACUTE MI

A.H.A. ACLS GUIDELINES

- 1. If patient has a CONFIRMED HISTORY of LBBB, rely on:
 - CARDIAC MARKERS
 - SYMPTOMS
 - RISK FACTOR PROFILE
 - HIGH INDEX OF SUSPICION

for diagnosis of STEMI

2. If patient has:

a) previously NORMAL ECGs (no LBBB)
-- or - b) no old ECGs available for comparison

consider diagnosis as STEMI until proven otherwise.



HELPFUL INDICATORS FOR ECG DIAGNOSIS OF STEMI in the presence of LBBB:

- ST ELEVATION > 5 mm

- COMPARE J POINT, ST SEGMENTS and T WAVES of previous ECG with LBBB to NEW ECG.

- CONVEX ST SEGMENT = poss. MI CONCAVE ST SEGMENT = normal

- CONCORDANT ST changes (1 mm or > ST DEPRESSION V1 - V3 or ST ELEVATION LEADS II, III, AVF)

- ST ELEVATION in LEADS II, III, and/or AVF

N. ENGL. J. MED v 348; p933 - 940 - Zimetbaum, et. al.

Patterns of ISCHEMIA:











Lead AVR views the BASILAR SEPTUM, the region perfused by the FIRST SEPTAL PERFORATOR originating from the PROXIMAL LAD.

Patients with PRIMARY ST ELEVATION in Lead AVR and GLOBAL ISCHEMIA (ST -DEPRESSION in 8 or more ECG Leads) who experience ANGINA at REST have a 75% incidence of CRITICAL STENOSIS of the LEFT MAIN CORONARY ARTERY or SEVERE TRIPLE- VESSEL DISEASE *

* 1. Circulation 2009: AHA/ACCF/HRS Recommendations for the Standardization and Interpretation of the ECG: Part VI: Acute Ischemia / Infarction;

2. Circulation 2013: ACCF/AHA Guidelines for Management of STEMI

PROXIMAL OCCLUSION of the RIGHT CORONARY ARTERY.

SUB-TOTAL OCCLUSION IF CIRCUMFLEX ARTERY.

RIGHT CORONARY ARTERY filling retrograde via COLLATERAL ARTERIES.

COLLATERAL CIRCULATION from SEPTAL PERFORATORS to RCA DISTRIBUTION.

OLD MI:

Within last 6 months ??

From: 2014 ACC/AHA Guideline on Perioperative Cardiovascular Evaluation and Management of Patients Undergoing Noncardiac Surgery: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines

MACE after noncardiac surgery is often associated with prior CAD events. The stability and timing of a recent MI impact the incidence of perioperative morbidity and mortality.

Recent MI,

defined as having occurred within 6 months of noncardiacsurgery, was also found to be an independent risk factor for perioperative stroke, which was associated with an 8-fold increase in the perioperative mortality rate From: 2014 ACC/AHA Guideline on Perioperative Cardiovascular Evaluation and Management of Patients Undergoing Noncardiac Surgery: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines

A study using discharge summaries demonstrated that the postoperative MI rate decreased substantially as the length of time from MI to operation Increased. This risk was modified by the presence and type of coronary revascularization (coronary artery bypass grafting [CABG] versus percutaneous coronary interventions [PCIs]) that occurred at the time of the MI.

Taken together, the data suggest that 60 days should elapse after a MI before noncardiac surgery in the absence of a coronary intervention.

• Q WAVES •

Normal Q Waves

caused by depolarization of the intraventricular septum

Abnormal Q Waves -

caused by:

- necrosis (old infarction)
- hypertrophy



THE QRS COMPLEX

GENERAL RULES FOR NORMAL Q WAVES - WIDTH



LESS THAN .40 (1 mm) WIDE
GENERAL RULES FOR NORMAL Q WAVES - HEIGHT





LESS THAN 1/3 THE HEIGHT OF THE R WAVE

NORMAL Q WAVES EXCEPTIONS TO THE RULES





LEAD AVR

LEAD 🎞

THE Q WAVE CAN BE ANY SIZE

NORMAL Q WAVES EXCEPTIONS TO THE RULES

THERE
SHOULD BE NO Q
WAVES PRESENT
IN LEADS: V1
V2
V3



Q WAVE RULES - SUMMARY:

- Q WAVES SHOULD BE LESS THAN .40 WIDE (1 mm)
- Q WAVES SHOULD BE LESS THAN 1/3 THE HEIGHT OF THE R WAVE
- Q WAVES CAN BE ANY SIZE IN LEADS III and AVR
- THERE SHOULD BE NO Q WAVES IN LEADS V1, V2, or V3





For Slides that are not in your handout:

www.ECGtraining.org

Select tab: ECG ID of SADS Conditions

Then choose: "ECG ID of SADS Conditions – PDF"

BRUGADA SYNDROME A DEADLY STEMI Mimic

CASE STUDY 18 -- BRUGADA SYNDROME

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

37 y/o FEMALE patient arrives via EMS after being involved in a low speed motor vehicle accident. Per EMS crew, patient was the driver and sole occupant of a car that struck a tree. Patient does not recall accident. Upon further questioning, patient admits to other episodes of syncope and near-syncope. Patient denies feeling any chest pain / pressure or shortness of breath. She states she "felt great" today, until just before the the accident, when she "suddenly felt lightheaded and must have blacked-out."

RISK FACTOR PROFILE:

● FAMILY HISTORY: MATERNAL AUNT DIED AT AGE 31, UNEXPECTEDLY. WAS RULED AS A "HEART ATTACK." THERE WAS NO PRIOR KNOWN HISTORY OF CAD.

PHYSICAL EXAM: Pt. CAO x 3, skin warm, dry, color normal. Abrasions /contusions on face (airbag deployment). Patient appears to be in excellent physical condition, states she exercises several times per week (aerobics, weight training, swimming).

VITAL SIGNS: BP: 112/66, P: , R: 20, SAO2: 100% on room air.

LABS: TROPONIN: < .04 BMP and CBC: all values within normal limits.



Brugada Syndrome -- 3 ECG Patterns:







Type 2: saddle-back type ST-segment elevation Type 3: Saddle-back type "ST-segment elevation"



Torsades de Pointes:

- Decreased to NO Cardiac Output
- Often patient PULSELESS during episode
- Causes SYNCOPE
- Often DETERIORATES into VENTRICULAR
 FIBRILLATION and CARDIAC ARREST.



Trigger for Torsades de Pointes – ECTOPIC BEAT during The "ELECTRICAL GRADIENT" phase shown above.

BRUGADA SYNDROME

- GENETIC DISORDER -GENE SCN5A, which encodes CARDIAC SODIUM CHANNELS. - CAUSES EARLY RIGHT
- VENTRICULAR SUB-EPICARDIAL REPOLARIZATION



- CAUSES RUNS OF TORSADES de POINTES, and SUDDEN DEATH from TORSADES and V-FIB.
- IS BELIEVED TO CAUSE 4 12 % of ALL SUDDEN DEATHS, and 50 % of ALL CARDIAC DEATHS where pt. has a STRUCTUALLY NORMAL HEART.

BRUGADA SYNDROME

- SEVERAL VARIATIONS of this disorder are known to exist.
- CONCEALED and NON-CONCEALED.
- The NON-CONCEALED version HAS THE V1-V3 abnormality VISIBLE at all times.



 The CONCEALED version - pt. has a NORMAL EKG at most times - a DRUG STUDY, an EP STUDY, and / or GENETIC TESTING must be done to rule out or confirm diagnosis.

BRUGADA SYNDROME

- YOUNG MALES of SOUTHEAST ASIAN DESCENT are in HIGH RISK GROUP, however this disorder affects ANY RACE or GENDER.



- BRUGADA SYNDROME is HEREDITARY.
- SUSPECT BRUGADA SYNDROME in patients with FAMILY HISTORY of BRUGADA / SUDDEN DEATH, and/or TORSADES.

BRUGADA SYNDROME - TREATMENT

ICD implantation is the only known effective treatment to date.



www.brugada.org

www.sads.org

www.QTsyndrome.ch

www.crediblemeds.org

THE Q - T INTERVAL



 BEGINNING OF QRS COMPLEX TO THE END OF THE T WAVE

- NORMAL VALUES VARY BASED ON HEART RATE
- SEVERAL WAYS TO DETERMINE NORMAL LIMITS

THE *QTc INTERVAL

*QTc = Q-T interval, corrected for heart rate

HEARI			
RAIE	MALE	FEIVIALE	
150	0.25	0.28	
125	0.26	0.29	
100	0.31	0.34	
93	0.32	0.35	
83	0.34	0.37	
71	0.37	0.40	
60	0.40	0.44	
50	0.44	0.48	
43	0.47	0.51	

Annals of Internal Medicine, 1988 109:905.

QT CORRECTION FORMULAS:

Bazett's Fredericia Framingham Rautaharju QTc=QT/ \sqrt{RR} QTc=QT/(RR)1/3 QTc=QT+0.154(1-RR) QTc=656/(1+HR/100) DETERMINING Q-T INTERVAL LIMITS THE "QUICK PEEK" METHOD (for Heart Rates 60 - 100)





ECG Indicators of Long QT Syndrome:

QTc 460ms or longer in females*

- QTc 450ms or longer in males*
- •T wave alterans
- •U waves >100% of the T wave
- •U waves merged with T waves
- •U waves >0.1mv (1mm on standard calibrated ECG)

*P. Rautaharju, et al, "<u>Standardization and Interpretation of the ECG, Part IV</u>" JACC2009;53, no. 11:982-991

WHEN LQTS IS SUSPECTED, TAKE THE FOLLOWING PRECAUTIONS

WHEN THE "QUICK PEEK" METHOD for QT INTERAL EVALUATION IS **APPLIED TO THE ABOVE ECG, WHAT IS THE RESULT?**



CASE PROGRESSION - 22 YEAR OLD FEMALE:

DIAGNOSED WITH "EPILEPSY." All anticonvulsant medications were INEFFECTIVE at Controling grand-mal seizure activity.

During visit with Electrophysiologist, patient exhibited Torsades de Pointes during EST, collapsed. DURING TDP EPISODE patient experienced "grand mal Seizure."

ICD Implanted. ECG finding also discovered in patient's infant son. Received ICD at age 5.



Torsades de Pointes:

- Decreased to NO Cardiac Output
- Often patient PULSELESS during episode
- Causes SYNCOPE
- Often DETERIORATES into VENTRICULAR
 FIBRILLATION and CARDIAC ARREST.

Etiology of Long QT Syndromes: Congenital (14 known subtypes) Genetic mutation results in abnormalities of cellular ion channels Acquired **Drug Induced** Metabolic/electrolyte induced Very low energy diets / anorexia **CNS & Autonomic nervous system disorders Miscellaneous**

Coronary Artery Disease Mitral Valve Prolapse

GENETICALLY ACQUIRED LONG QT SYNDROMES: ECG PATTERNS of 3 MOST COMMON VARIATIONS:

Туре	Current	Functional Effect	Frequency Among LQTS	ECG ^{12,13}	Triggers Lethal Cardiac Event ¹⁰	Penetrance*
LQTS1	к	Ļ	30%-35%		Exercise (68%) Emotional Stress (14%) Sleep, Repose (9%) Others (19%)	62%
LQTS2	к	Ļ	25%-30%		Exercise (29%) Emotional Stress (49%) Sleep, Repose (22%)	75%
LQTS3	Na	Ť	5%-10%		Exercise (4%) Emotional Stress (12%) Sleep, Repose (64%) Others (20%)	90%



15 year old male, suffered sudden cardiac arrest. Successful out-of-hospital

GENETICALLY ACQUIRED LONG QT SYNDROMES: ECG PATTERNS of 3 MOST COMMON VARIATIONS:

Туре	Current	Functional Effect	Frequency Among LQTS	ECG ^{12,13}	Triggers Lethal Cardiac Event ¹⁰	Penetrance*
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Moss A et al. Circulation 1995;92:2929-2934



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Rhythm strip of II showing QTc of 720 msec at admission

Etiology of Long QT Syndromes:

Congenital (14 known subtypes)

Genetic mutation results in abnormalities of cellular ion channels

Acquired

Drug Induced

Metabolic/electrolyte induced

Very low energy diets / anorexia

CNS & Autonomic nervous system disorders

Miscellaneous

Coronary Artery Disease Mitral Valve Prolapse

If patient has a PROLONGED Q-T INTERVAL, AVOID DRUGS THAT LENGTHEN THE Q-T. **Such drugs include:** -Amiodarone -Ritalin -Procainamide -Benadryl -Haloperidol -Levaquin -Erythromycin -Thorazine -Norpace -Propulcid AND MANY MORE -Tequin

www.torsades.org , & www.azcert.org

PATIENT 1: NORMAL

PATIENT 2: GENETIC REPOLARIZATION ABNORMALITIES and/or HYPOKALEMIA is PRESENT.




If patient has a PROLONGED Q-T INTERVAL, AVOID DRUGS THAT LENGTHEN THE Q-T. Such drugs include: -Amiodarone -Pseudophedrine -Procainamide -Haloperidol -Levaquin -Erythromycin -Thorazine -Norpace -Propulcid -Tequin AND MANY MORE AND MANY MORE www.torsades.org / JAMA See:







Q: What is the ideal medication to treat Torsades?

Q: What is the ideal medication to treat Torsades?

A: Magnesium Sulfate, 1 – 2 grams over 5 – 60 minutes (AHA ACLS)

ABSOLUTELY **NO DRUGS** THAT PROLONG THE **O-T INTERVAL!!**

ECG Indicators of Long QT Syndrome:

•QTc 460ms or longer in females*
•QTc 450ms or longer in males*
•T wave alterans
•U waves >100% of the T wave
•U waves merged with T waves

 •U waves >0.1mv (1mm on standard calibrated ECG)

*P. Rautaharju, et al, "<u>Standardization and Interpretation of the ECG, Part IV</u>" JACC2009;53, no. 11:982-991

WHEN LQTS IS SUSPECTED, TAKE THE FOLLOWING PRECAUTIONS

Long QT Syndrome: T wave Alterans:

ECG Indicating T Wave Alternans



ECG Indicators of Long QT Syndrome:

•QTc 460ms or longer in females*
•QTc 450ms or longer in males*
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WHEN LQTS IS SUSPECTED, TAKE THE FOLLOWING PRECAUTIONS

THE U WAVE

- SEEN INFREQUENTLY

U WAVES PANIC VALUES (CONSIDERED INDICATOR OF LONG QT SYNDROME) WHEN:

- 100% or more SIZE OF T WAVE
- MERGED WITH T WAVE
- MORE THAN 1mm IN HEIGHT

THE U WAVE



- MOST VISIBLE IN LEADS V2 & V3

- OFTEN NOT PRESENT IN LEADS II, III, AVF





When ECG Indicators of Long QT Synrome are present:

- Obtain a thorough patient history, to rule out incidence of syncope and family history of sudden death/ near sudden death.
- Evaluate patient's meds list for meds that prolong the QT Interval.
- Rule out hypothermia
- Rule out CVA
- Evaluate the patient's electrolyte levels, and
- MONITOR PATIENT'S ECG FOR RUNS OF TORSADES
- Consider "expert consult" (electrophysiologist) to rule out LQTS

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- Rule out CVA
- Evaluate the patient's electrolyte levels, and
- MONITOR PATIENT'S ECG FOR RUNS OF TORSADES
- Consider "expert consult" (electrophysiologist) to rule out LQTS

LQTS + ECTOPY = Prisk of Torsades de Pointes!

Suspected LQTS Considerations include:

•Avoidance of Meds that are known to prolong the QT Interval. (refer to LIST OF MEDS KNOWN TO PROLONG THE QT INTERVAL).

TREATMENT OF TORSADES de POINTES per AHA ACLS:

-TRANSIENT: MAGNESIUM SULFATE 1 – 2 gm IV infusion over 5 – 60 minutes.

-PERSISTENT, PATIENT UNSTABLE: DEFIBRILLATION (prior to 2010: Synchronized Cardioversion)

-CARDIAC ARREST: FOLLOW Ventricular Fibrillation Algorithm.

LQTS / Brugada – Anesthesia:

- Avoid Triggers of QT Prolongation and Torsades
- Maintain:
 - -Normothermia
 - Normoxia
 - Euglycemia
 - Normocarbia
- Provide Peri-op:
 - Anxiolysis
 - Beta Blockade
 - Analgesia

LQTS / Brugada – Anesthesia, con't:

- Avoid Hemodynamic Extremes:
 - Bradycardia / Tachycardia
 - Hypo / Hypertension
- Correct Syrum Electrolytes, especially:
 - Potassium (K+)
 - Magnesium (Mg++)
- Prevent & Treat Dysrhythmias:
 - Monitor ECG in 2+ leads (II, V1, V5)
 - Have Defibrillator / Pacer available
 - Consult Cardiology as needed

Anesthesia Related Drugs:

- Midazolam has not been shown to prolong QT interval
- Propofol produces an insignificant QT prolongation compared to Thiopental
 - Another study shortens QT interval
 - Preferred agent for maintenance (TIVA)
- Vecuronium and Cisatracurium show no QT interval prolongation
- Succynlcholine consistently prolonged QT interval

Anesthesia Related Drugs, Con't:

- Reversals:
- neostigmine-atropine,
- edrophonioum-atropine,
- neostigmine-glycopyrrolate
 - All Prolong the QT interval
- Droperidol 0.75mg IV and Zofran 4mg IV produced similar QT prolongation
- Droperidol 0.625mg did not produce a significant prolongation of the QT interval, compared to saline placebo (separate study)

Volatile Agents

- Sevoflurane, Isoflurane and Desflurane at 1 MAC all prolong the QT interval
- Halothane significantly shortens QT interval
- But Halothane also sensitizes cardiac myoctyes to catecholamines
- Should be avoided in susceptible patients

Agents have all been administered safely with peri-operative beta blockade, in patients with known LQTS.

Regional Anesthesia

Study in ASA I and II male patients undergoing elective surgery under spinal anesthesia showed significant QT prolongation after onset of blockade

WOLFF-PARKINSON-WHITE EKG CHARACTERISTICS



WOLFF-PARKINSON-WHITE EKG CHARACTERISTICS



WOLFF-PARKINSON-WHITE EKG CHARACTERISTICS











Wolff-Parkinson-White + A-fib = DISASTER

37 y/o male

Chief Complaint: Lightheadedness, Palpitations, Shortness of Breath

HPI: Sudden onset of above symptoms approx. 1 hour ago

PMH: HTN (non-compliant)

37 y/o male

PE: Alert, oriented, restless, cool, pale, dry skin. PERL, No JVD, Lungs clear.Abd soft non tender, Extremities: WNL, no edema

Meds: None, NKDA

VS: BP 106/50, P 180, R 26, SAO2 93%




Physician correctly identified Atrial Fibrillation with Rapid Ventricular Response.

However did NOT identify the Wolff-Parkinson-White component.

Patient was given Diltiazem – promptly converted to -VENTRICULAR FIBRILLATION.

AHA ACLS 2010 STANDARDS WIDE COMPLEX TACHYCARDIA (QRS > 120 ms) MONOPHASIC **ABC** s **NO PULSE** PULSE - UNSTABLE **PULSE - STABLE** IMMEDIATE SYNC. O2, IV-IO, EKG GO TO MEDS: CARDIOVERSION: V - FIB ADENOSINE 6-12 - 100 j biphasic ALGORITHM ! (only if REGULAR) - consider sedation PROCAINAMIDE INCREASE joules (20-50mg/min) MEDS: -PROCAINAMIDE AMIODARONE (150 over 10min + -AMIODARONE 1mg/min INFUSION

AHA ACLS 2010 STANDARDS WIDE COMPLEX TACHYCARDIA (QRS > 120 ms)MONOPHASIC **ABC** s **NO PULSE** PULSE - UNSTABLE **PULSE - STABLE** IMMEDIATE SYNC. O2, IV-IO, EKG GO TO MEDS: CARDIOVERSION: V - FIB · ADENOSINE 0-12 - 100 j biphasic (or DELEVILAR) ALGORITHM ! - consider sedation PROCAINAMIDE INCREASE joules (20-50mg/min) MEDS: MUUDARUNE -PROCAINAMIDE (150 o ... <10min + -AMIODARONE 1mg/ min INTUSION





CHARACTERISTICS of W-P-W with Afib & RVR:

- WIDE COMPLEX TACHYCARDIA
- IRREGULARLY IRREGULAR R R INTERVALS !!



NO AV NODAL BLOCKERS (e.g. ADENOSINE, CALCIUM CHANNEL BLOCKERS) FOR WIDE COMPLEX TACHYCARDIAS THAT COULD **BE ATRIAL FIBRILLATION with Pre-Excitation (W-P-W)**

Arrhythmogenic Right Ventricular Dysplasia

- A genetically acquired myocardial disease associated with paroxysmal ventricular arrhythmias and sudden cardiac death.
- Characterized pathologically by fibro-fatty replacement of the right ventricular myocardium.
- The second most common cause of sudden cardiac death in young people (after HOCM), causing up to 20% of sudden cardiac deaths in patients < 35 yrs of age.
- Typically inherited as an autosomal dominant trait, with variable penetrance and expression (there is an autosomal recessive form called<u>Naxos Disease</u>, which is associated with woolly hair and skin changes).
- More common in men than women (3:1) and in people of Italian or Greek descent.
- Estimated to affect approximately 1 in 5,000 people overall.

From: 2014 ACC/AHA Guideline on Perioperative Cardiovascular Evaluation and Management of Patients Undergoing Noncardiac Surgery: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines

Arrhythmogenic Right Ventricular (RV) Cardiomyopathy and/or Dysplasia:

In 1 autopsy study examining a series of 200 cases of sudden death associated with arrhythmogenic RV cardiomyopathy and/or dysplasia, death occurred in 9.5% of cases during the perioperative period. This emphasizes the importance of close perioperative evaluation and monitoring of these patients for ventricular arrhythmia. Most of these patients require cardiac electrophysiologist involvement and consideration for an implantable cardioverter-defibrillator (ICD) for long-term management.





- 1. "Incomplete RBBB" Pattern
- 2. V1, V2 Rs pattern
- 3. Inverted T waves, symmetrical, - Global

ARVD – 12 Lead ECG Indicators

EPSILON WAVES





EARLY REPOLARIZATION

- POSSIBLY CAUSED BY ACCELERATED SUBEPICARDIAL REPOLARIZATION
- MILD S-T ELEVATION, USUALLY LESS THAN 2mm HEIGHT
- S-T ELEVATION SEEN IN LIMB AND PRECORDIAL LEADS (NEARLY ALL !)
- S-T ELEVATION MOST PROMINENT IN V-4 thru V6

EARLY REPOLARIZATION

- MAY APPEAR IDENTICAL TO ACUTE PERICARDITIS EKG
- ONE POSSIBLE DISTINGUISHING CHARACTERISTIC IS THE
 - * S-T : T RATIO IS LESS THAN 0.25 in lead V6

* ACCORDING TO: "CARDIOLOGY" the HOUSE OFFICER SERIES, PUBLISHED BY WILLIAMS AND WILKENS, 1998

EARLY REPOLARIZATION *S-T: T RATIO in LEAD V6:



LESS THAN 0.25 FAVORS EARLY REPOLARIZATION

* ACCORDING TO: "CARDIOLOGY" the HOUSE OFFICER SERIES, PUBLISHED BY WILLIAMS AND WILKENS, 1998





EARLY REPOLARIZATION

- REMEMBER TO CONSIDER THE BIG PICTURE ...
- Pt. HAS "UNEXPLAINED" DIFFUSE S-T ELEVATION IN NEARLY ALL LEADS
- Pt. IS ASYMPTOMATIC
- Pt. HAS NO REPRODUCEABLE CHEST PAIN
- Pt. IS USUALLY A YOUNG, HEALTHY, ATHLETIC MALE !!



PERICARDITIS

COMMON EKG FEATURES

- DIFFUSE S-T ELEVATION (1-2mm) IN MANY OR ALL LEADS.
- USUALLY GREATEST S-T ELEVATION NOTED IN LEADS I, II, V5 and V6
- P-R SEGMENT ELEVATION IN LEAD AVR P-R SEGMENT DEPRESSION IN LEAD II
- S-T ELEVATION (J POINT) USUALLY GREATER THAN 25% OF HEIGHT OF T-WAVE IN V-6



PERICARDITIS EKG CLASS #WR03999999

DEPRESSED P-R SEGMENT LEAD II



ELEVATED P-R SEGMENT LEAD AVR



PERICARDITIS EKG CLASS #WR03999999 * S-T : T RATIO in LEAD V6:



MORE THAN 0.25 FAVORS PERICARDITIS

* ACCORDING TO: "CARDIOLOGY" the HOUSE OFFICER SERIES, PUBLISHED BY WILLIAMS AND WILKENS, 1998



-- EKG ALERT --

- DIFFERENTIATION OF PERICARDITIS vs. EARLY REPOLARIZATION BASED ON EKG ALONE CAN BE DIFFICULT AND MISLEADING.

- THE NEXT TWO EKGs ARE FROM THE SAME ASYMPTOMATIC HEALTHY YOUNG MALE, TAKEN ON DIFFERENT DATES . . .





PERICARDITIS vs. EARLY REPOLARIZATION DIFFERENTIAL DIAGNOSIS

PERICARDITIS

- Recent FLU or VIRAL infection
- Complaint of Chest Pain
- HS=Friction Rub

EARLY REPOLARIZATION

- No Hx of recent illness.
- No chest- related complaints
- Normal Heart Sounds





CARDIOMYOPATHY -

DAMAGE or DEATH OF CARDIAC MUSCLE CELLS, USUALLY THE RESULT OF A PATHOLOGICAL PROCESS, RESULTING IN A GLOBAL DECREASE OF VENTRICULAR FUNCTION.



ETIOLOGY (NON-ISCHEMIC):

- IDIOPATHIC
- INFECTIOUS
- AUTOIMMUNE
- GENETIC
- ALCOHOLIC / TOXIC
- CARDIOVASCULAR DISEASE / CHRONIC HYPERTENSION



INFECTIOUS ETIOLOGIES:

VIRAL: COXSACKIE A & B ECHO INFLUENZA POLIO HERPES ADENOVIRUS MUMPS RUBELLA / RUBEOLA HEPATITIS B & C HIV

SPIROCHETAL: LYME'S DISEASE BACTERIAL: SALMONELLA LEGIONELLA CLOSTRIDIUM

RICKETTSIAL

FUNGAL: CRYPTOCOCCUS

PROTOZOAN:

TOXOPLASMOSIS GONDI TYPANOSOMIASIS CRUZI



SPECTRUM OF CLINICAL PRESENTATIONS:

- WEAKNESS
- DYSPNEA (often exertional)
- CONGESTIVE HEART FAILURE
- ANGINA / CHEST DISCOMFORT
- MIMIC ACUTE MI
- SYMPTOMS OF PERICARDITIS
- CONGESTIVE HEART FAILURE
- PALPITATIONS
- SUDDEN DEATH

EKG FINDINGS MAY INCLUDE:

- PERCARDITIS CHANGES

THE CARDIOMYOPATHIES

- CHAMBER HYPERTROPHY (A/V)
- Q/QS COMPLEXES
- POOR R WAVE PROGRESSION
- AV NODAL / BBB (LBBB common)
- ATRIAL FIBRILLATION
- VENTRICULAR COMPLEXES



EKG FINDINGS, con't:

- Normal EKG
- ACUTE MI (S-T ELEVATION
 > 1mm IN TWO or more consecutive leads)
THE GARDIOMYOPATHIES



CASE STUDY: 19 y/o Female presents to ER via EMS, C/O shortness of breath. Her skin is pale, clammy, and diaphoretic. EMS states they found her lethargic, with a BP of 66/38. Her O2 SAT was 79. They placed her on O2 15 LPM via NRB mask, and started IV NS KVO, then bolused her with a 250cc fluid challenge. Currently she is awake, C/O DIB, weakness and nausea. She's "had the flu" for the last 10 days.

THE GARDIOMYOPATHIES



CASE STUDY: PHYSICAL EXAM reveals JVD, BBS= Coarse Crackles in bases and mid fields, rales in the upper fields. Pt is becoming increasingly anxious by the minute, C/O increased DIB. Her family states she has been "too weak to get out of bed for the last few days." Repeat BP = 56 / 30, HR = 134, R = 36, SAO2 = 88% on 15 LPM O2 via NRB.

YOUR COURSE OF ACTION IS?



THE CARDIOMYOPATHIES



EKG FINDINGS MAY INCLUDE:

- A NORMAL EKG
- PERCARDITIS CHANGES
- CHAMBER HYPERTROPHY (A/V)
- Q/QS COMPLEXES
- POOR R WAVE PROGRESSION
- AV NODAL / BBB (LBBB common)
- ATRIAL FIBRILLATION
- VENTRICULAR COMPLEXES



THE CARDIOMYOPATHIES



CASE STUDY:

STAT ECHOCARDIOGRAM REVEALED GLOBAL HYPOKINESIS, EF < 20%. NO PERICARDIAL EFFUSION NOTED. NO VALVULAR DYSFUNCTION NOTED.

NEXT COURSE OF ACTION?



NORMAL EJECTION FRACTION =

55 - 70%



NORMAL LEFT VENTRICULAR FUNCTION





THE CARDIOMYOPATHIES



CASE STUDY:

IABP INSERTED DURING CARDIAC CATH.

STAT TRANSFER TO REGIONAL CARDIAC TRANSPLANT FACILITY ORDERED.

PATIENT EXPERIENCED VENTRICULAR TACHYCARDIA and IRREVERSIBLE V-FIB BEFORE HELICOPTER ARRIVAL.

CARDIAC VALVE DISORDERS Such as:

•AORTIC STENOSIS •MITRAL STENOSIS

•AORTIC REGURGITATION •MITRAL REGURGITATION

result in CHAMBER HYPERTROPHY on the 12 lead ECG. When ECG Indicators of CHAMBER HYPERTROPHY Are present on the 12 Lead ECG, An ECHOCARDIOGRAM should Be obtained and evaluated to:

• CONFIRM HYPERTROPHY • DETERMINE ETIOLOGY (VALVULAR STENOSIS / VALVULAR REGURGITATION vs other etiology)

SYSTOLIC OVERLOAD

A CONDITION WHERE THE HEART MUST OVERCOME UNUSUAL RESISTANCE TO EJECT BLOOD. THIS RESULTS IN MUSCLE THICKENING, or HYPERTROPHY.

- VALVULAR STENOSIS
- SYSTEMIC HYPERTENSION
- PULMONARY HYPERTENSION
- CONGENITAL ABNORMALITIES

DIASTOLIC OVERLOAD

A CONDITION WHERE DURING DIASTOLE, THE CHAMBER IS OVER-ENGORGED BY EXCESSIVE BLOOD VOLUME. THIS RESULTS IN "STRETCHING" or DILATION OF THE CHAMBER.

- VALVULAR REGURGITATION - FLUID VOLUME OVERLOAD

EKG CHANGES

INCREASE IN CHAMBER SIZE and/or MASS RESULTS IN AN INCREASE IN AMPLITUDE and/or TIME IN ORDER TO ACHIEVE DEPOLARIZATION.

SIMPLY PUT, THE EKG WAVEFORMS ARE BIGGER AND LONGER THAN NORMAL IN CHAMBER ENLARGEMENT.

ATRIAL ENLARGEMENT

- IF THE P-WAVE IS TOO HIGH IN LEAD II, WE SUSPECT RIGHT ATRIAL ENLARGEMENT.
- IF THE P-WAVE IS TOO LONG IN LEAD II, WE SUSPECT LEFT ATRIAL ENLARGEMENT.

ATRIAL ENLARGEMENT

- P-WAVES THAT ARE "POINTY" IN LEAD II (as opposed to rounded) . FAVOR RIGHT ATRIAL ENLARGEMEN1
- P-WAVES THAT LOOK LIKE THE LETTER "M" FAVOR LEFT ATRIAL ENLARGEMENT

THE P WAVE

R

WHEN THE P
WAVE IS BI PHASIC IN V1, _
IT DISPLAYS
BOTH R and
L ATRIAL
DEPOLARIZATION













VENTRICULAR ENLARGEMENT



VENTRICULAR ENLARGEMENT



ENLARGEMENT OF THE RIGHT OR LEFT VENTRICLE IS FREQUENTLY ACCOMPANIED BY ENLARGEMENT OF THE CORRESPONDING ATRIUM !

- THIS MAY BE A HELPFUL CLUE IN DIAGNOSING THE EKG !

"SEE-SAW EFFECT" of RVH on R WAVE PROGRESSION



V1 V2 V3 V4 V5 V6











VENTRICULAR STRAIN PATTERNS



asymmetrical



VENTRICULAR STRAIN PATTERNS

T WAVES ARE INVERTED AND ASMMETRICAL THERE MAY BE S-T SEGMENT DEPRESSION

VENTRICULAR STRAIN PATTERNS

STRAIN PATTERNS ARE INDICATIVE OF SYSTOLIC OVERLOAD -- THE VENTRICLES HAVING TO OVERCOME GREAT FORCE TO EXPEL BLOOD.

VENTRICULAR STRAIN PATTERNS


VENTRICULAR ENLARGEMENT

I USE SEVERAL TECHNIQUES FOR IDENTIFYING RIGHT AND LEFT VENTRICULAR HYPERTROPHY.

- 1. AXIS OF LEAD I and V1
- 2. PRESENCE OF ATRIAL HYPERTROPHY
- 3. R-WAVE PROGRESSION OF V LEADS
- 4. STRAIN PATTERN OF T WAVES IN V1 - V2 and V5 - V6
- 5. MATHEMATICAL FORMULAS

*MATHEMATICAL FORMULAS FOR DETERMINING LVH and RVH

LVH

- R-WAVE V1 + S-WAVE LEAD III > 25mm
- R-WAVE V5 or V6 > 26mm
- S-WAVE V1 + R-WAVE V5 or V6 > 35mm
- LARGEST R-WAVE + LARGEST S-WAVE in V-LEADS > 45mm

RVH

- R-WAVE V1 + S-WAVE V5 or V6 > 10.5mm
- rSR' in V1 where R' \geq 10mm

* THIS IS A PARTIAL LIST.





CASE STUDY 1:

THE EKG COMPUTER MISSED THE LEFT ATRIAL ENLARGEMENT, WHICH IS A KEY FACTOR IN THE DIAGNOSIS OF THE PATIENT'S CONDITION.

WHAT ARE SOME COMMON CAUSES OF LEFT ATRIAL AND LEFT VENTRICULAR ENLARGEMENT ? ?

CASE STUDY 1:

COMMON CAUSES OF LEFT ATRIAL and LEFT VENTRICULAR ENLARGEMENT:

- AORTIC VALVE STENOSIS
- COARCTATION OF THE AORTA
- MITRAL REGURGITATION
- SYSTEMIC HYPERTENSION

CASE STUDY 1:

WHAT ASPECT OF THE PHYSICAL EXAMINATION COULD AID YOU IN MAKING A DIAGNOSIS ?

CASE STUDY 1:

WHAT ASPECT OF THE PHYSICAL EXAMINATION COULD AID YOU IN MAKING A DIAGNOSIS ?

AUSCULTATION OF HEART SOUNDS . . .

CASE STUDY 1:

WHAT ASPECT OF THE PHYSICAL EXAMINATION COULD AID YOU IN MAKING A DIAGNOSIS ?

AUSCULTATION OF HEART SOUNDS . . .

THIS PT. EXHIBITED A PRONOUNCED SYSTOLIC (S-1) MURMUR -- STRONGLY INDICATING MITRAL VALVE REGURGITATION.

CAUSE OF SYSTOLIC (S 1) MURMUR

DAMAGE TO
MITRAL and/or
TRICUSPID
VALVE(s)

CAUSES REGURGITATION





CASE STUDY 1:

SUMMARY

EKG FINDINGS OF LEFT ATRIAL and VENTRICULAR ENLARGEMENT, COMBINED WITH A PRONOUNCED S-1 HEART MURMUR INDICATE MITRAL REGURGITATION. THE LEFT VENTRICULOGRAM OBTAINED DURING CARDIAC CATHETERIZATION CONFIRM THE DIAGNOSIS. THIS PATIENT WAS SENT FOR A SURGICAL CONSULTATION. From: 2014 ACC/AHA Guideline on Perioperative Cardiovascular Evaluation and Management of Patients Undergoing Noncardiac Surgery: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines

Valvular Heart Disease

CLASS I Recommendations:

1. It is recommended that patients with clinically suspected moderate or greater degrees of valvular stenosis or regurgitation undergo preoperative echocardiography if there has been either:

1) no prior echocardiography within 1 year or

2) a significant change in clinical status or physical examination since last evaluation (60). (Level of Evidence: C)

2. For adults who meet standard indications for valvular intervention (replacement and repair) on the basis of symptoms and severity of stenosis or regurgitation, valvular intervention before elective noncardiac surgery is effective in reducing perioperative risk (15). (Level of Evidence: C) From: 2014 ACC/AHA Guideline on Perioperative Cardiovascular Evaluation and Management of Patients Undergoing Noncardiac Surgery: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines

Aortic Stenosis: Recommendation

CLASS lia

Elevated-risk elective noncardiac surgery with appropriate intraoperative and postoperative hemodynamic monitoring is reasonable to perform in patients with asymptomatic severe aortic stenosis (AS) (48,75–84). (Level of Evidence: B)

Hypertrophic Cardiomyopathy (HCM)



12 Lead ECG Traits:

- QRS Height -- exceeds normal size, "spearing through QRS in other leads"
- Inverted T waves appear in multiple regions (INFERIOR, ANTERIOR, LATERAL)
- T WAVES are SYMMETRICAL (No STRAIN PATTERN noted).

Hypertrophic Cardiomyopathy (HCM)



12 Lead ECG Traits:

- QRS Height -- exceeds normal size, "spearing through QRS in other leads
- Inverted T waves appear in multiple regions (ANTERIOR, LATERAL)
- BiPHASIC T waves in Inferior Leads.
- T WAVES are SYMMETRICAL (No STRAIN PATTERN noted).

Typical 12 Lead ECG Indicators Suggestive of HCM:

LVH Inverted T waves (predom V4-V6, II, aVF, I, L) ST Depression / Strain pattern Q waves Left Axis Dev LAE

Over 90% of patients with HCM will have an abnormal ECG.^[19–21] ECG abnormalities include T wave inversion (TWI), ST segment depression, pathological Q waves, conduction delay, left-axis deviation (LAD) and left atrial enlargement (LAE).

In a patient with KNOWN Hypertrophic Cardiomyopathy, OR a patient with NO PREVIOUS DIAGNOSIS OF HCM who presents with an ECG like the one on the previous slide, what DIAGNOSIS of HCM

..... WHAT DIAGNOSTIC TEST would be most appropriate to order next ???

(HINT....we want to assess Left Ventricular function !)

From: 2014 ACC/AHA Guideline on Perioperative Cardiovascular Evaluation and Management of Patients Undergoing Noncardiac Surgery: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines

HYPERTROPHIC CARDIOMYOPATHY (HCM) MANAGME NT CONSIDERATIONS:

Decreased systemic vascular resistance (arterial vasodilators), volume loss, or reduction in preload or LV filling may increase the degree of dynamic obstruction and further decrease diastolic filling and cardiac output, with potentially untoward results.

Over diuresis should be avoided, and inotropic agents are usually not used in these patients because of increased LV outflow gradient. Studies have reported mixed results for perioperative risk in patients with hypertrophic obstructive cardiomyopathy. Patients with HCM are at significant risk for cardiac complications during surgery and anesthesia. Although studies looking specifically at the pediatric population are lacking, one study of adults with HCM undergoing surgery and anesthesia found a 40% risk of experiencing at least one adverse cardiac event. Anesthetic management focuses on maintaining preload and afterload, avoiding increases in contractility and avoiding tachycardia. Ideally patients with HCM should be well hydrated prior to the induction of anesthesia. Agents that maintain afterload without a significant increase in contractility or heart rate are preferable for both induction and maintenance of anesthesia. Appropriate intra-operative monitoring and a postoperative care plan are dependent on the procedure and the severity of HCM.

"Anesthetic Management of Hypertrophic Cardiomyopathy and Challenges in the MRI Suite," Emory University School of Medicine, Children's Healthcare of Atlanta



My top two reasons for giving everything in life the best I have to offer. Wayne W Ruppert, 352-521-1544 wayne.ruppert@bayfronthealtth.com