TREATMENT IN BRADYCARDIA

DEFINITION OF BRADYCARDIA

- 12- LEAD ECG
- 24-hour ambulatory monitoring

DEFINITION OF BRADYCARDIA

12- LEAD ECG

- 0-3 years: <100 bpm
- 3-9 years: <60 bpm.
- 9-16 years: <50 bpm

DEFINITION OF BRADYCARDIA

24-hour ambulatory monitoring

- 0-2 years : <60 bpm/asleep <80 bpm/ awake
- 2-6 : <60 bpm.
- 6-11: <45 bpm
- >11: <40 bpm
- > 11 years who are well-trained athletes: <30 bpm

MECHANISMS OF BRADYCARDIA

- Sinus bradycardia
- AV node or the bundle of His block

CAUSES

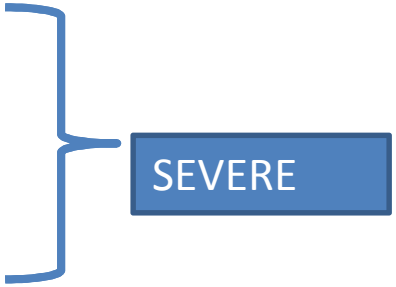
Intrinsic causes
Cardiomyopathy
Familial
Inflammatory
Myocarditis
Pericarditis
Collagen vascular disease
Systemic lupus erythematosus
Congenital Heart Disease
Atrial septal defect
Atrioventricular canal
Long QT syndrome
Pulmonary stenosis
Ventricular septal defect
Transposition of the great arteries
Wolff Parkinson White syndrome
Myocardial ischemia or infarction
Surgical trauma
Atrial operations

CAUSES

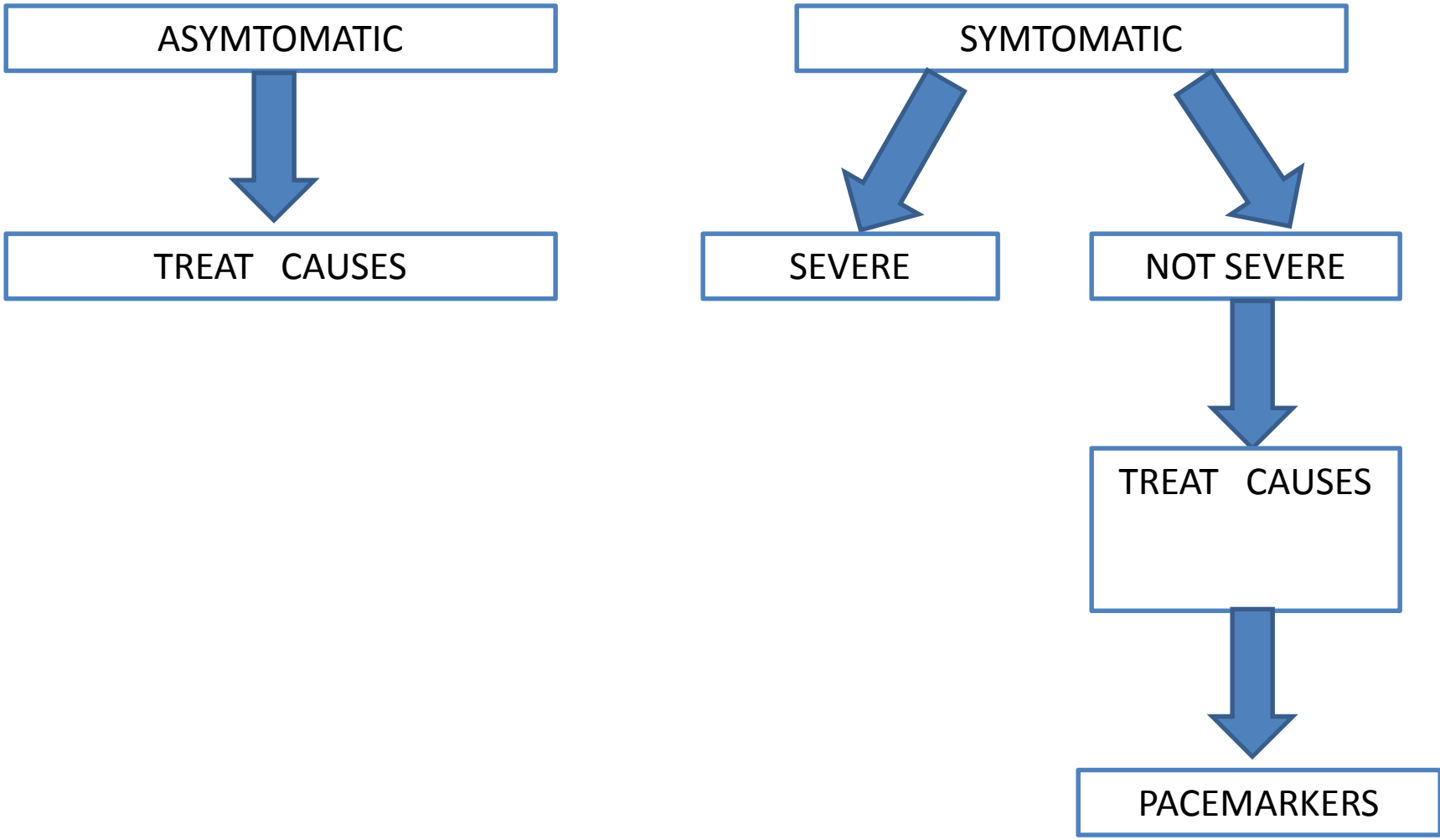
Extrinsic causes
Medications
Antiarrhythmic agents
Beta-adenergic blockers
Calcium-channel blockers
Clonidine
Digoxin
Hypothermia
Hypervagotonia
Breath-holding spells
Coughing
Esophageal or nasopharyngeal stimulation
Increased intracranial pressure
Medications
Edrophonium, physostigmine, bethanechol, neostigmine, acetylcholine, hyoscyamine, phenylephrine, methoxamine, morphine
Neurocardiac syncope
Sleep

CLINICAL PRESENTATIONS

- Asymptomatic
- Dizziness
- Syncope
- Exercise intolerance
- Poor systemic perfusion or shock
- Cardio-respiratory arrest
- Sudden death

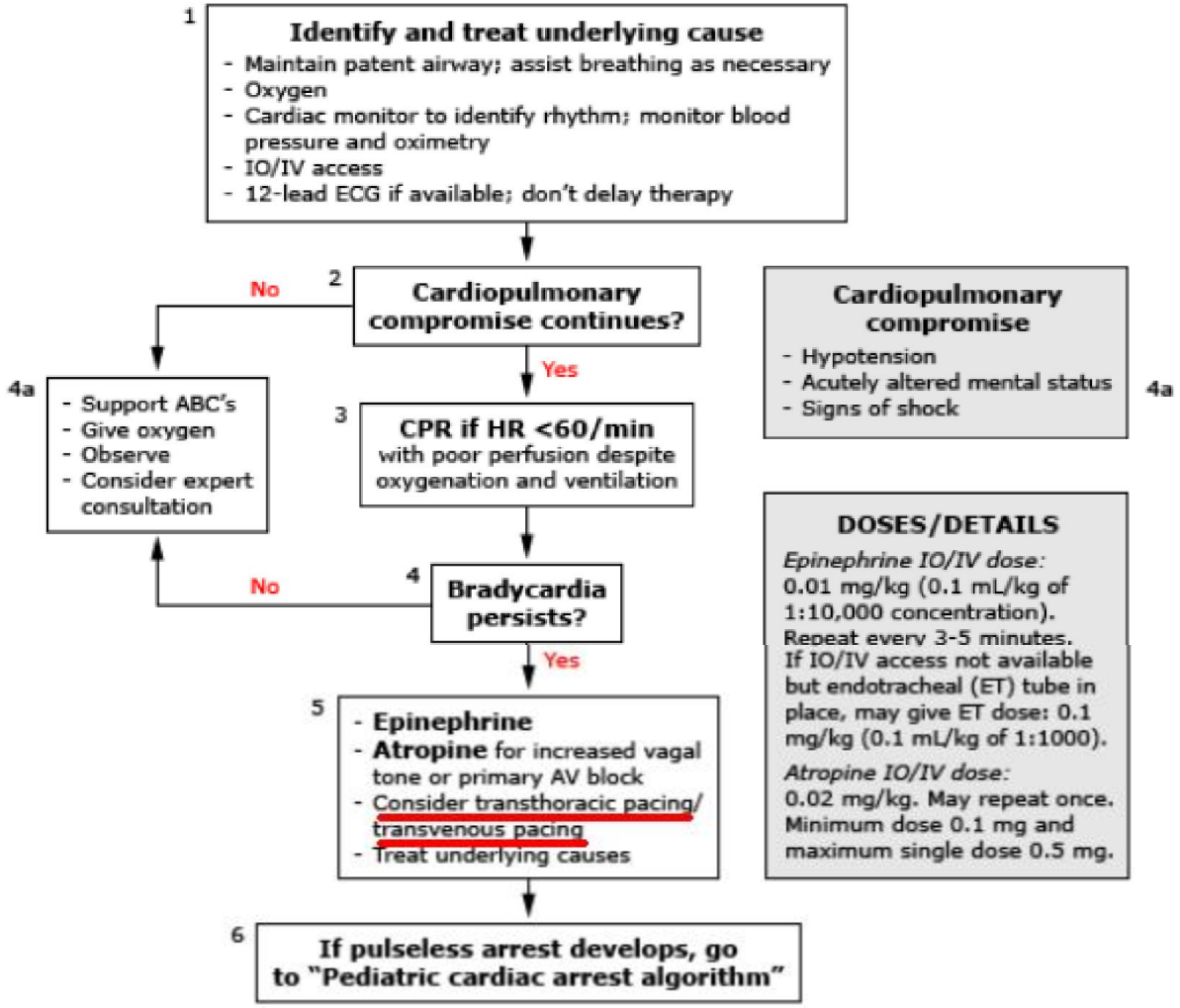


Treatment


ASYMTOMATIC
TREAT CAUSES
SYMTOMATIC
SEVERE
NOT SEVERE
TREAT CAUSES
PACEMARKERS

Treatment- Severe Bradycardia

Pediatric bradycardia algorithm (with a pulse and poor perfusion): 2010 PALS guidelines



AMERICAN
COLLEGE *of*
CARDIOLOGY

Heart Rhythm Society℠

AHA Practice Guideline

ACC/AHA/HRS 2008 Guidelines for Device-Based Therapy of Cardiac Rhythm Abnormalities

A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the ACC/AHA/NASPE 2002 Guideline Update for Implantation of Cardiac Pacemakers and Antiarrhythmia Devices): *Developed in Collaboration With the American Association for Thoracic Surgery and Society of Thoracic Surgeons*

SIZE OF TREATMENT EFFECT →

ESTIMATE OF CERTAINTY (PRECISION) OF TREATMENT EFFECT

	CLASS I *Benefit >>> Risk* Procedure/Treatment **SHOULD** be performed/ administered	CLASS IIa *Benefit >> Risk* *Additional studies with focused objectives needed* **IT IS REASONABLE** to perform procedure/administer treatment	CLASS IIb *Benefit ≥ Risk* *Additional studies with broad objectives needed; additional registry data would be helpful* Procedure/Treatment **MAY BE CONSIDERED**	CLASS III *Risk ≥ Benefit* Procedure/Treatment should **NOT** be performed/administered **SINCE IT IS NOT HELPFUL AND MAY BE HARMFUL**
LEVEL A Multiple populations evaluated* Data derived from multiple randomized clinical trials or meta-analyses	■ Recommendation that procedure or treatment is useful/effective ■ Sufficient evidence from multiple randomized trials or meta-analyses	■ Recommendation in favor of treatment or procedure being useful/effective ■ Some conflicting evidence from multiple randomized trials or meta-analyses	■ Recommendation's usefulness/efficacy less well established ■ Greater conflicting evidence from multiple randomized trials or meta-analyses	■ Recommendation that procedure or treatment is not useful/effective and may be harmful ■ Sufficient evidence from multiple randomized trials or meta-analyses
LEVEL B Limited populations evaluated* Data derived from a single randomized trial or nonrandomized studies	■ Recommendation that procedure or treatment is useful/effective ■ Evidence from single randomized trial or nonrandomized studies	■ Recommendation in favor of treatment or procedure being useful/effective ■ Some conflicting evidence from single randomized trial or nonrandomized studies	■ Recommendation's usefulness/efficacy less well established ■ Greater conflicting evidence from single randomized trial or nonrandomized studies	■ Recommendation that procedure or treatment is not useful/effective and may be harmful ■ Evidence from single randomized trial or nonrandomized studies
LEVEL C Very limited populations evaluated* Only consensus opinion of experts, case studies, or standard of care	■ Recommendation that procedure or treatment is useful/effective ■ Only expert opinion, case studies, or standard of care	■ Recommendation in favor of treatment or procedure being useful/effective ■ Only diverging expert opinion, case studies, or standard of care	■ Recommendation's usefulness/efficacy less well established ■ Only diverging expert opinion, case studies, or standard of care	■ Recommendation that procedure or treatment is not useful/effective and may be harmful ■ Only expert opinion, case studies, or standard of care

TREATMENT- NOT SEVERE

- SINUS BRADYCARDIA
- AV BLOCK

TREATMENT- NOT SEVERE SINUS BRADYCARDIA

- Causes :
  - Sick sinus syndrome
  - Exaggerated vagal activity
  - Increased intracranial pressure
  - Acute myocardial infarction
  - Obstructive sleep apnea
  - Drugs
- Atropine in acute myocardial infarction
- Chronic medical therapy for symptomatic sinus bradycardia is usually not effective
- Pacemarker

Pacing in SINUS BRADYCARDIA

- **Class I :**
  - Sinus node dysfunction with correlation of symptoms during age-inappropriate bradycardia (Level of Evidence B)
- **Class IIa:**
  - Sinus bradycardia for the prevention of recurrent episodes of intra-atrial reentrant tachycardia; SND may be intrinsic or secondary to antiarrhythmic treatment. (Level of Evidence: C)
  - Sinus bradycardia with complex congenital heart disease with a resting heart rate less than 40 bpm or pauses in ventricular rate lasting longer than three seconds, Impaired hemodynamics due to sinus bradycardia (Level of Evidence: C)

AV BLOCK

- First-degree AV block does **not** cause bradycardia
- Second-degree AV block Mobitz 1 : asymptomatic, not progress to complete block
- Second-degree AV block Mobitz 2: frequently progresses to complete heart block
- Advanced second-degree AV block: two consecutive P waves present that should but fail to conduct to the ventricle
- Third-degree AV block

TREATMENT NOT SEVERE AV BLOCK

CAUSES

- Congenital complete heart block
  - Neonatal lupus
  - Structural cardiac defects
    - Corrected transposition of the great arteries
    - Polysplenia with atrioventricular canal defect
- Acquired complete heart block
  - Myocarditis
  - Acute rheumatic disease
  - Myocardial infarction
  - Trauma
  - Injury from surgery or catheterization
  - Cardiomyopathy

Pacing in AV block

- **Class I**
  - Third and advanced second-degree AV heart block that is associated with symptomatic bradycardia, ventricular dysfunction, or low cardiac output (Level of Evidence C)
  - Children who have third or advanced second-degree AV heart block after cardiac surgery that is not expected to resolve or that persists **seven days** after surgery (Level of Evidence B)
  - Congenital third-degree AV block with a wide QRS escape rhythm, complex ventricular ectopy, or ventricular dysfunction. (Level of Evidence B)
  - Congenital third-degree AV block in the infant with a ventricular rate less than **55 bpm** or with congenital heart disease and a ventricular rate less than **70 bpm**. (Level of Evidence C)
- **Class IIa**
  - Congenital third-degree AV block beyond the first year of life with an average heart rate less than 50 bpm, abrupt pauses in ventricular rate that are two or three times the basic cycle length, or symptoms due to chronotropic incompetence. (Level of Evidence: B)
  - Unexplained syncope in the patient with prior congenital heart surgery complicated by transient complete heart block with residual fascicular block after a careful evaluation to exclude other causes of syncope. (Level of Evidence: B)

BRADYCARDIA IN FETUS

DEFINITION

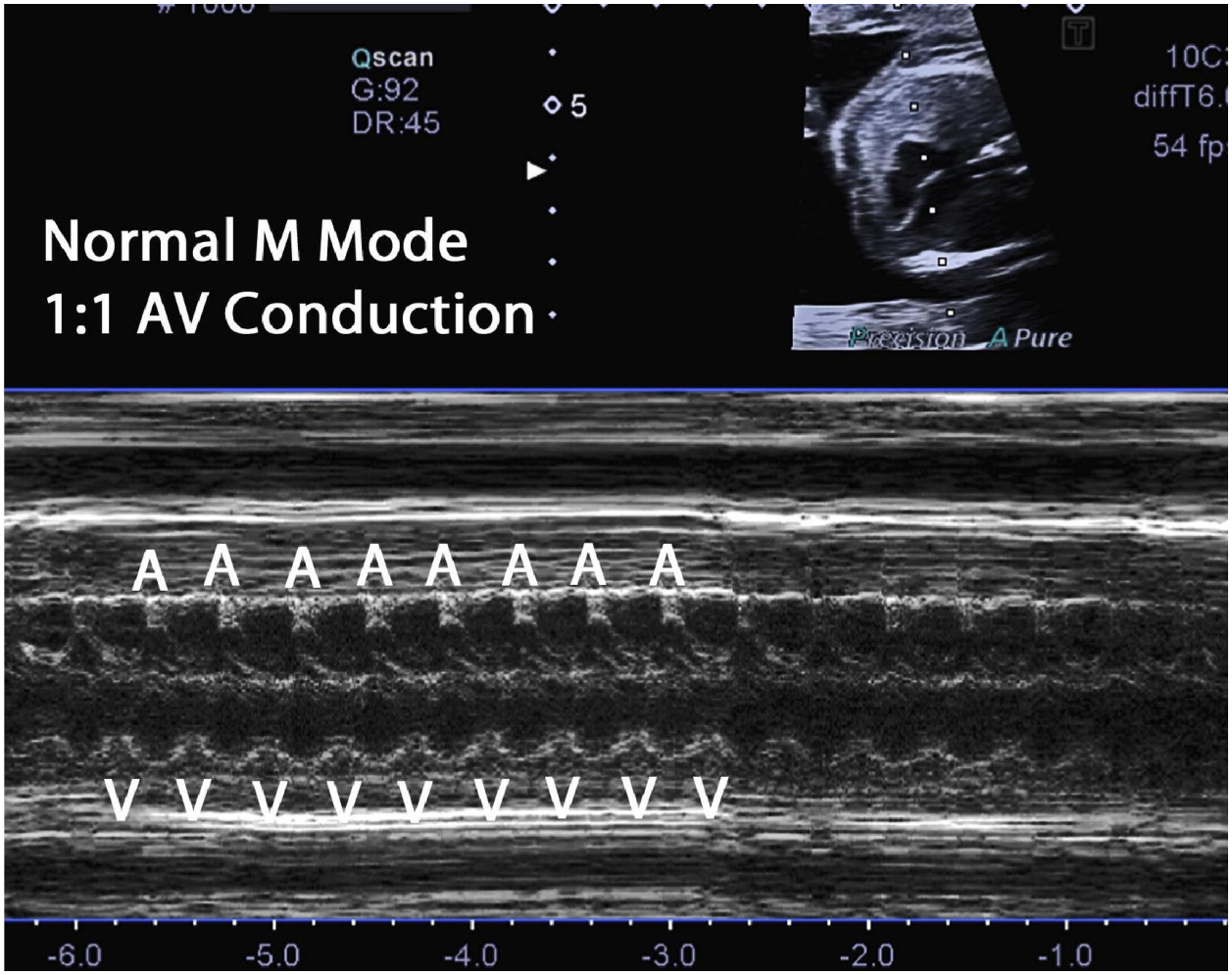
- Normal: 110 to180 bpm
- Bradycardia: < 110 bpm />10 m
- Distinguished from fetal heart rate changes in response to hypoxia

DIAGNOSIS

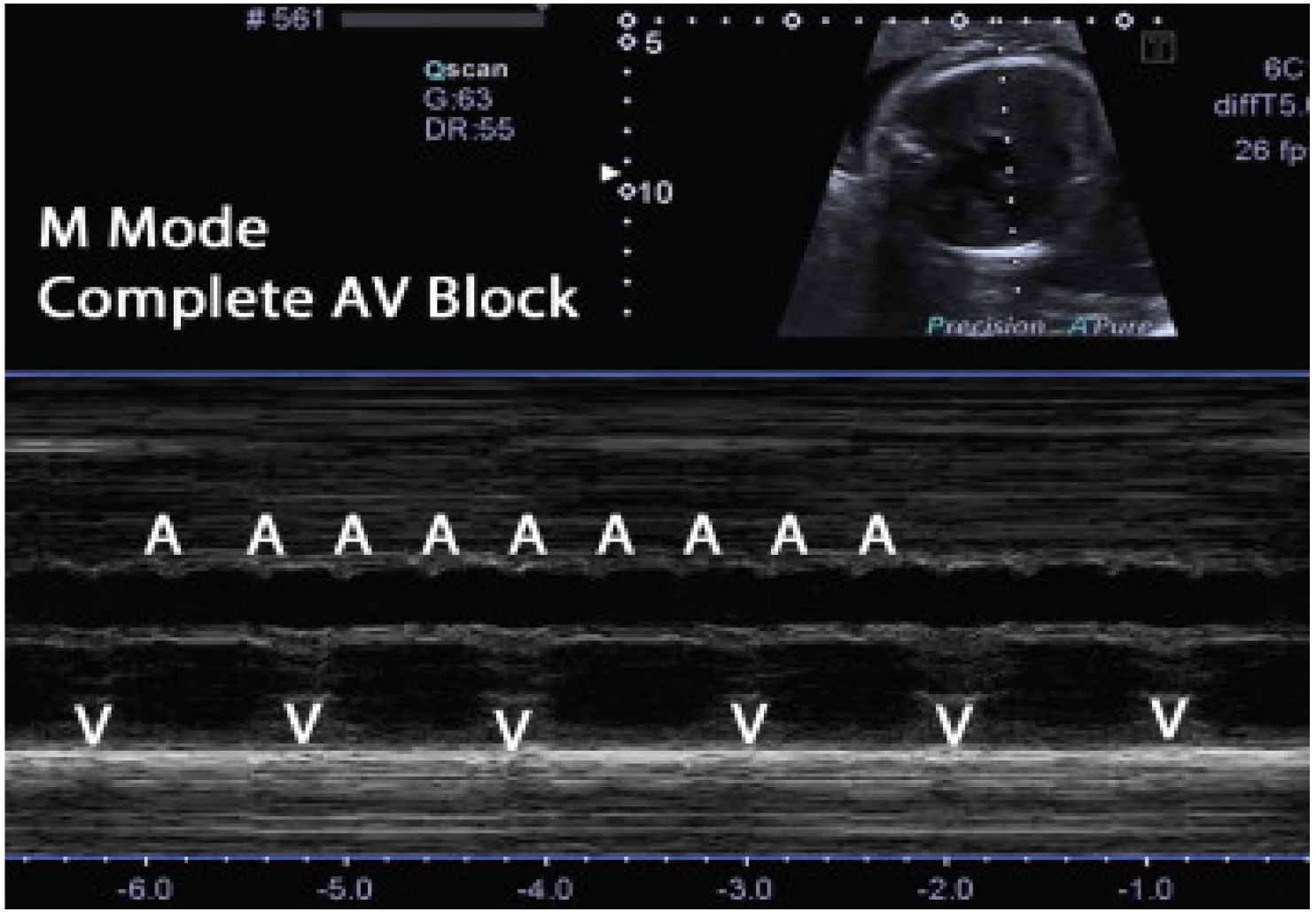
- Two-dimensional ultrasound
  - M –mode
  - Doppler

  →*Evaluation of AV Relationship and Atrial/ventricular Rate*

M-MODE


Qscan
G:92
DR:45
5
Normal M Mode
1:1 AV Conduction
A A A A A A A A
V V V V V V V V V
-6.0
-5.0
-4.0
-3.0
-2.0
-1.0

M-MODE


561
Qscan
G:63
DR:55
5
10
M Mode
Complete AV Block
A A A A A A A A A
V V V V V V
-6.0
-5.0
-4.0
-3.0
-2.0
-1.0

DOPPLER

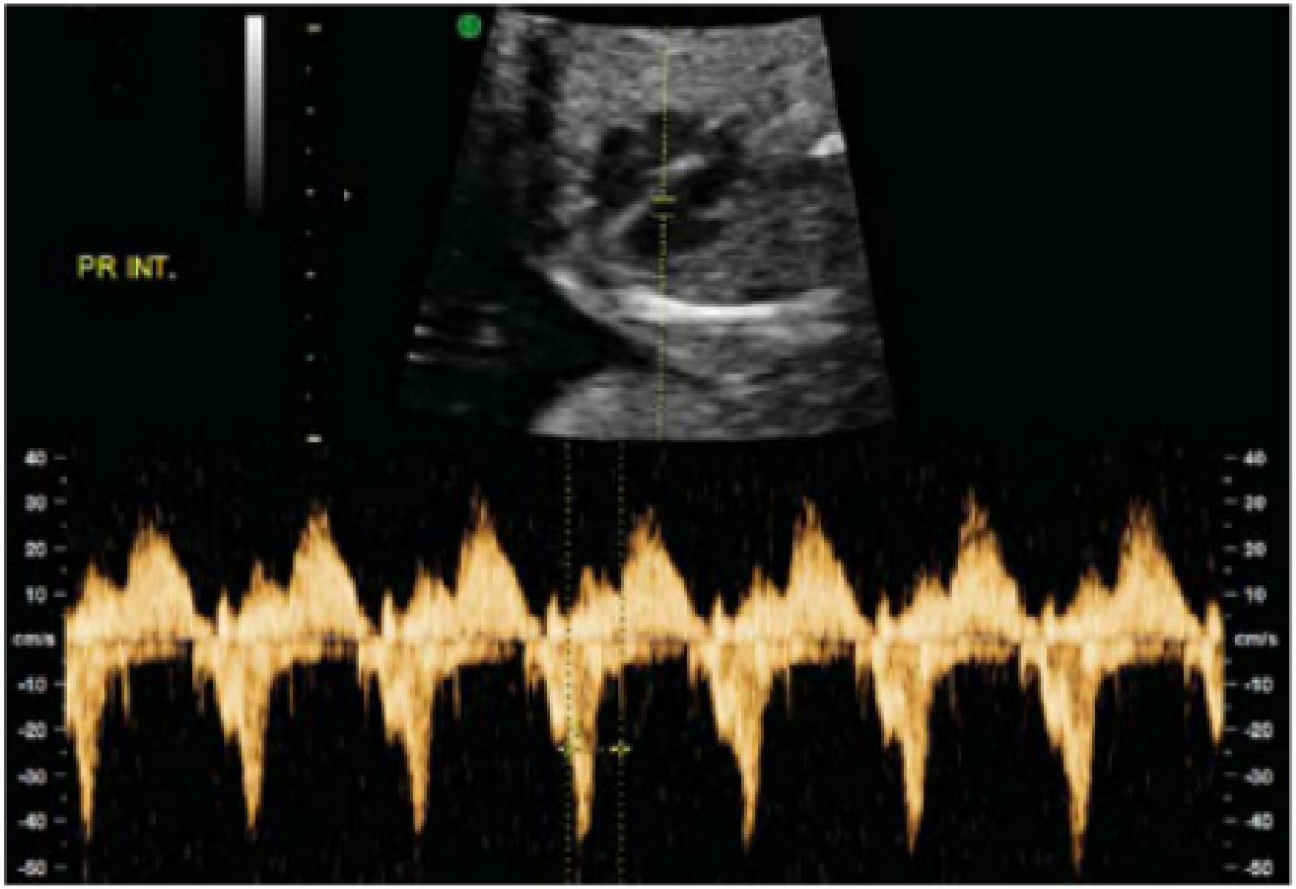
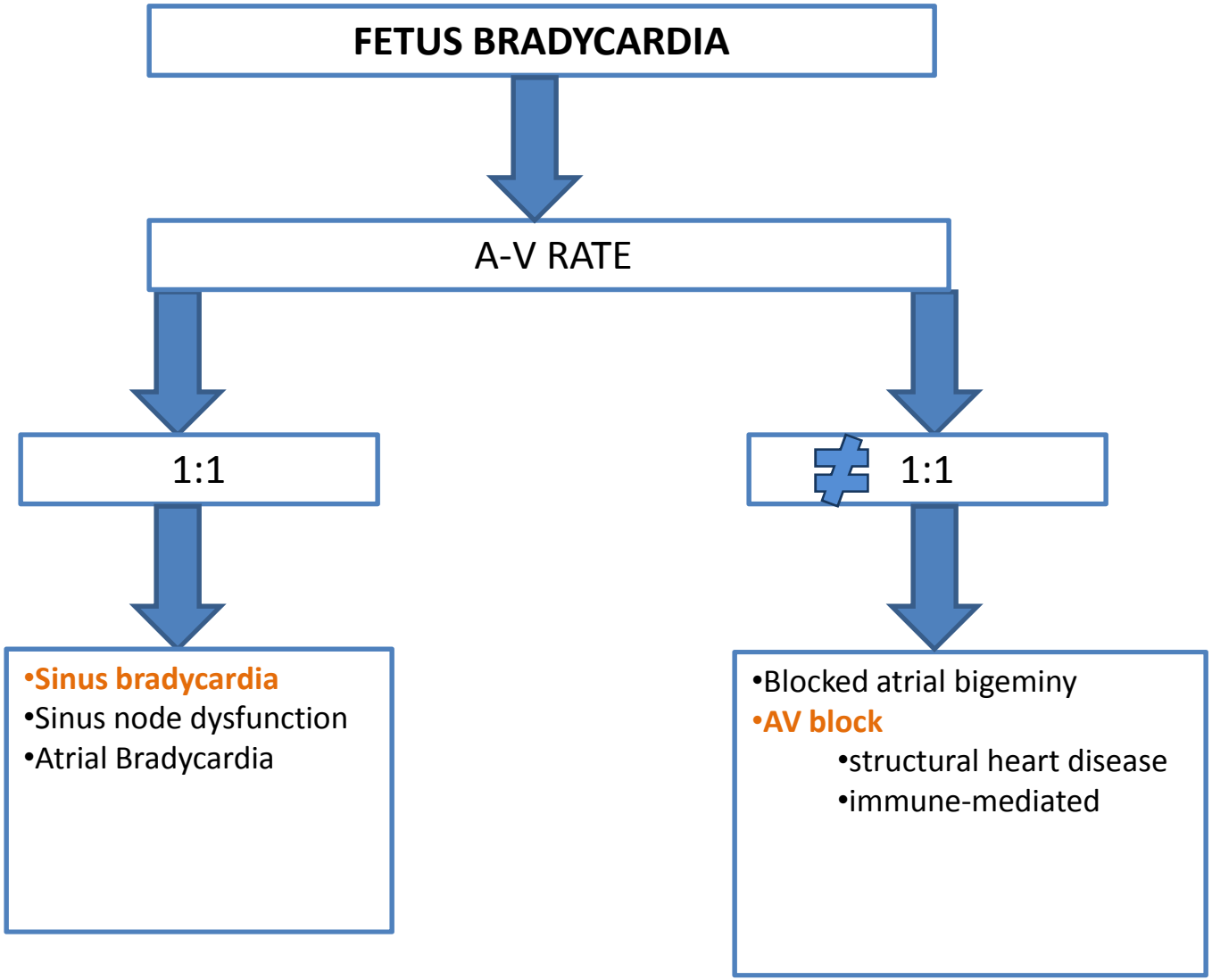


Figure 3: PPulsed Doppler of the mechanical PR interval


FETUS BRADYCARDIA
A-V RATE
1:1
≠ 1:1
•Sinus bradycardia
•Sinus node dysfunction
•Atrial Bradycardia
•Blocked atrial bigeminy
•AV block
•structural heart disease
•immune-mediated

Sinus bradycardia in fetus

- Causes **(100 to 110 bpm**)
  - Fetal distress
  - Structural cardiac anomalies: heterotaxy
  - Long Q-T syndrome
  - Marternal hypothyroidism
  - Fetal CNS abnormalities
  - Maternal medications or illness
  - Familial sinus bradycardia

Sinus bradycardia in fetus

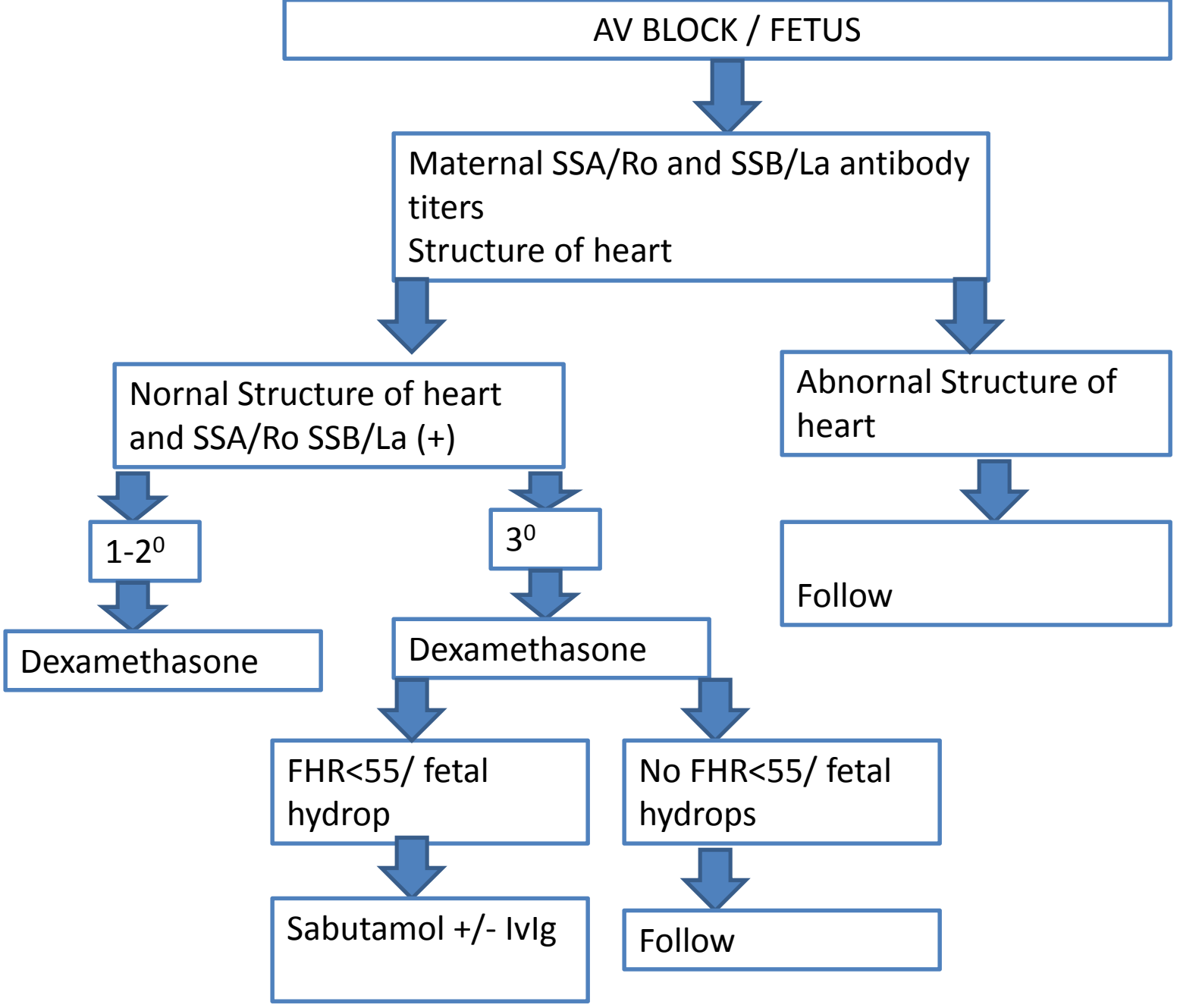
- Treatment
  - Cause
  - Weekly obstetrical follow-up

AV BLOCK in fetus

- **CAUSE (<60 bpm or 60 and 80 bpm**)
  - L-transposition of the great arteries
  - Polysplenia
  - Maternal lupus autoantibodies
    - 2-18% AVB

TREATMENT AV block in fetus

Due to the low incidence of complete AV block in the general population, studies are mainly *observational, retrospective and involve small cohorts of patients*


AV BLOCK / FETUS
Maternal SSA/Ro and SSB/La antibody titers
Structure of heart
Nornal Structure of heart and SSA/Ro SSB/La (+)
Abnornal Structure of heart
1-2⁰
3⁰
Follow
Dexamethasone
Dexamethasone
FHR<55/ fetal hydrop
No FHR<55/ fetal hydrops
Sabutamol +/- Ivlg
Follow