

Left ventricular hypertrophy on electrocardiography: -Clinical implications for adult congenital heart disease -

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The biggest children's hospital in Japan, promoting transition for adults with congenital heart disease (ACHD)



Tokyo Metropolitan
Tama Medical Center

Tokyo Metropolitan
Children's Medical Center

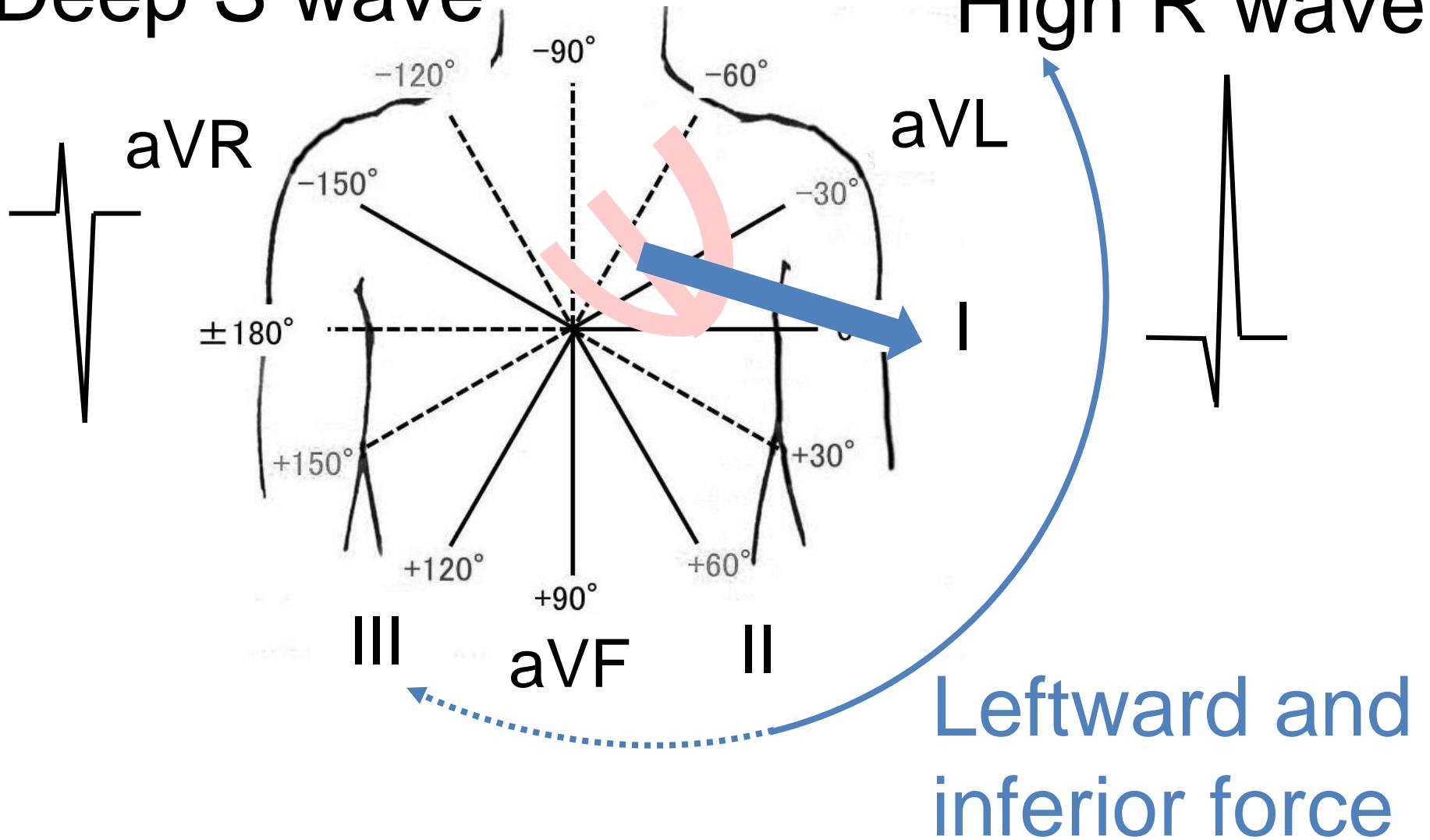
Significance of ECG for ACHD

- Non-invasive and cost effective
- Provides clues for first diagnosis in adulthood
- Evaluates cardiac load
 - ↓
Left ventricular hypertrophy (LVH)
- Helps manage arrhythmia



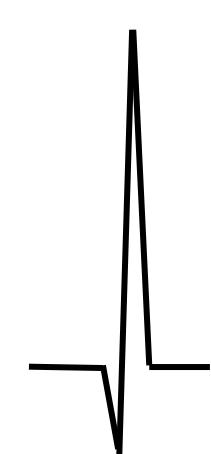
Limb leads in LVH

Deep S wave



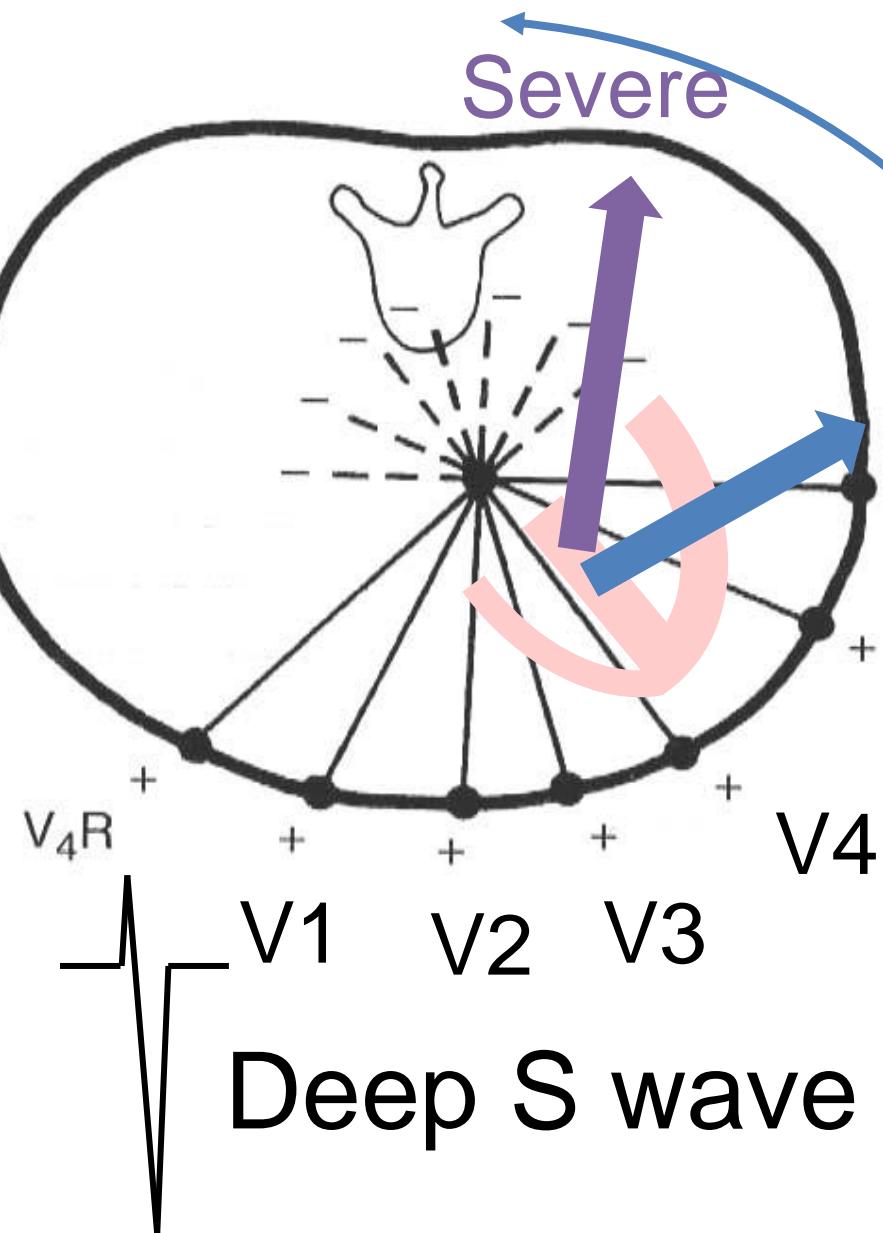
High R wave

aVL

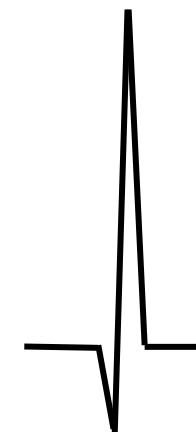


Leftward and
inferior force

Chest leads in LVH



High R wave



Deep Q wave

Leftward and
posterior force

Sokolow-Lyon voltage criteria

$SV1 + RV5 \text{ (or V6)} > 3.5 \text{ mV}$

$RaVL > 1.1 \text{ mV}$

Cornell voltage criteria

$RaVL + SV3 > 2.8 \text{ mV} \text{ (men)}$

$2.0 \text{ mV} \text{ (women)}$

Cornell product

Romit-Estes score

Several other criteria

ECG criteria
for LVH

Factors influencing voltage

Age —— applies to > 35 years old

Gender

Race —————

Body habitus

Cornell voltage criteria
for Koreans

2.0 mV (men)

1.6 mV (women)

Jin Kyu Park, et al.

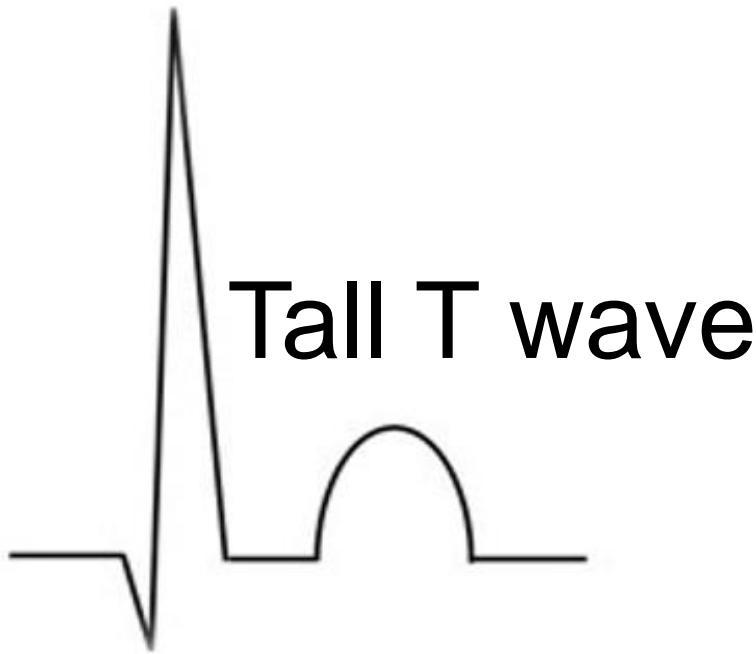
Korean Circ J 2012;42:606

+ Congenital heart diseases

LVH patterns in left-sided leads

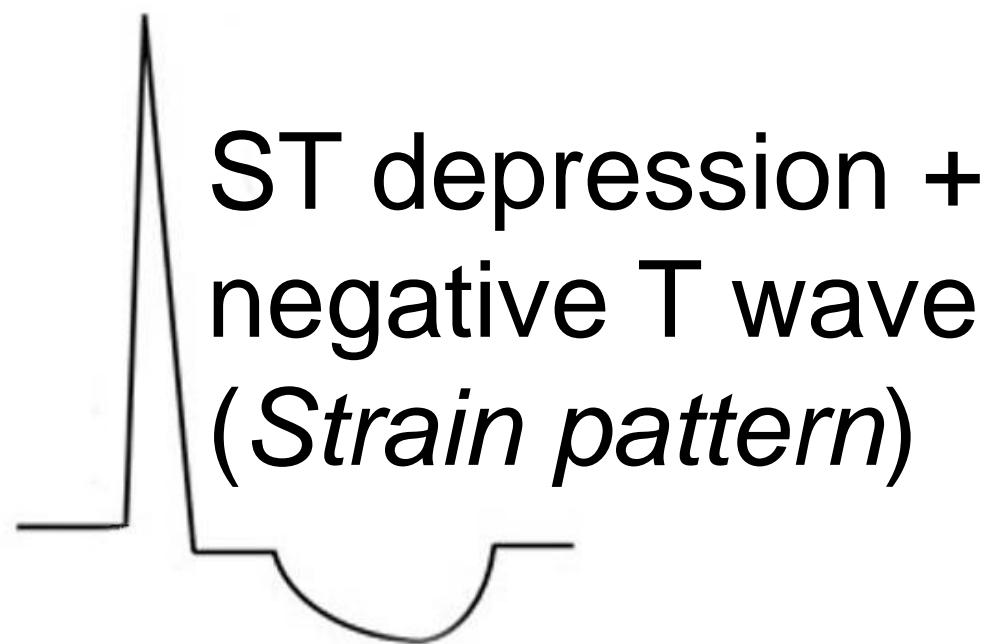
Volume overload

High R wave



Pressure overload

High R wave



Deep Q wave

Shallow Q wave

LVH mechanisms in ACHD

Volume overload

Left-to-right shunt

Right-to-left shunt at atrial level

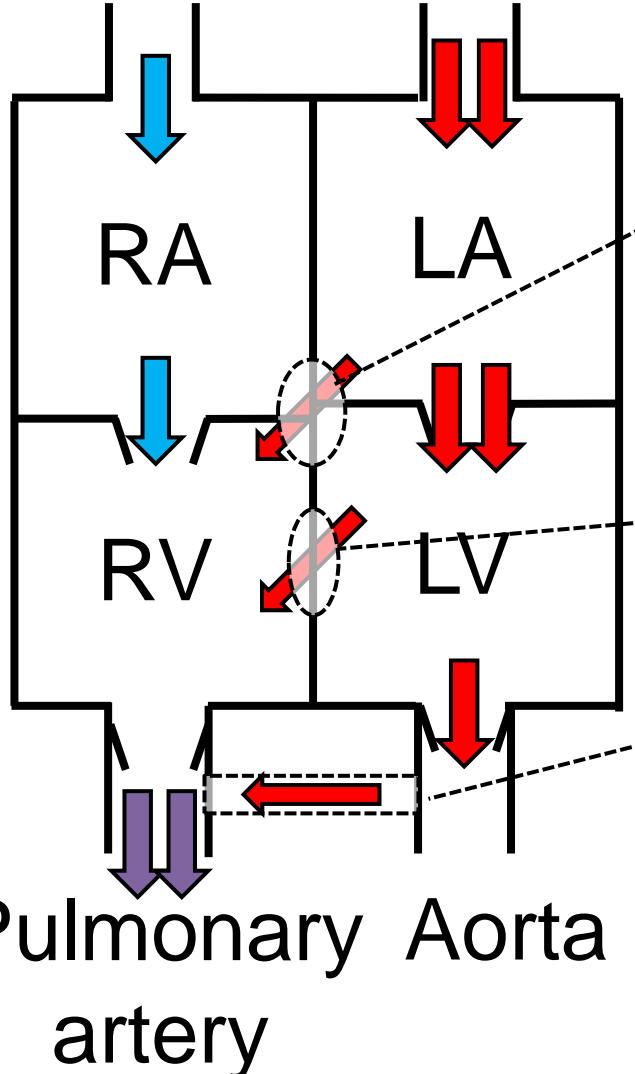
Valve regurgitation

Pressure overload

Valve or aortic stenosis

Hypertension

Vena cava
Pulmonary vein



Left-to-right shunt

Atrioventricular septal defect (AVSD)

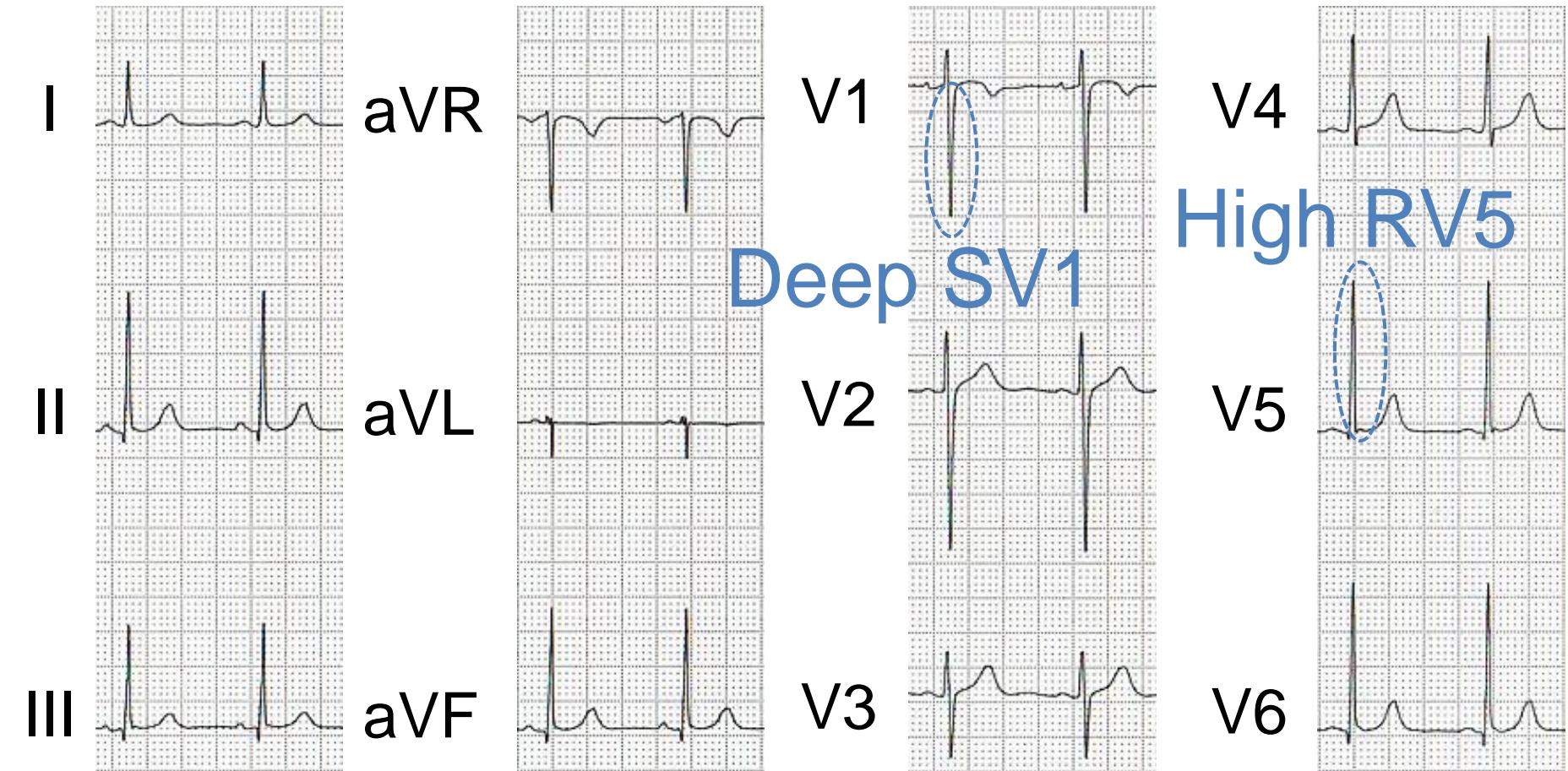
Ventricular septal defect (VSD)

Patent ductus arteriosus (PDA)

Blalock–Taussig shunt

Volume overload

18 y/o F Patent ductus arteriosus



$$SV1 + RV5 = 4.0 \text{ mV} \rightarrow LVH$$

First diagnosed at 18 y/o

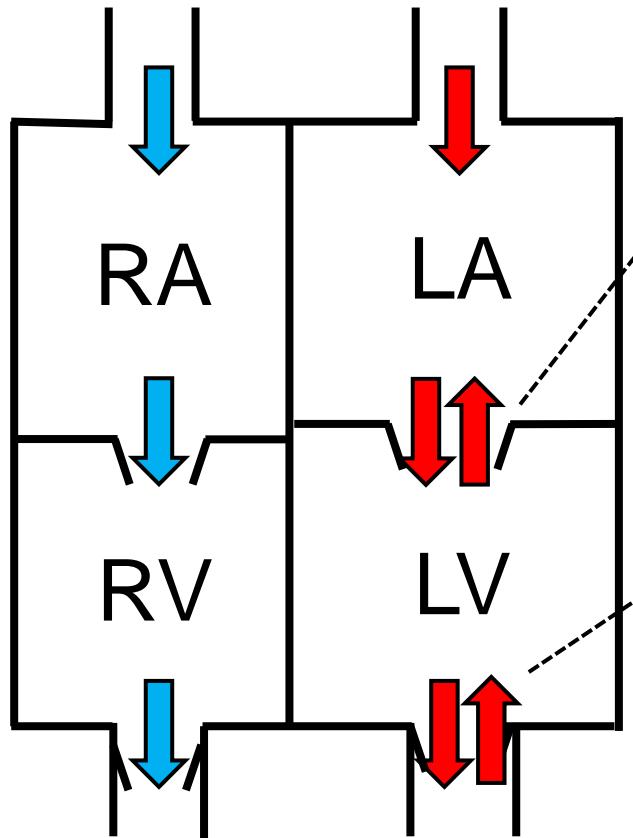
Mitral valve
regurgitation

AVSD

Aortic valve
regurgitation

Outlet VSD & coronary
cusp prolapse

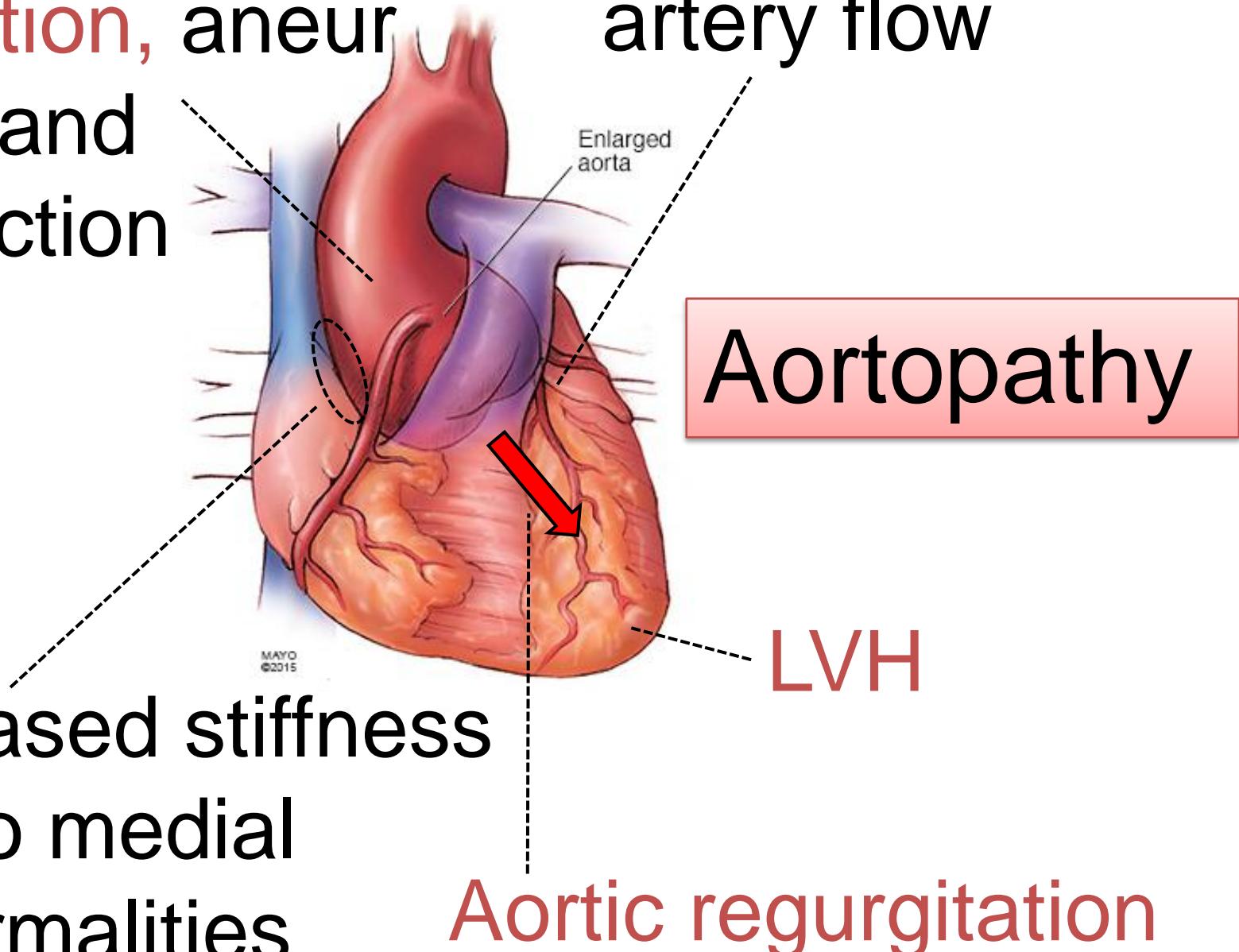
Aortopathy



Volume overload

Aortic dilatation, aneurysm, and dissection

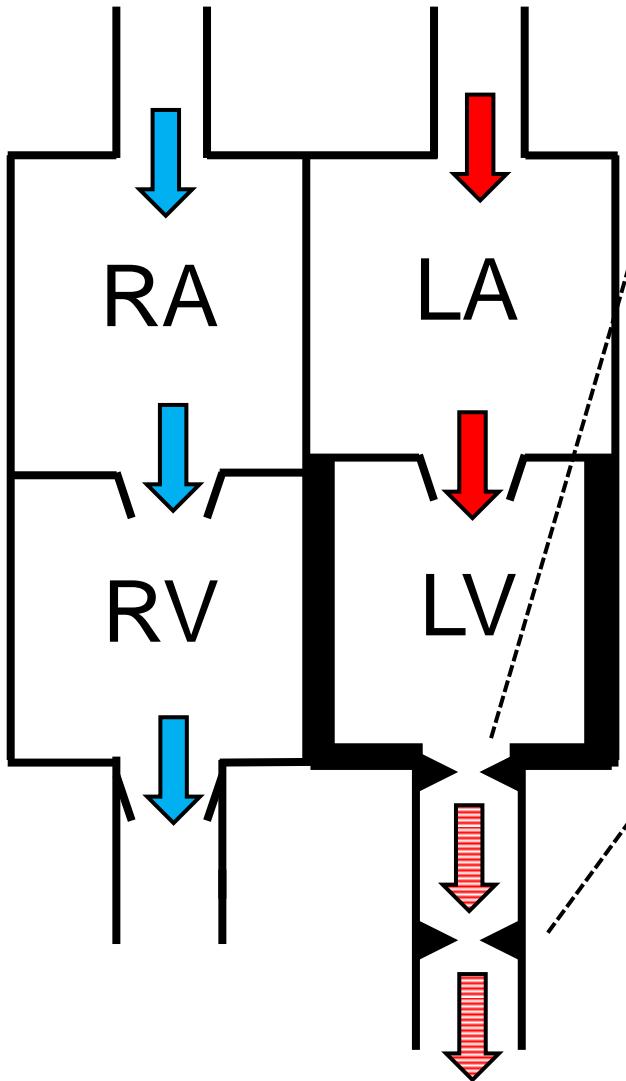
Reduced coronary artery flow



Increased stiffness due to medial abnormalities

Diseases causing aortopathy

- Marfan syndrome
- Turner syndrome
- Bicuspid aortic valve (BAV)
- Tetralogy of Fallot (TOF)
- Single ventricle
- Coarctation of the aorta (COA)
- Persistent truncus arteriosus
- Transposition of the great arteries (TGA)
- Hypoplastic left heart syndrome (HLHS)

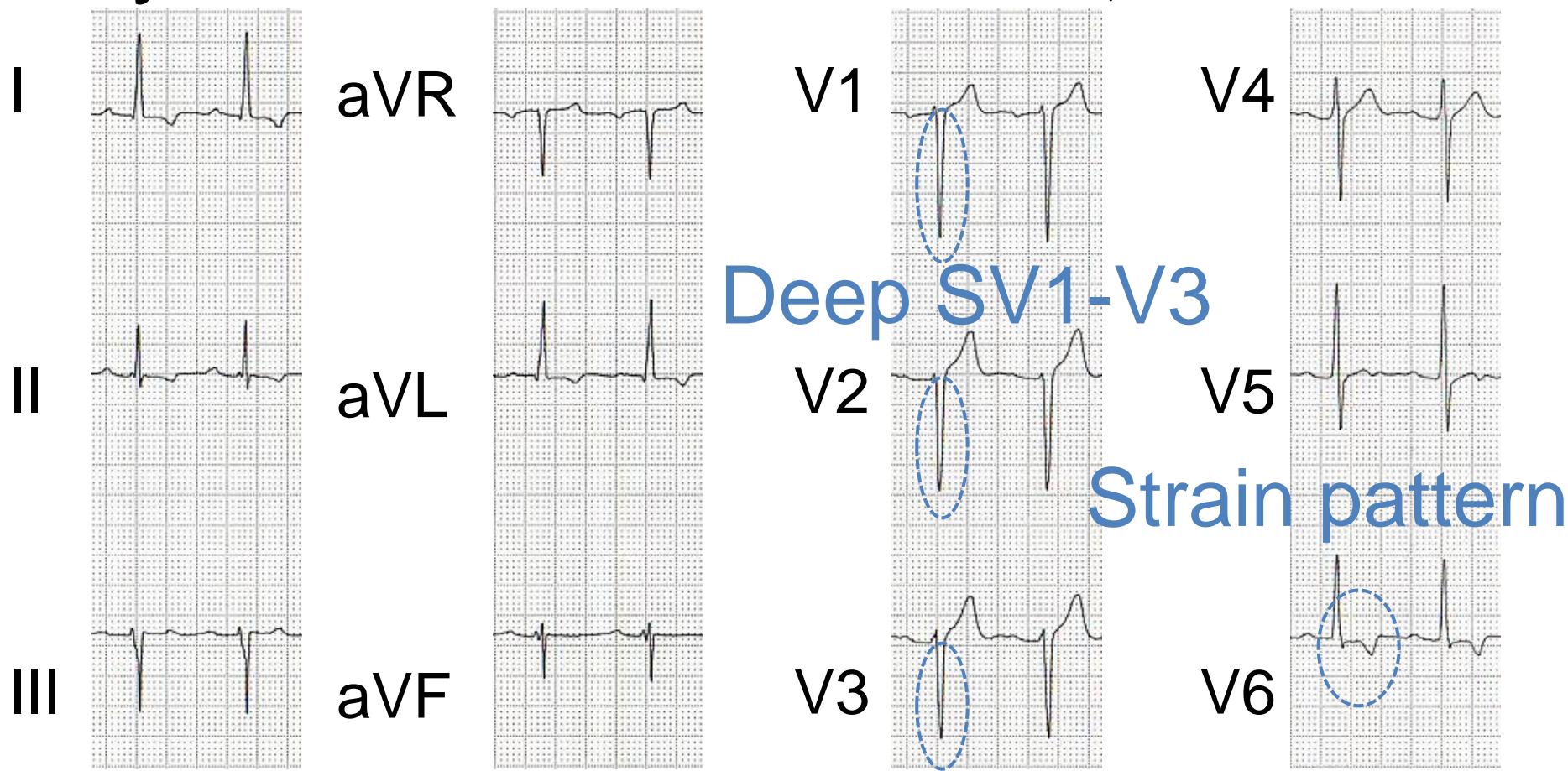


Aortic valve stenosis
Valvular
Supra-valvular
Sub-valvular

Aortic stenosis
Coarctation of the aorta
Interrupted aortic arch

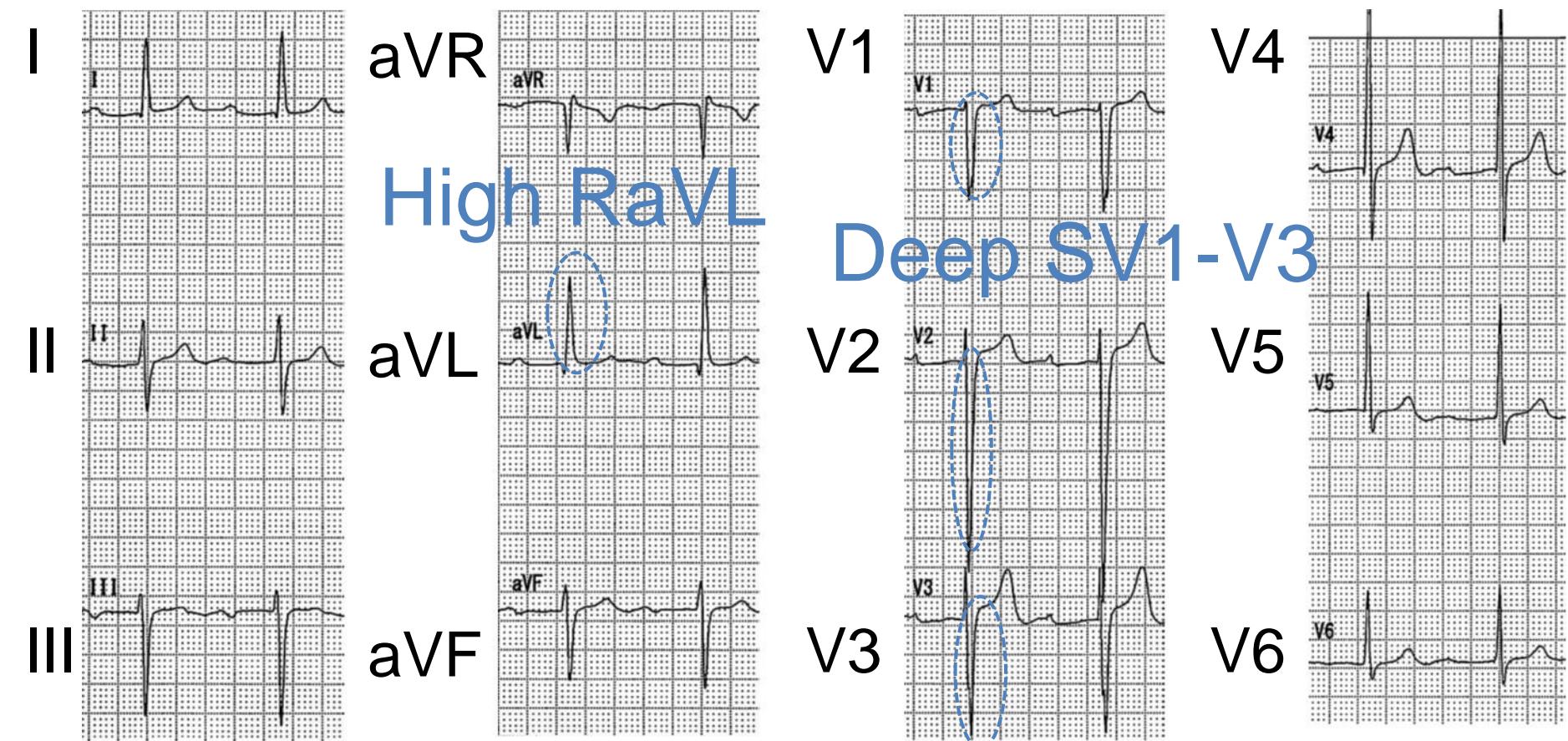
Pressure overload

42 y/o M Aortic stenosis, BAV



ST-T changes in I, aVL, and V6 → LVH
Health exam abnormality and dyspnea

27y/o M Interrupted aortic arch p.o.

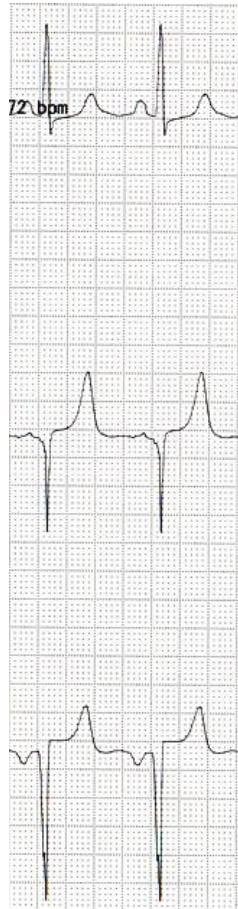


$RaVL + SV3 = 3.6 \text{ mV} \rightarrow LVH$

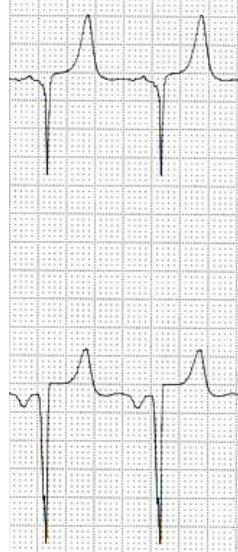
Hypertension without re-stenosis of aortic arch

20 y/o F HOCM, Noonan synd.

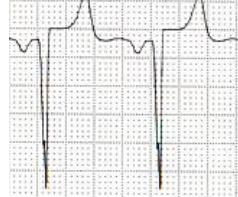
I



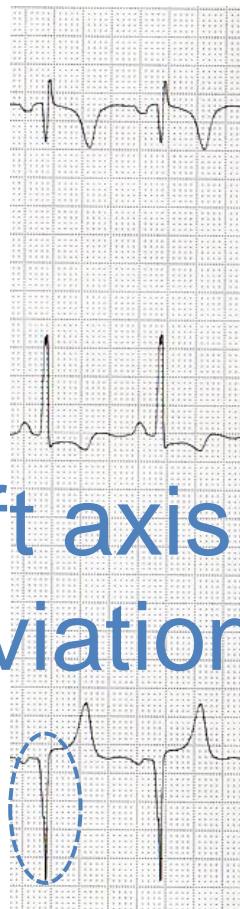
II



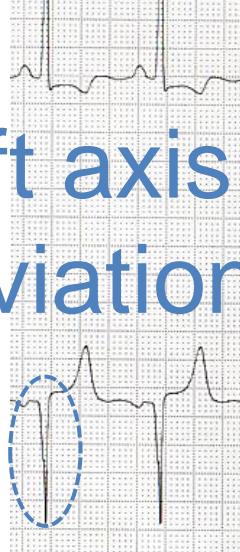
III



aVR

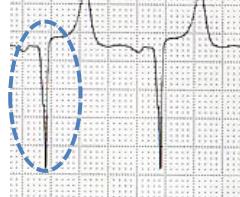


aVL

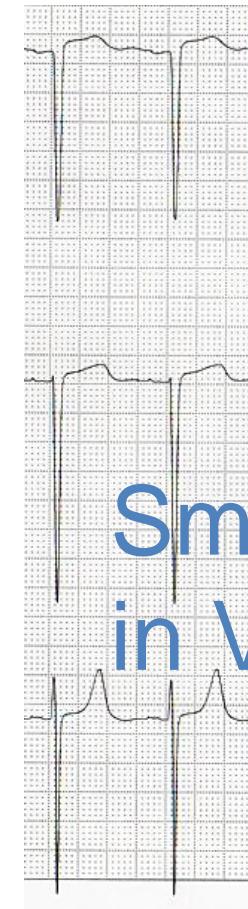


Left axis deviation

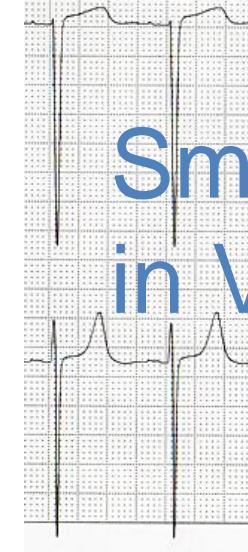
aVF



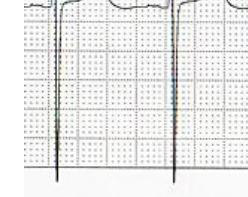
V1



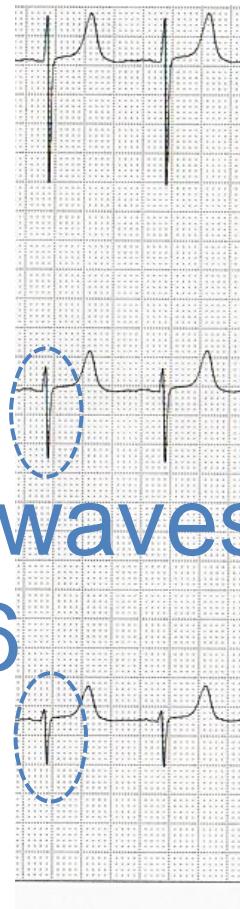
V2



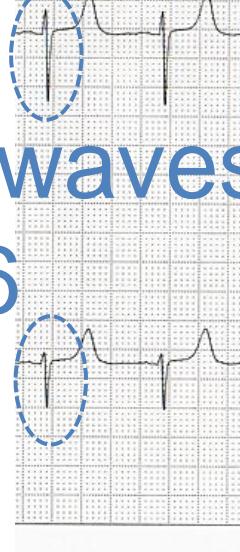
V3



V4

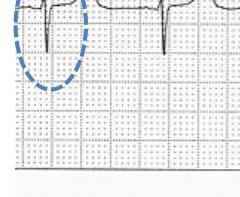


V5



Small R waves
in V5, V6

V6



5 mm/mV

R_{aVL} + S_{V3} = 7.7 mV → LVH?

Progressive obstruction of LV outflow

ACHD with left axis deviation

AVSD

Tricuspid atresia

Noonan syndrome

Often

Perimembranous VSD

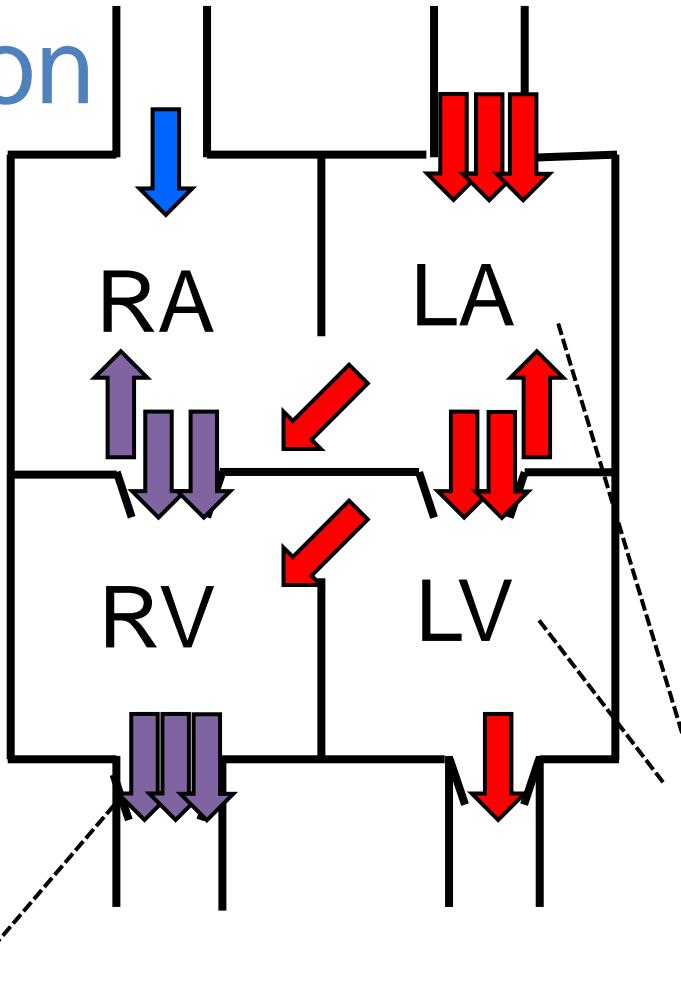
Corrected TGA

Single ventricle

Sometimes

Complete AVSD

Left axis deviation

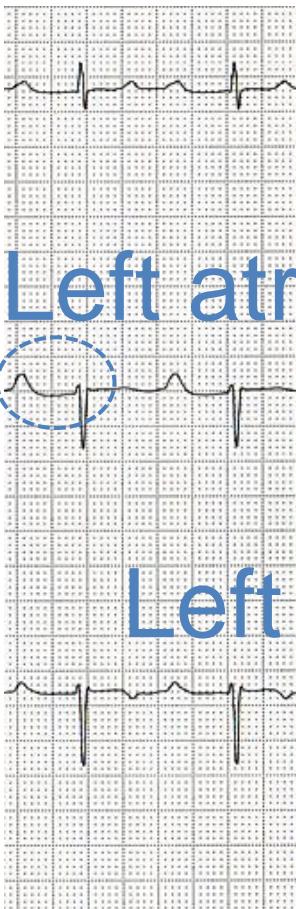


Left-to-right shunt
+
Atrioventricular
valve regurgitation

Volume overload

Increased pulmonary blood flow

36 y/o F Complete AVSD



aVR

Left atrial enlargement

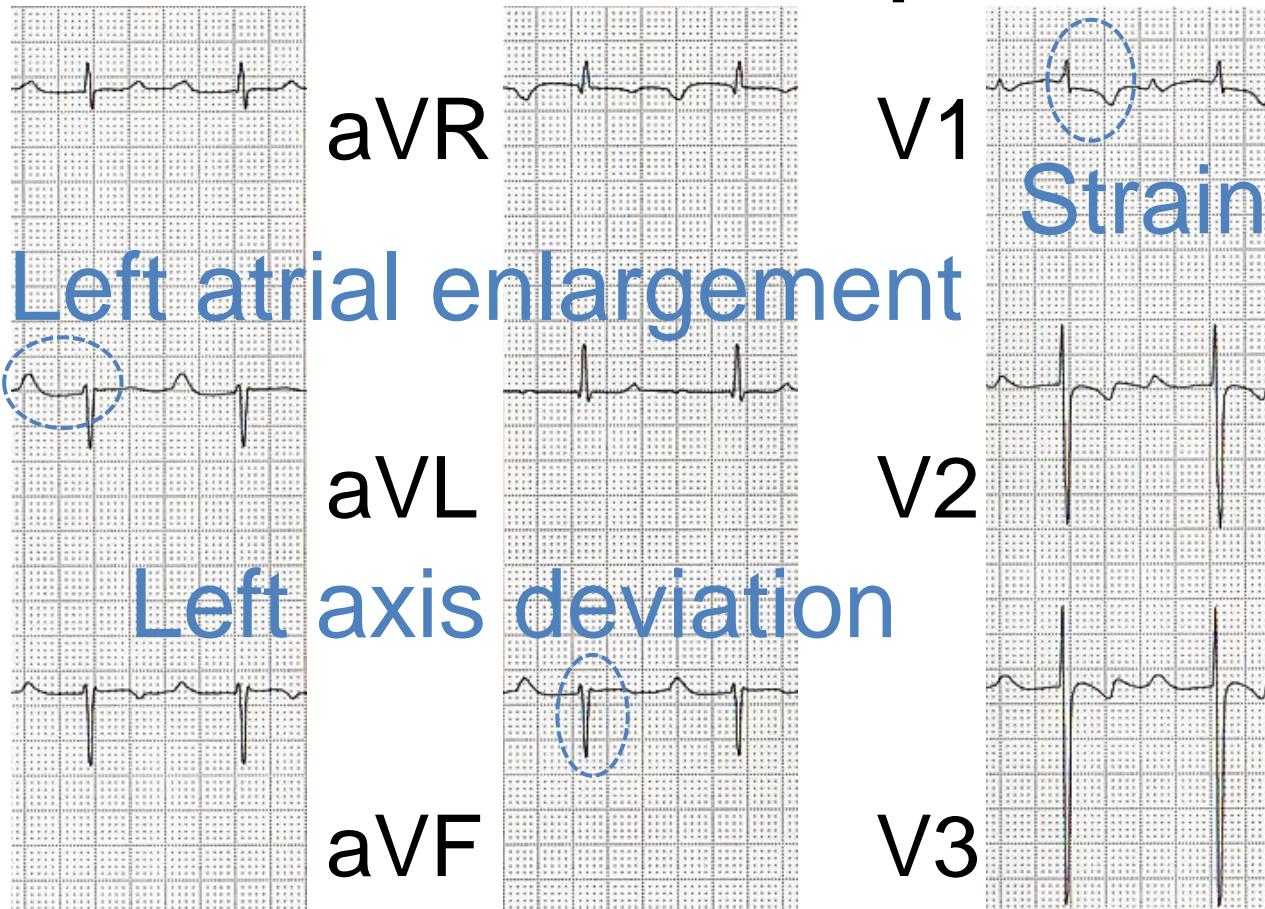


aVL

Left axis deviation



aVF



V1

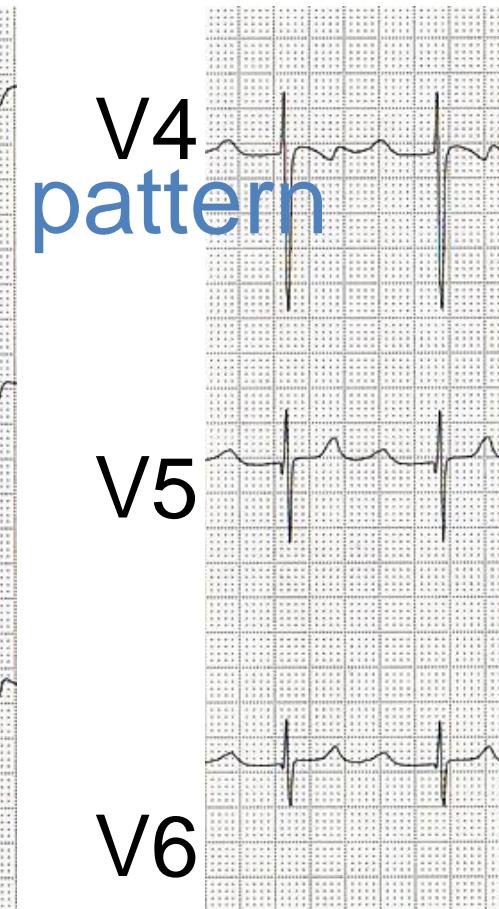
Strain pattern

V2

V4

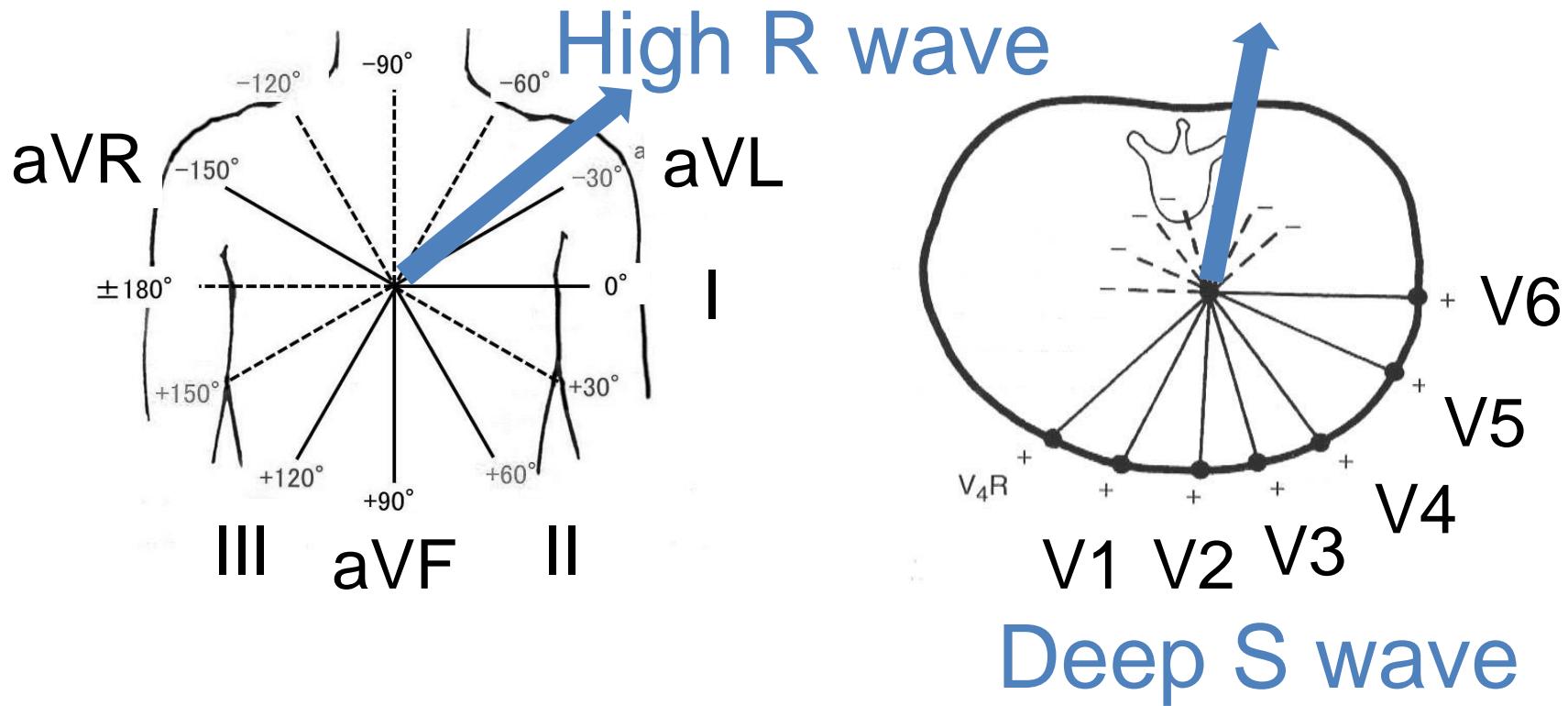
V5

V6



$$RaVL + SV3 = 3.7 \text{ mV} \rightarrow \text{LVH?}$$

Down syndrome, Mitral valve regurgitation,



In ACHD with left axis deviation, RaVL is higher. If associated with clockwise rotation, SV1 to SV3 are deeper.

Voltage criteria may **NOT** be applied.

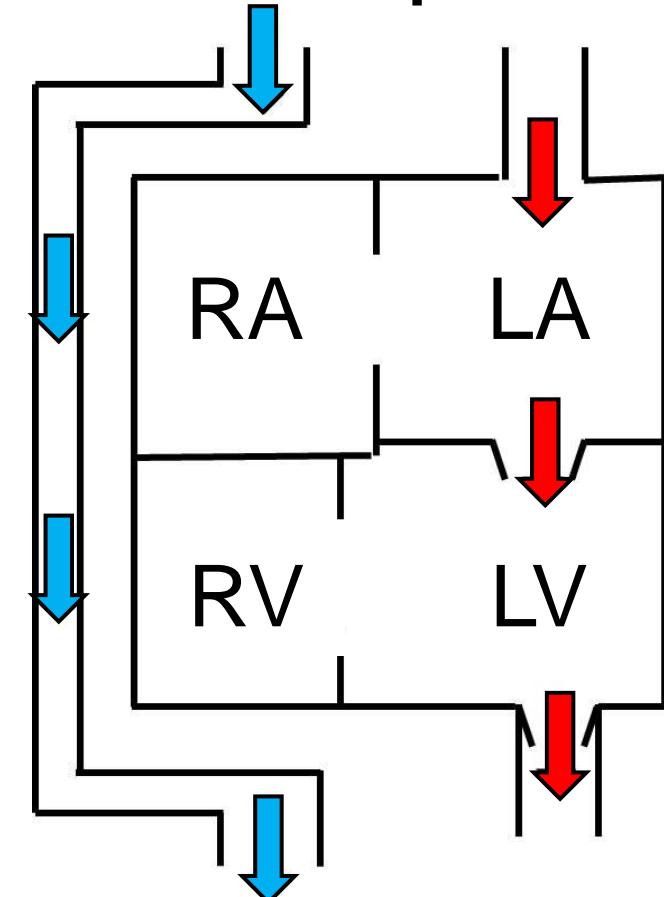
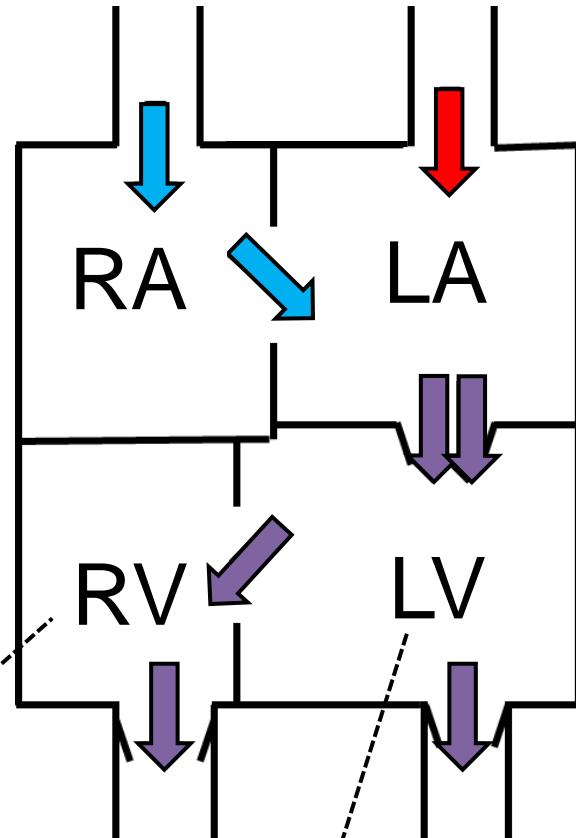
Tricuspid atresia

Fontan operation

Left axis deviation

Hypoplasia

Volume overload



20 y/o F Tricuspid atresia p.o.



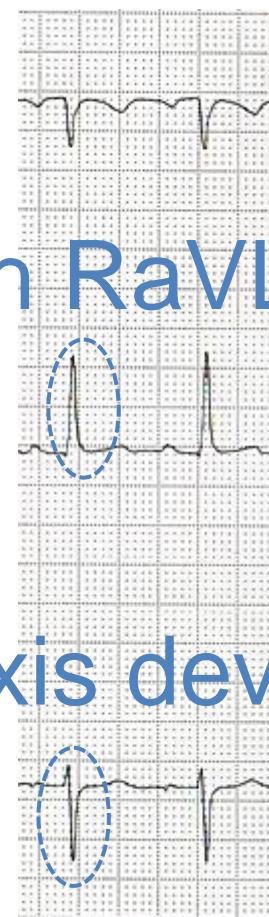
I
aVR

High RaVL

II
aVL

Left axis deviation

III
aVF

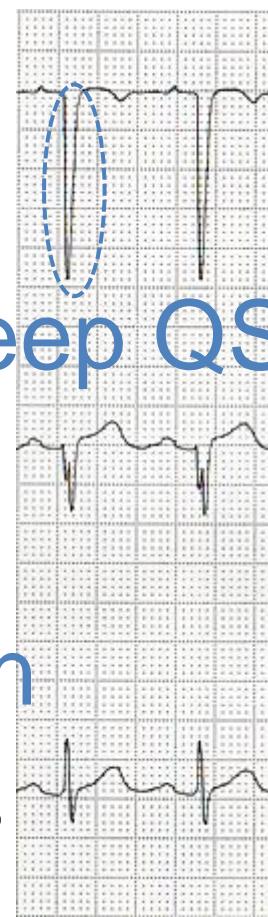


V1

Deep QSV1

V2

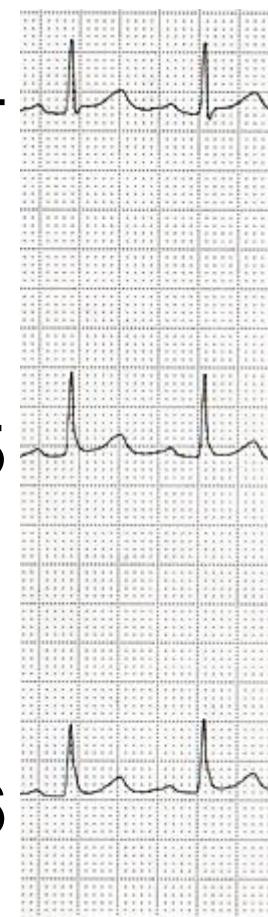
V3



V4

V5

V6



$RV5 + SV1 = 3.5 \text{ mV} \rightarrow \text{LVH?}$

Good course after Fontan operation

Take home messages

- 1) LVH is induced by several ACHD including aortopathy.
- 2) ECG is useful for evaluating cardiac load on LV in ACHD.
- 3) Criteria for LVH and the clinical impact for ACHD should be investigated.