Diplôme d'Université de Cardiologie Congénitale et Pédiatrique 2020-21

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CRMR Maladies Cardiaques Héréditaires et Rares
Centre de Compétences du CRMR Hypertensions pulmonaires
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Diplôme d'Université de Cardiologie Congénitale et Pédiatrique

- 3 semaines d'enseignement en « distanciel » ou en présentiel en fonction de l'évolution des conditions sanitaires
- Examen écrit et oral en juin 2022
  - modalités à définir en fonction de l'évolution de la pandémie
    - Ecrit: Questions courtes/Problème de physiologie/Question de cours
    - Oral: Cas cliniques
  - note minimale requise 12/20 aux deux épreuves écrites et orales
- Principales sources d'information: WEB

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Université de Paris

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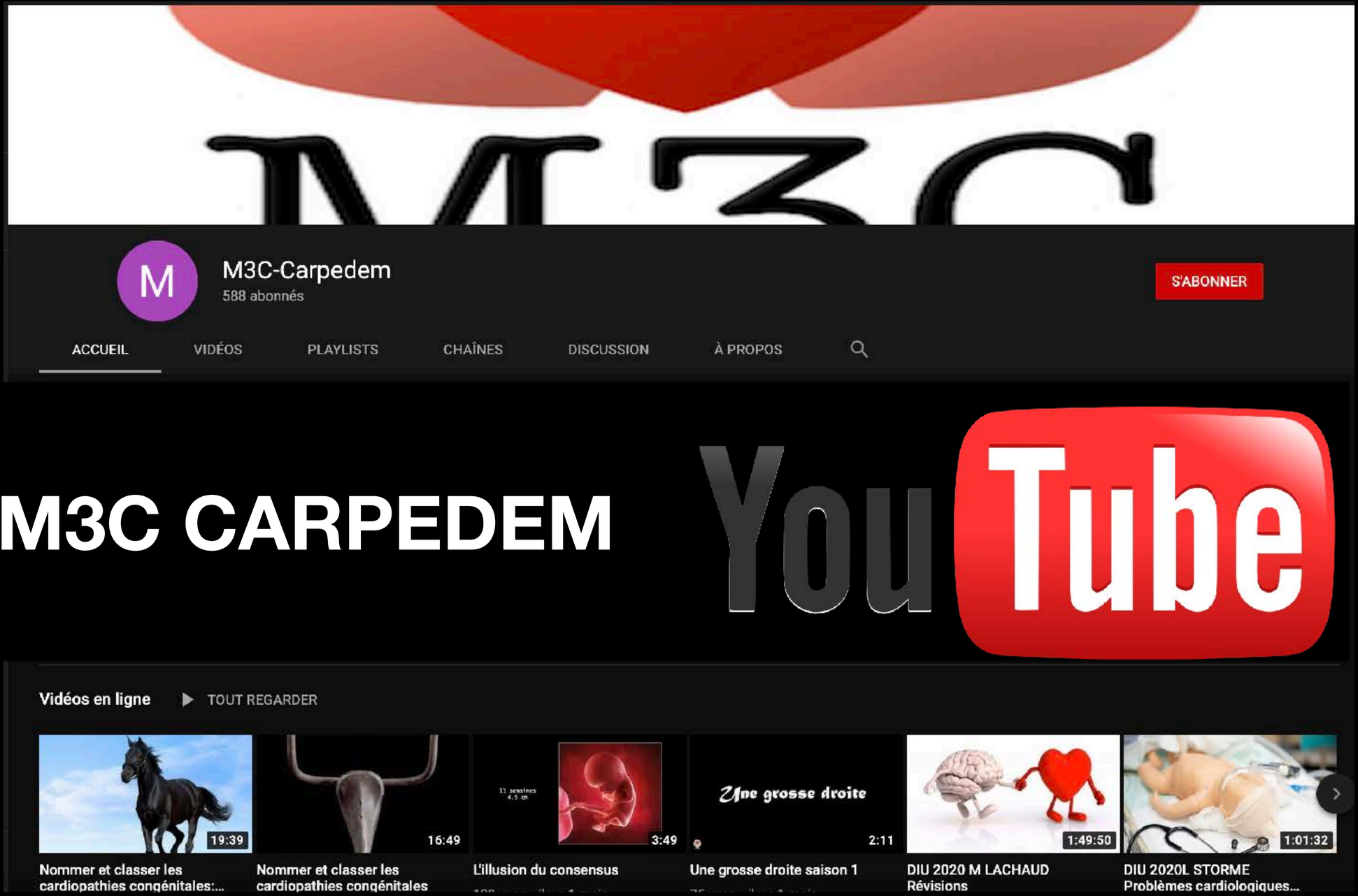

M3C CARPEDEM

Physiologie cardiaque appliquée au développement cardiaque

Damien Bonnet

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Faculté de Médecine Paris Descartes
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Centre de Référence Maladies Rares
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European Reference Network
for rare or low prevalence complex diseases
Network Respiratory Diseases (ERN-LUNG)

European Reference Network
for rare or low prevalence complex diseases
Network Heart Diseases (ERN GUARD-HEART)

Physiologie cardiaque appliquée aux cardiopathies congénitales

Partie 1

Physiologie du coeur foetal et conséquences sur la morphogenèse cardiaque

Professeur Damien Bonnet
Service de Cardiologie Congénitale et Pédiatrique
Hôpital Universitaire Necker Enfants malades – APHP, Université de Paris
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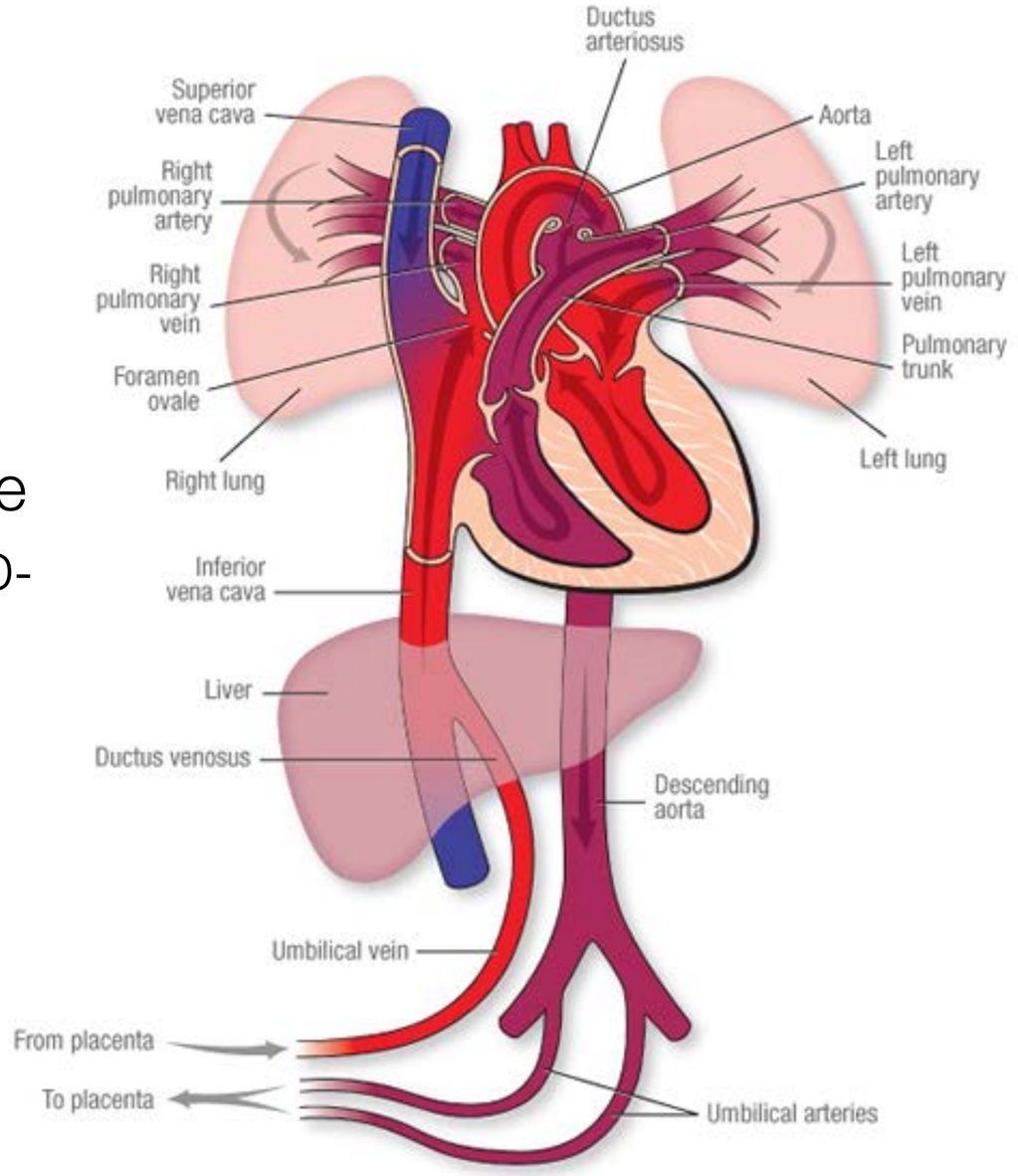
Maladies Cardiaques Héréditaires- CARDIOGEN

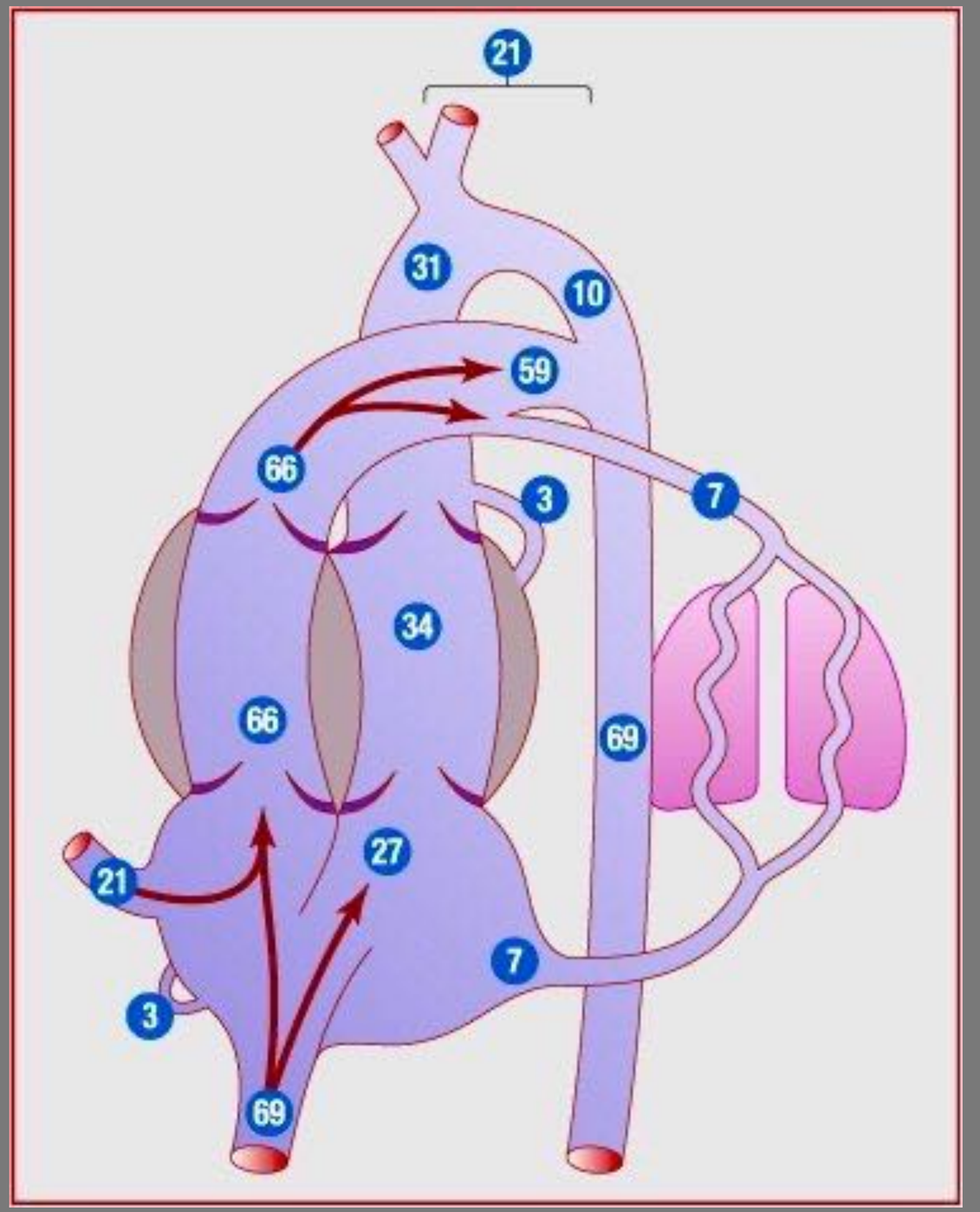
Anatomie du cœur fœtal et Conséquences hémodynamiques

- Les shunts et la circulation en parallèle
  - Le placenta et le ductus venosus ou canal d'Arantius
  - Le court-circuit de la circulation pulmonaire par le Canal Artériel
  - Le Foramen Ovale (CIA) qui permet d 'alimenter le Cœur Gauche

- Pour l'oxygénation la circulation est presque en série
  - PI->VO->PFO->OG->VG->AoA->VCS->OD->VD->AP->AoD->AO->PI
- Le sang oxygéné va en priorité au cœur gauche
  - cœur et cerveau



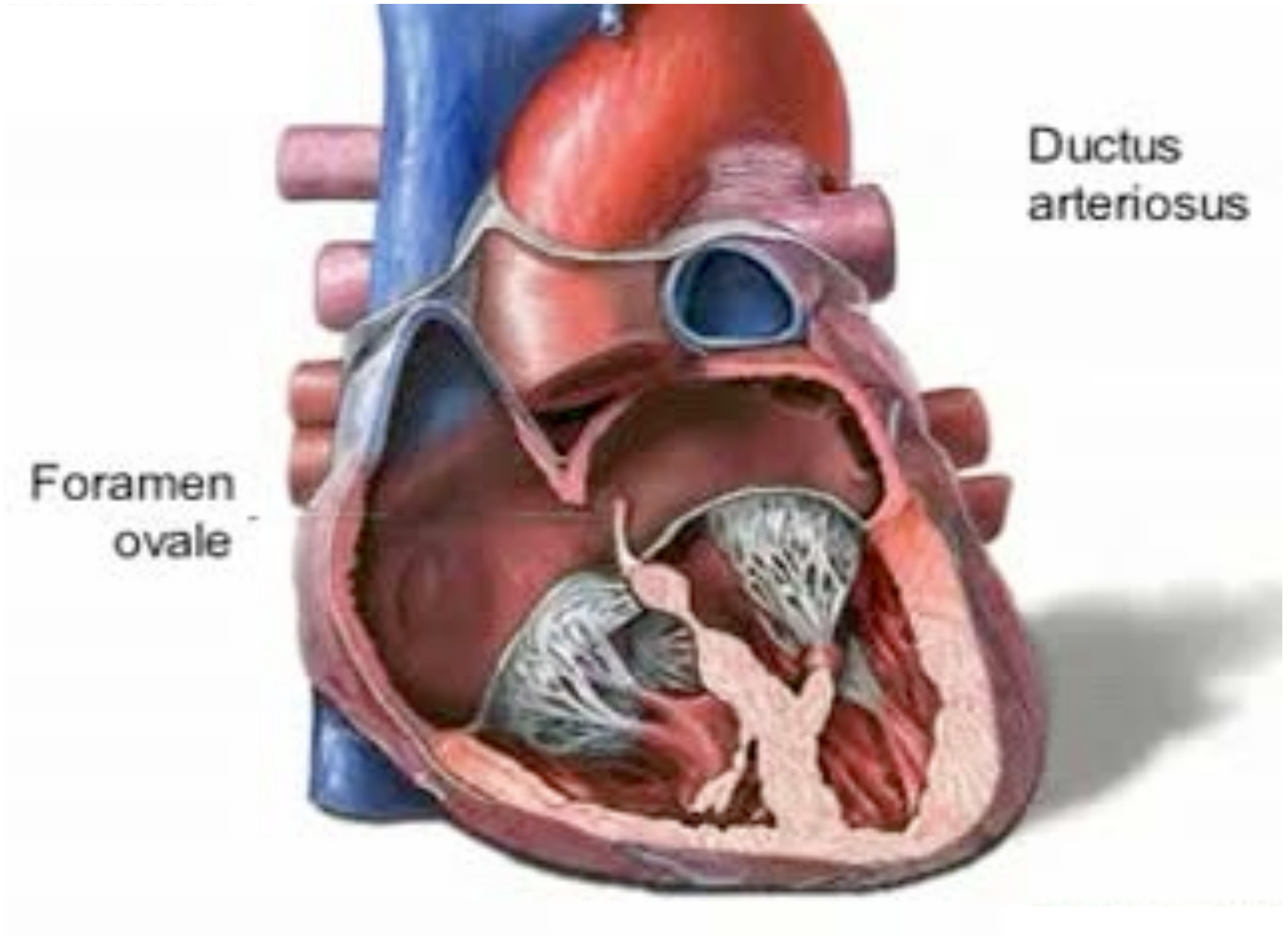
Débit sanguin foetal combiné



Conséquence sur les volumes des Ventricules

Les 2 ventricules se remplissent à la même pression (pré-charge)

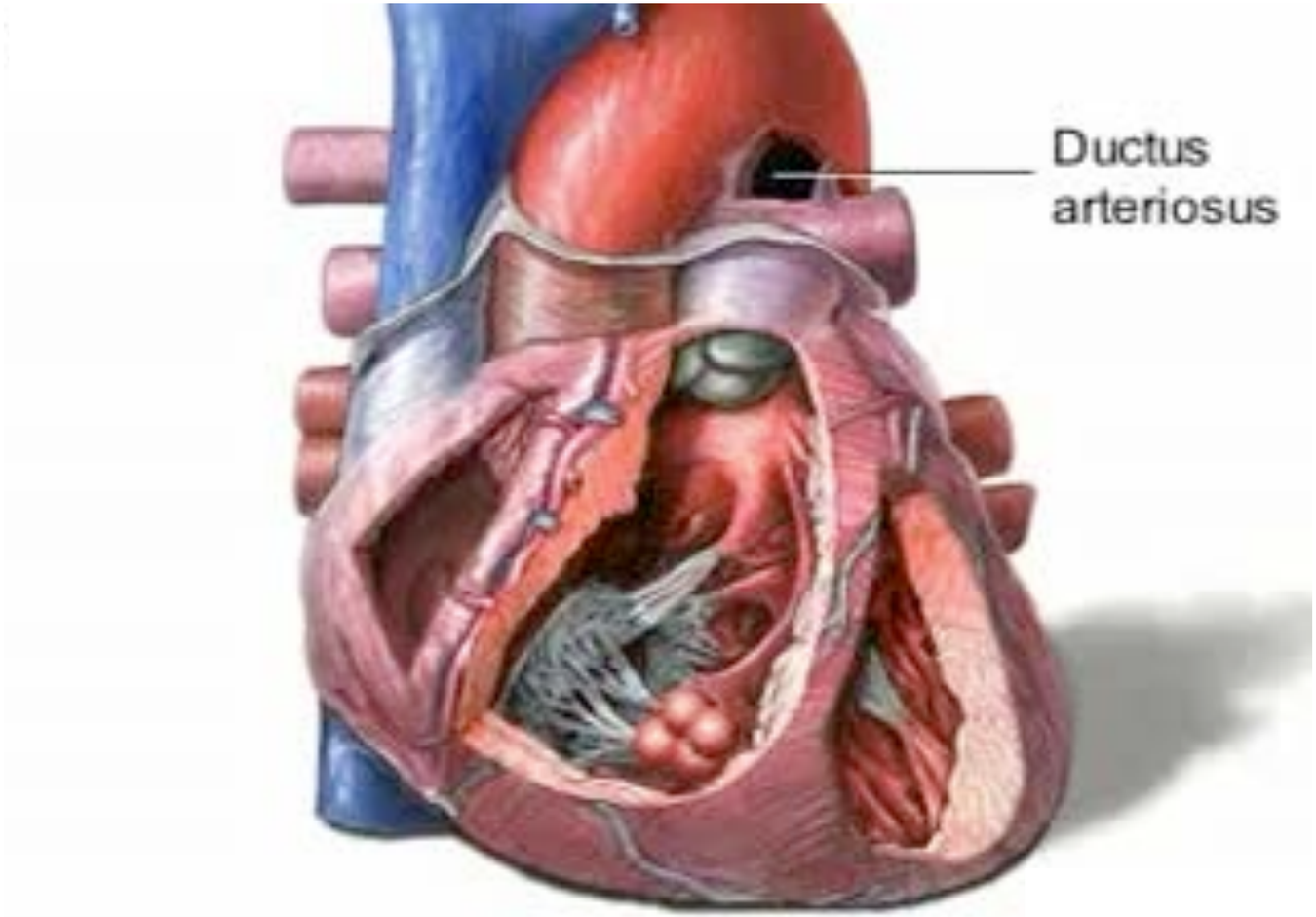
Les volumes télé-diastoliques des ventricules & les compliances respectives



Conséquence sur les volumes des Ventricules

Les 2 Ventricules se vident à la même pression (post-charge)

Les volumes télé-systoliques des Ventricules & les contractilités respectives



Conséquences sur les volumes des ventricules

- Les volumes des ventricules sont définis par
  - Les conditions de charge du cœur
  - Les propriétés du cœur

- Courbe pression/volume
  - Le Volume télé-systolique = contractilité/post-charge
  - Le Volume télé-diastolique= compliance/pré-charge

Circulation Fœtale

Conséquence sur les volumes des Ventricules

Les 2 ventricules se remplissent à la même pression (pré-charge)

Les volumes télé-diastoliques des Ventricules & les compliances respectives

Les 2 Ventricules se vident à la même pression (post-charge)

Les volumes télé-systoliques des Ventricules & les contractilités respectives

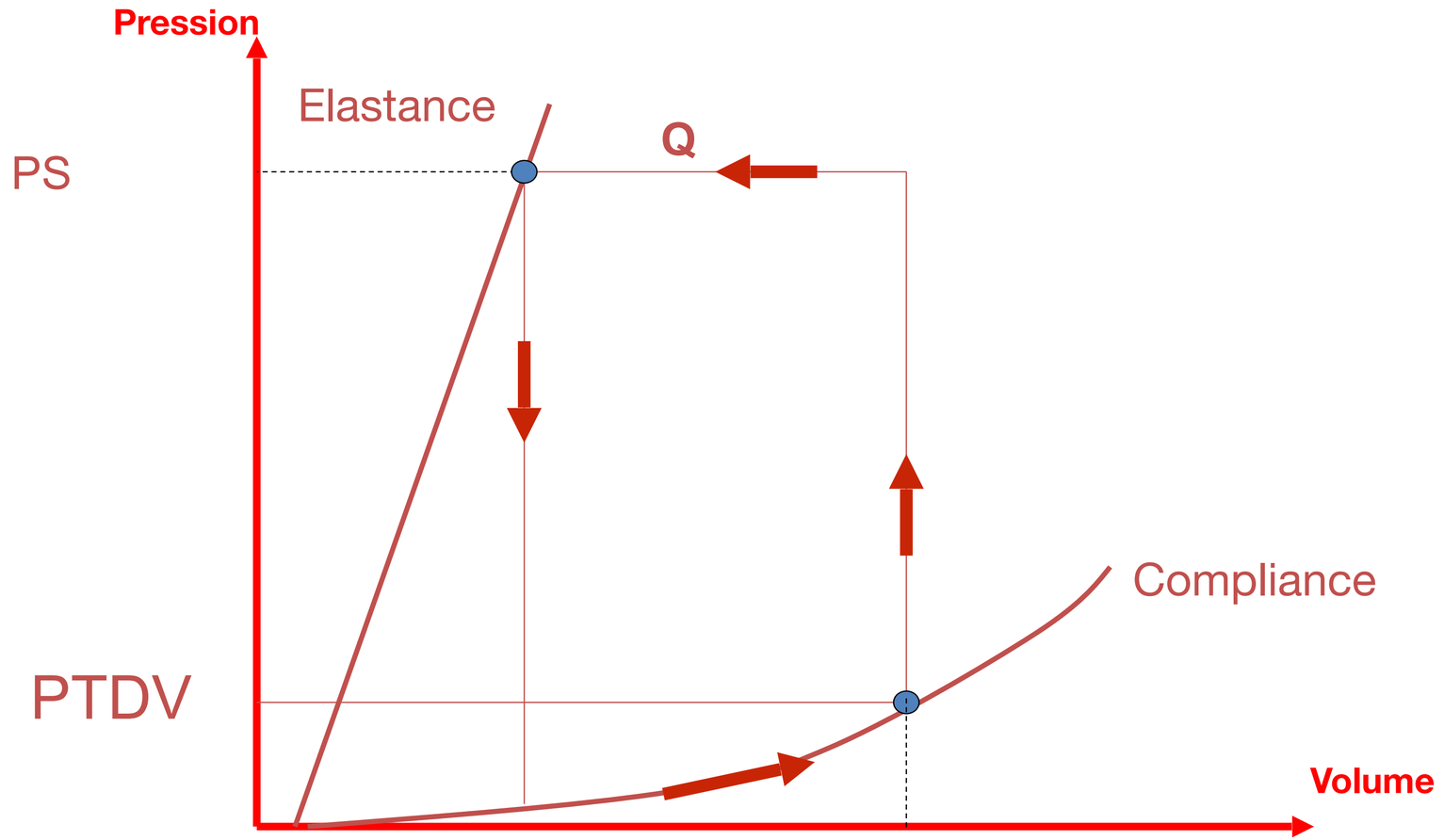
Volume d’éjection systolique (VES)= VTD-VTS

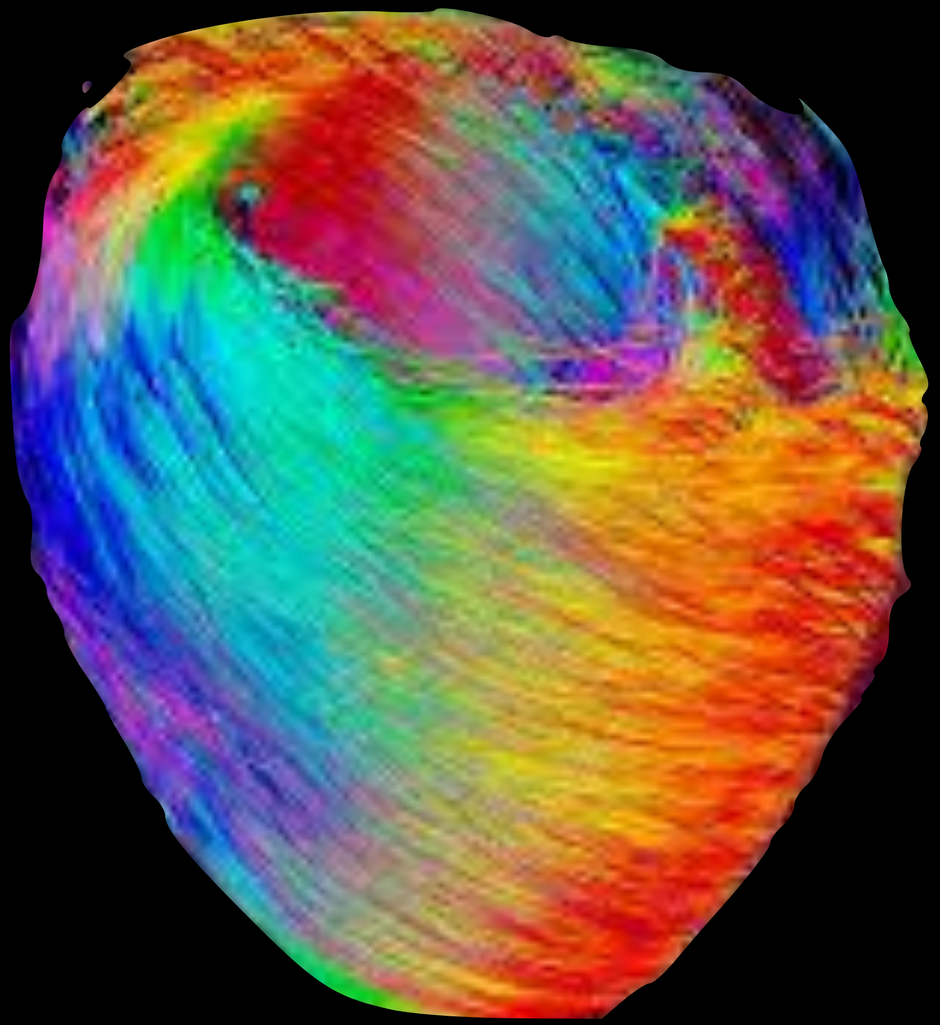
Les volumes éjectés sont fonction des propriétés myocardiques

Conséquences sur la croissance de ventricules et des vaisseaux

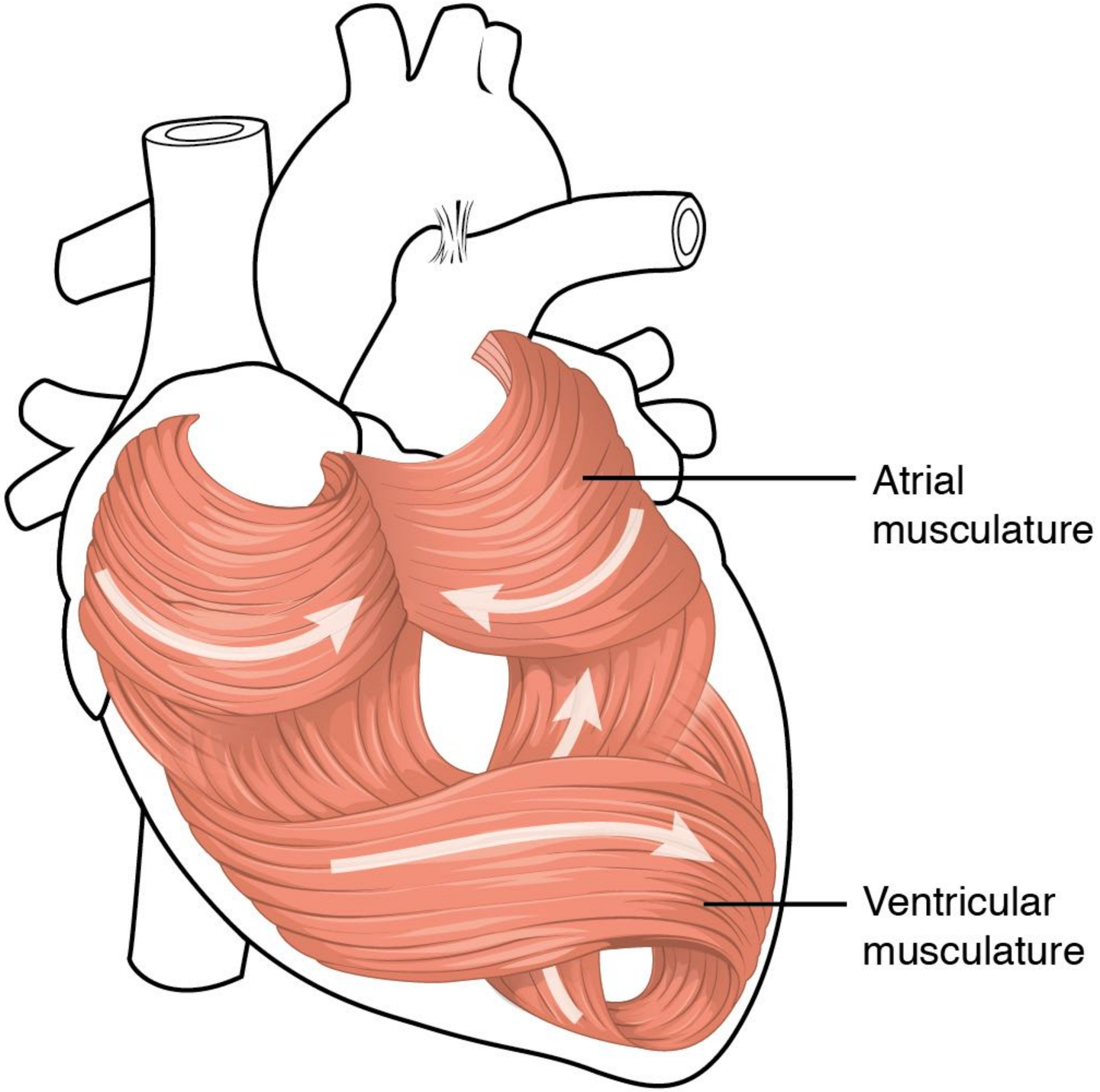
Les volumes éjectés sont fonction des propriétés myocardiques

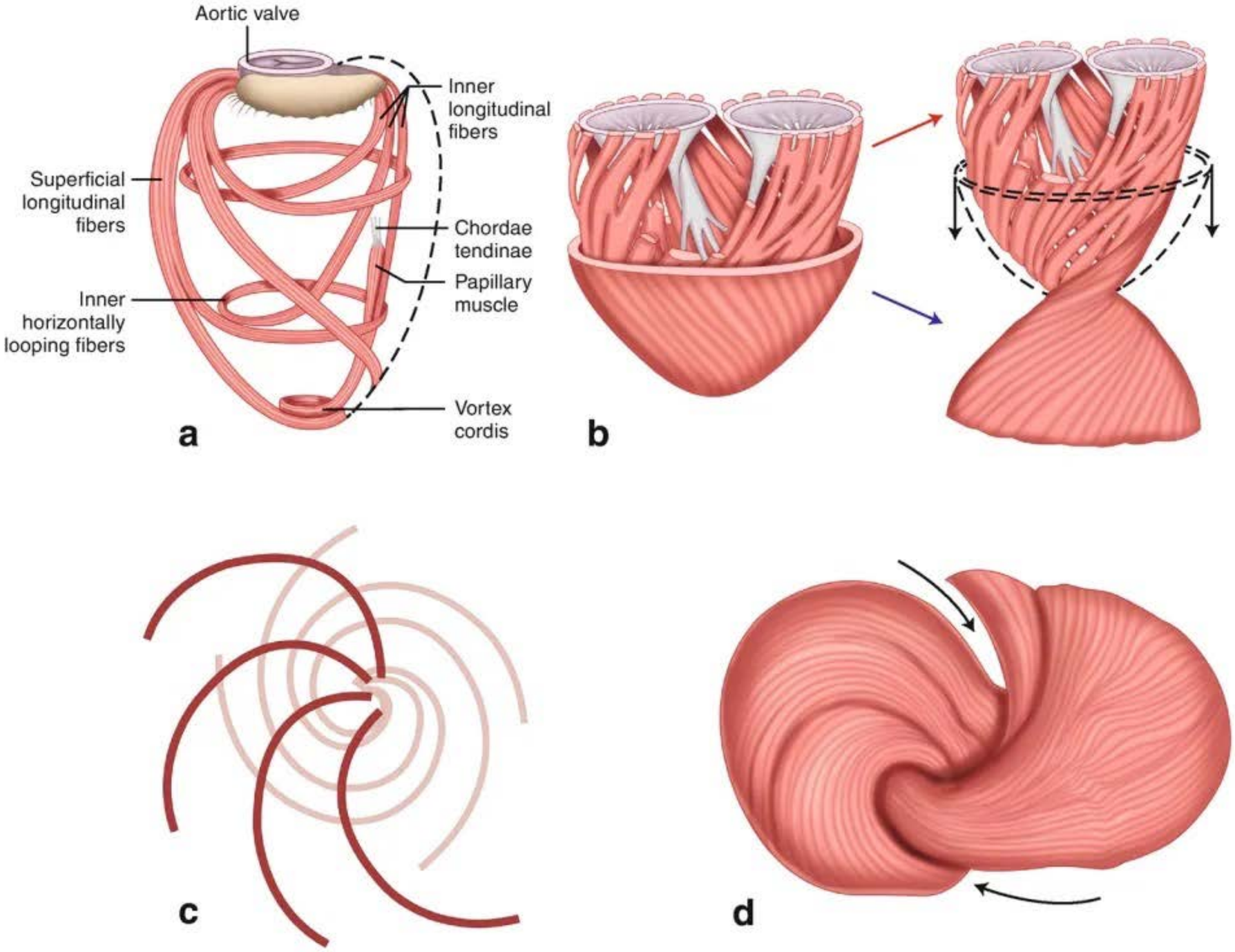
Conséquences sur la croissance de ventricules et des vaisseaux

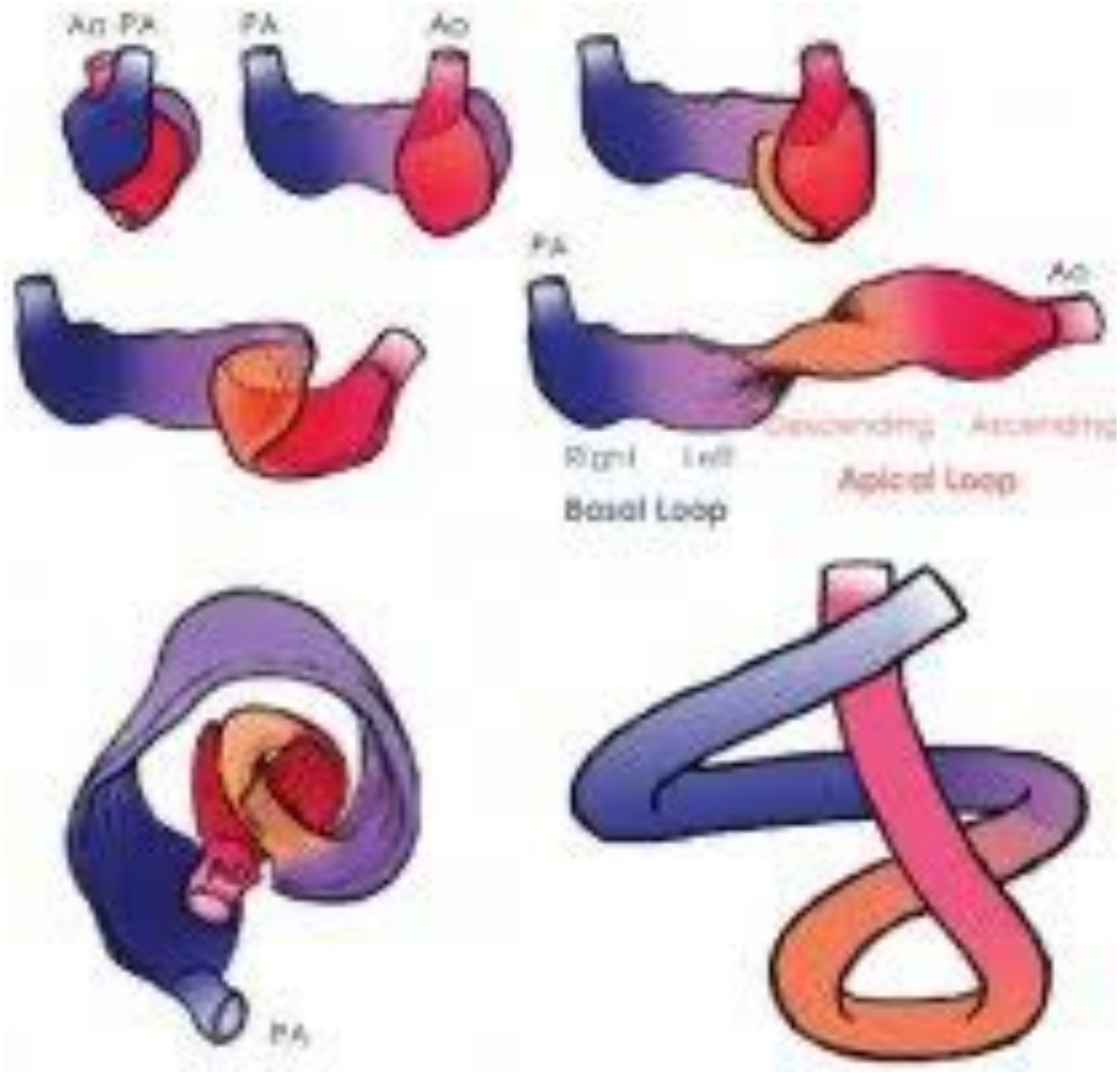


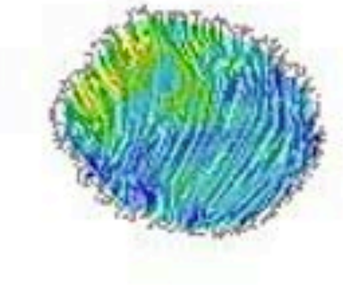


Propriétés intrinsèques du myocarde

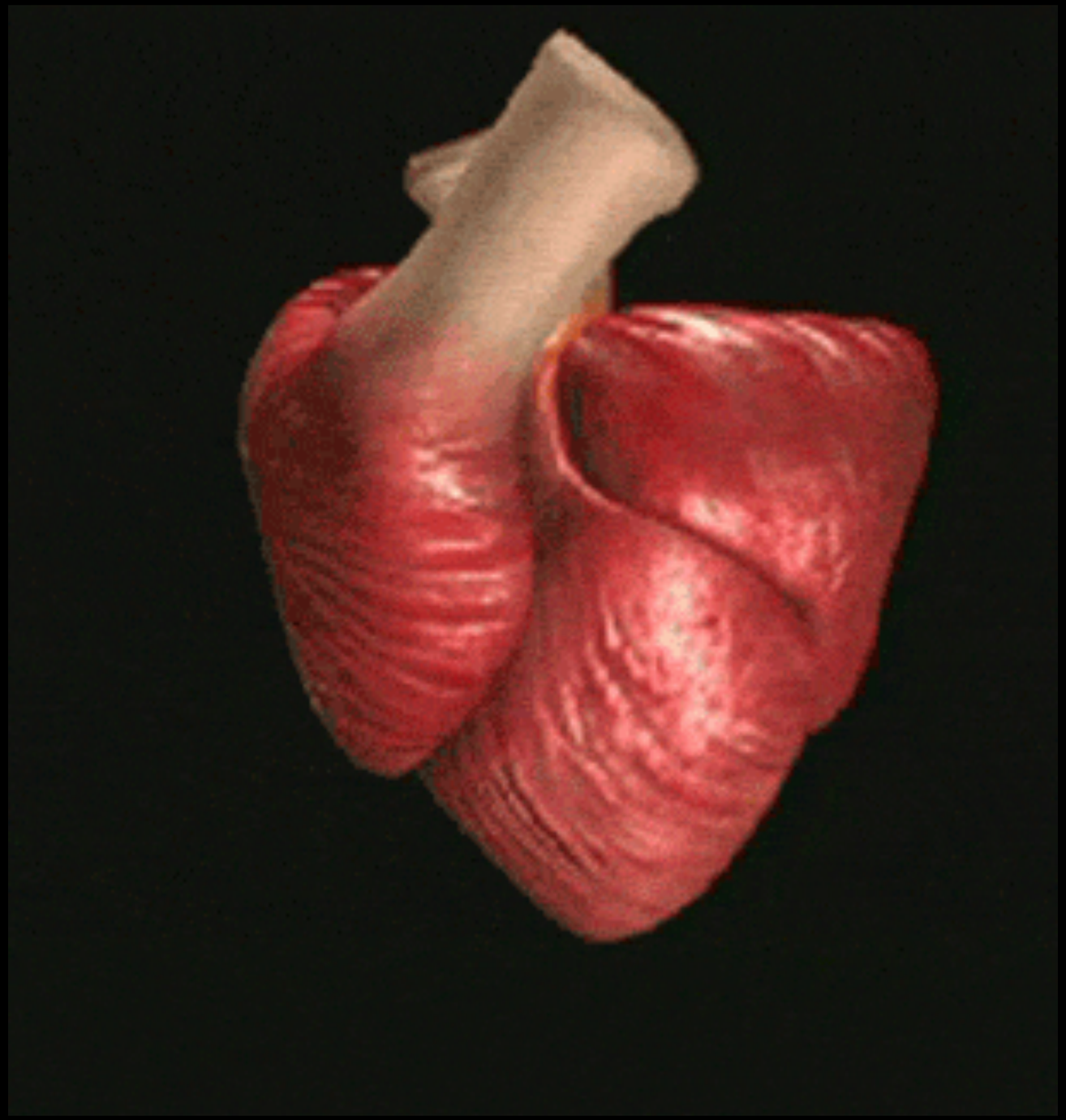

Atrial
musculature
Ventricular
musculature

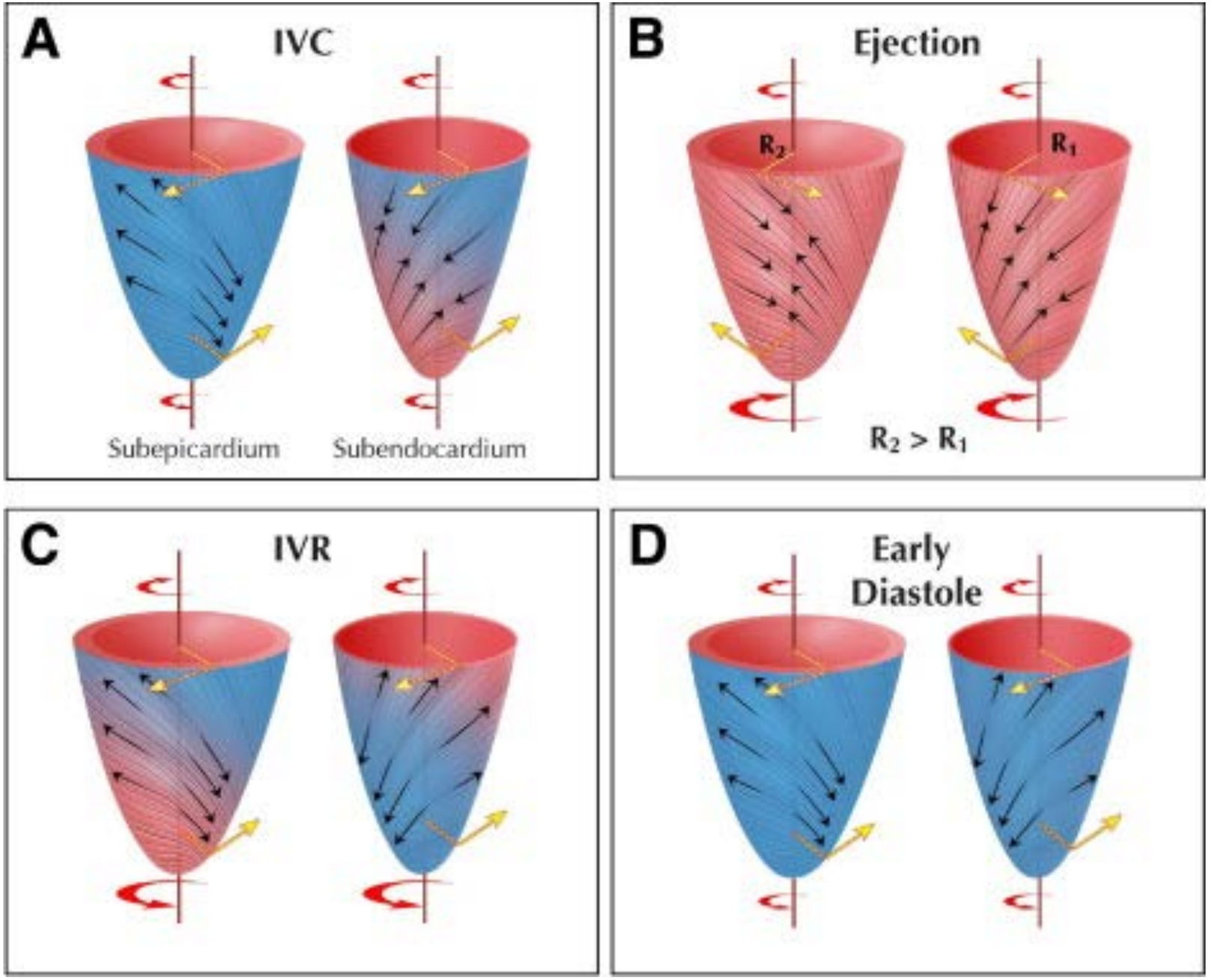

Aortic valve
Inner longitudinal fibers
Superficial longitudinal fibers
Chordae tendinae
Papillary muscle
Inner horizontally looping fibers
Vortex cordis
a
b
c
d

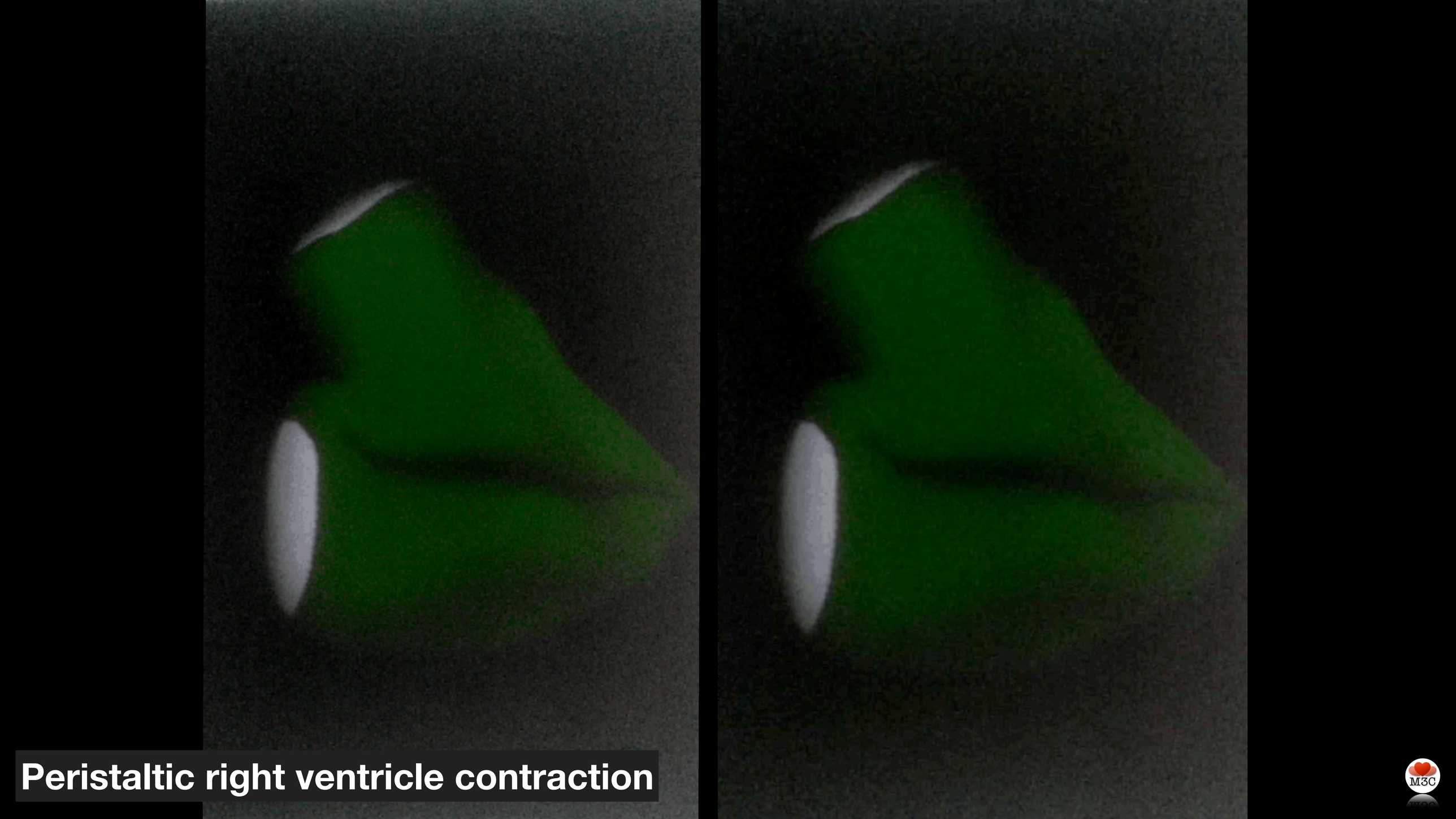

Ao PA
PA
Ao
PA
Ao
Right
Left
Basal Loop
Apical Loop
PA



5mm



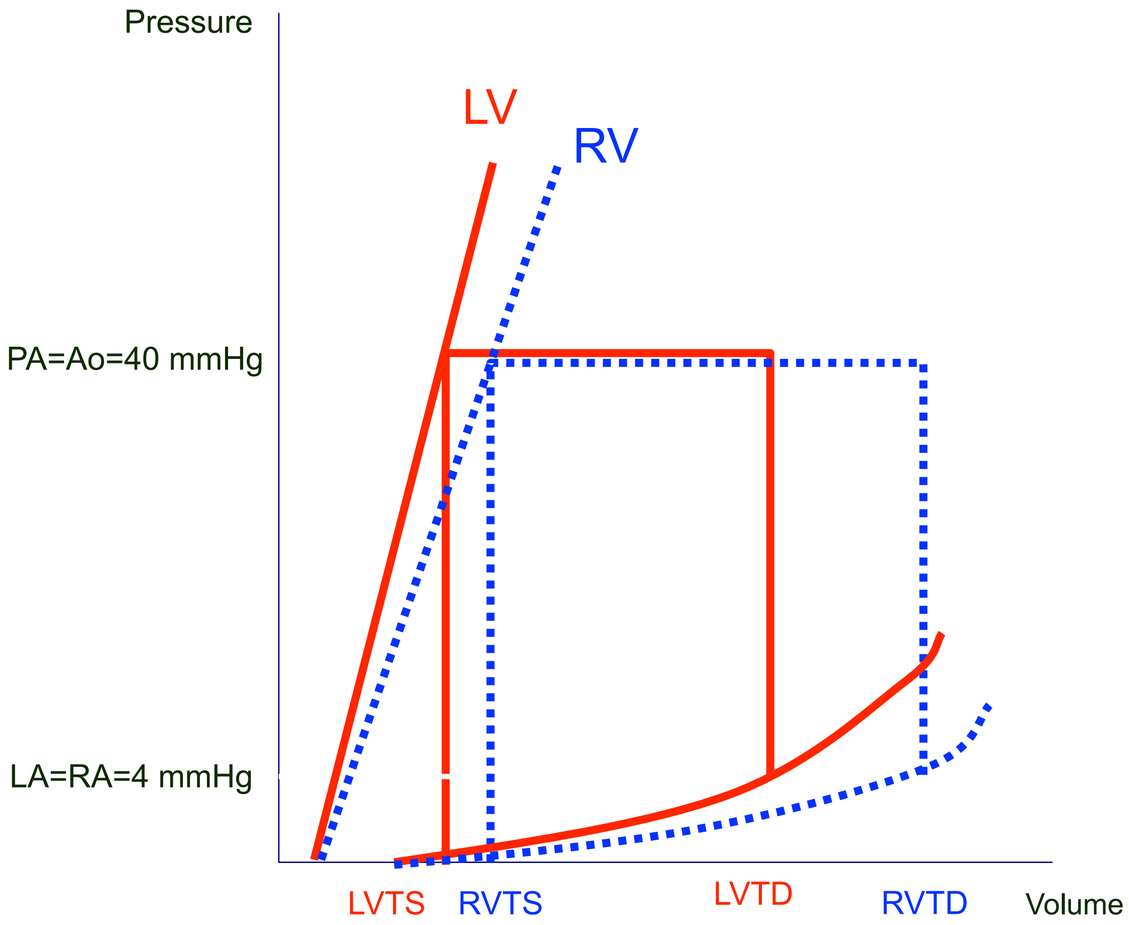

A
IVC
Subepicardium
Subendocardium
B
Ejection
R_2
R_1
$R_2 > R_1$
C
IVR
D
Early
Diastole

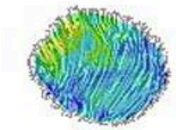


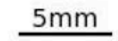
Peristaltic right ventricle contraction

Approche de la fonction myocardique par les courbes pression-volume

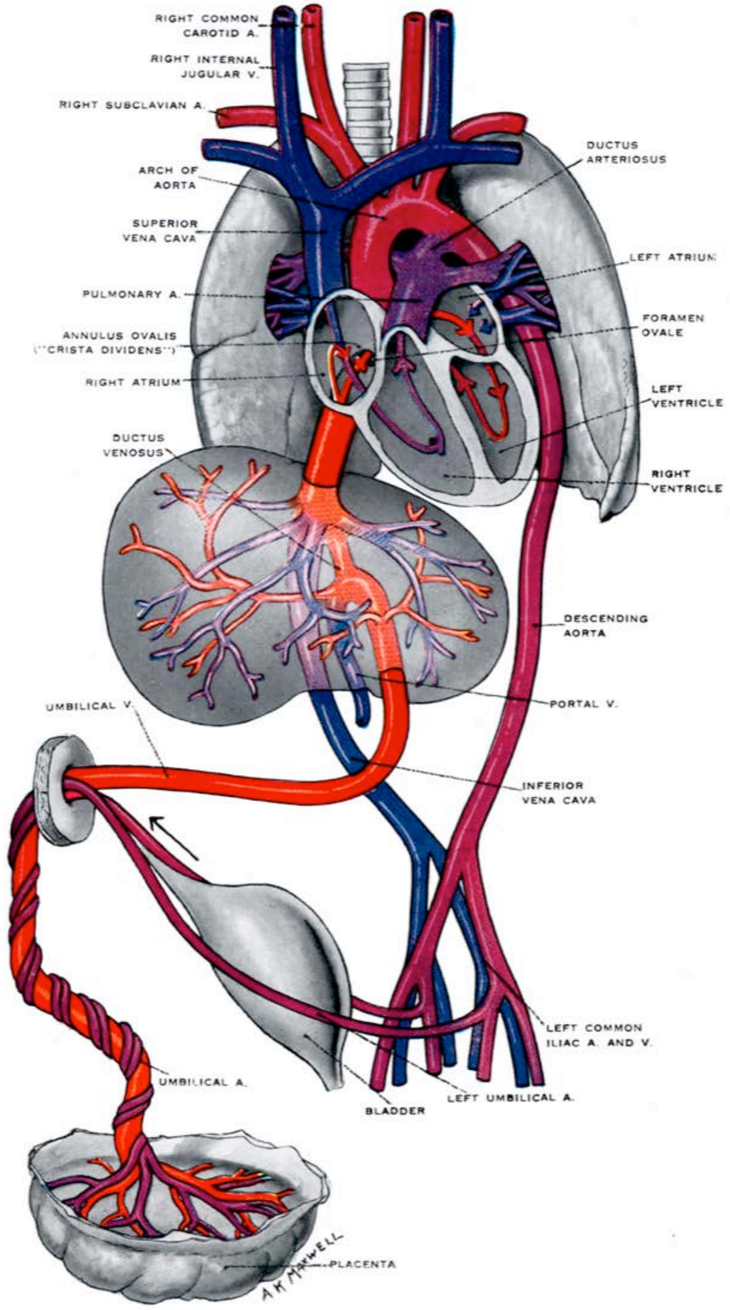
Pressure volume loop in a fetus with ASD and PDA

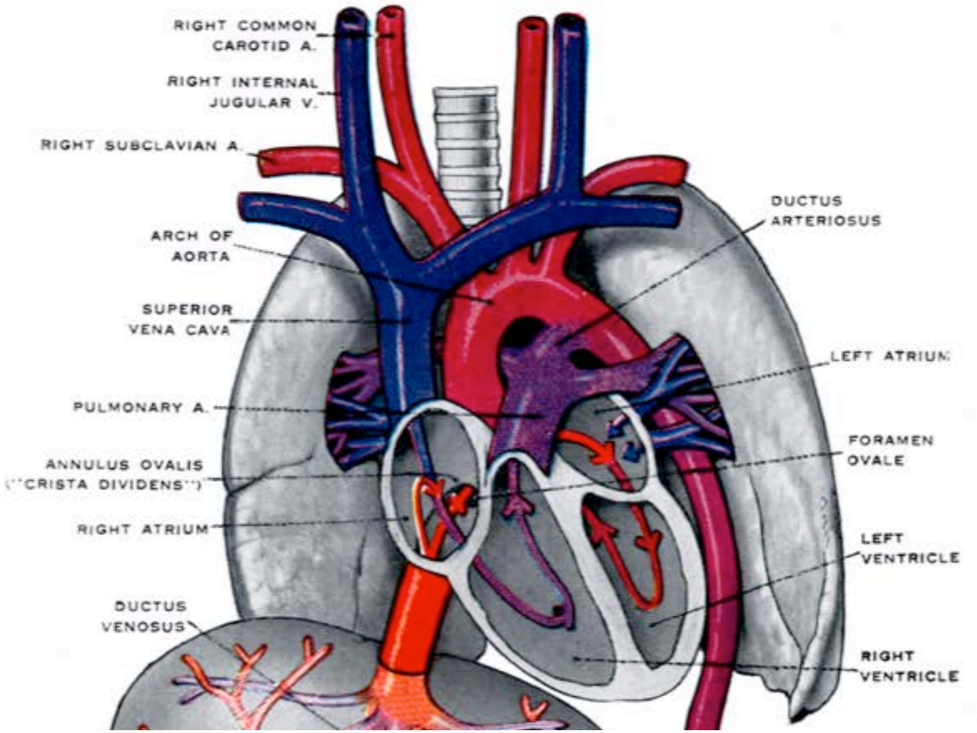


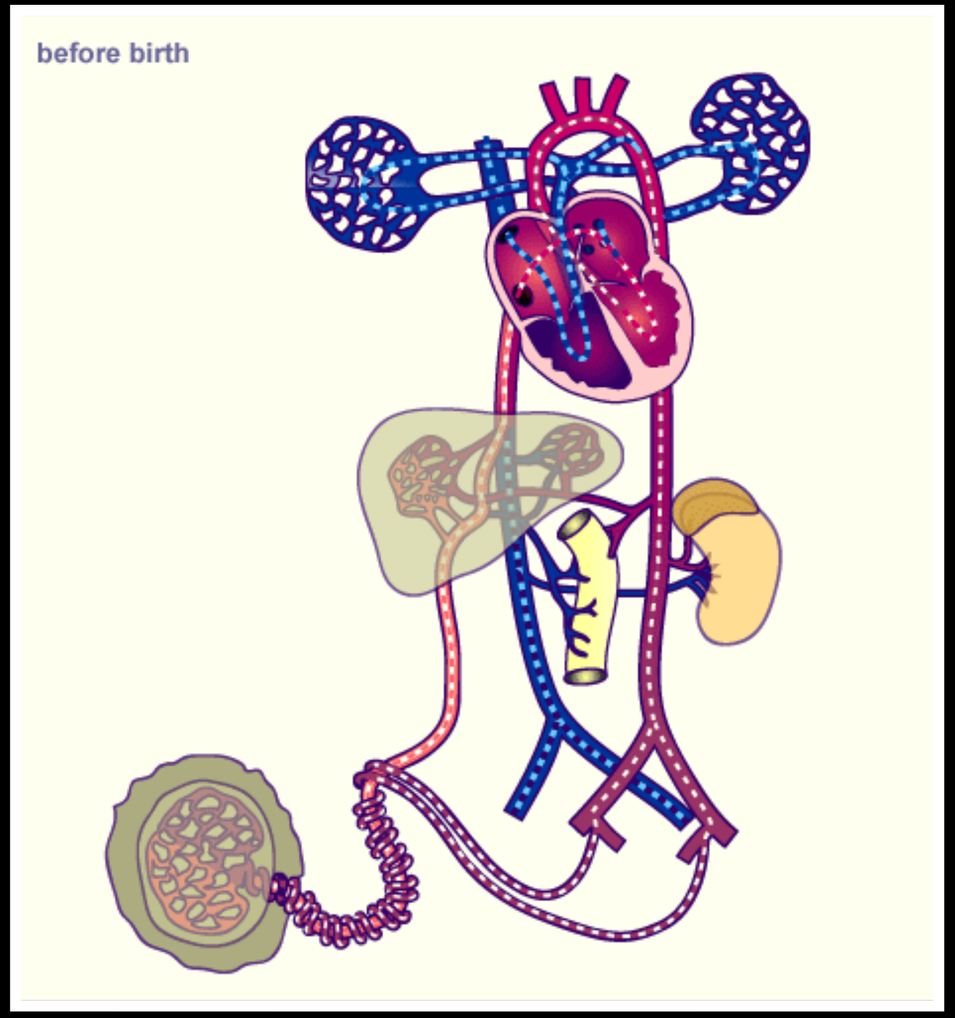


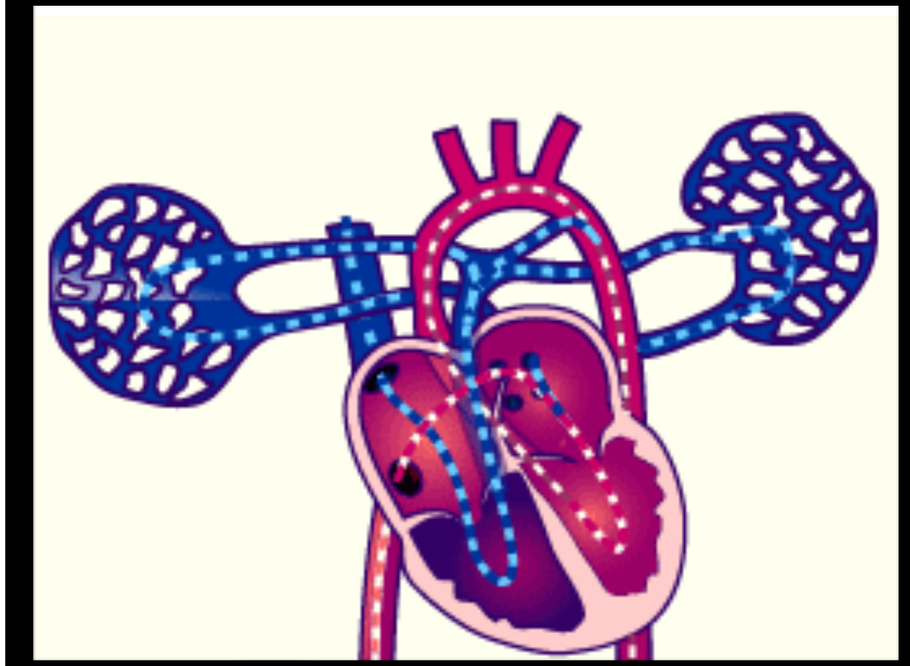


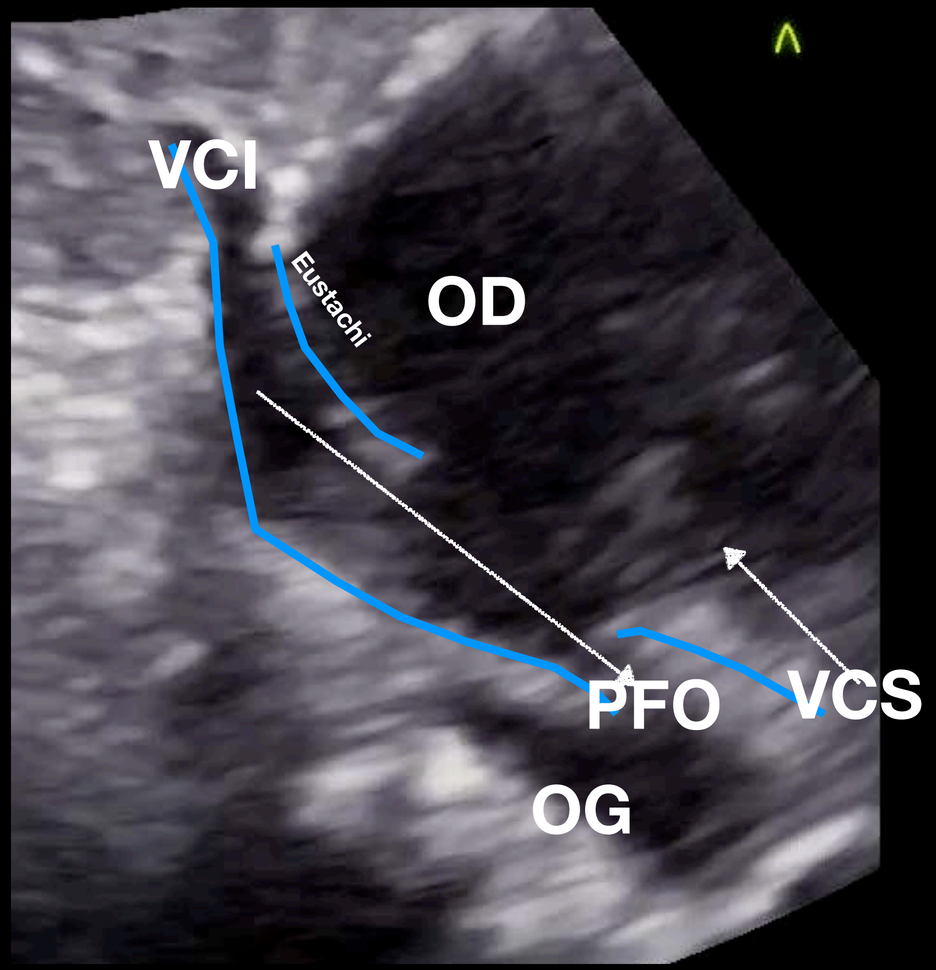
Pour l'oxygénation la circulation est presque en série

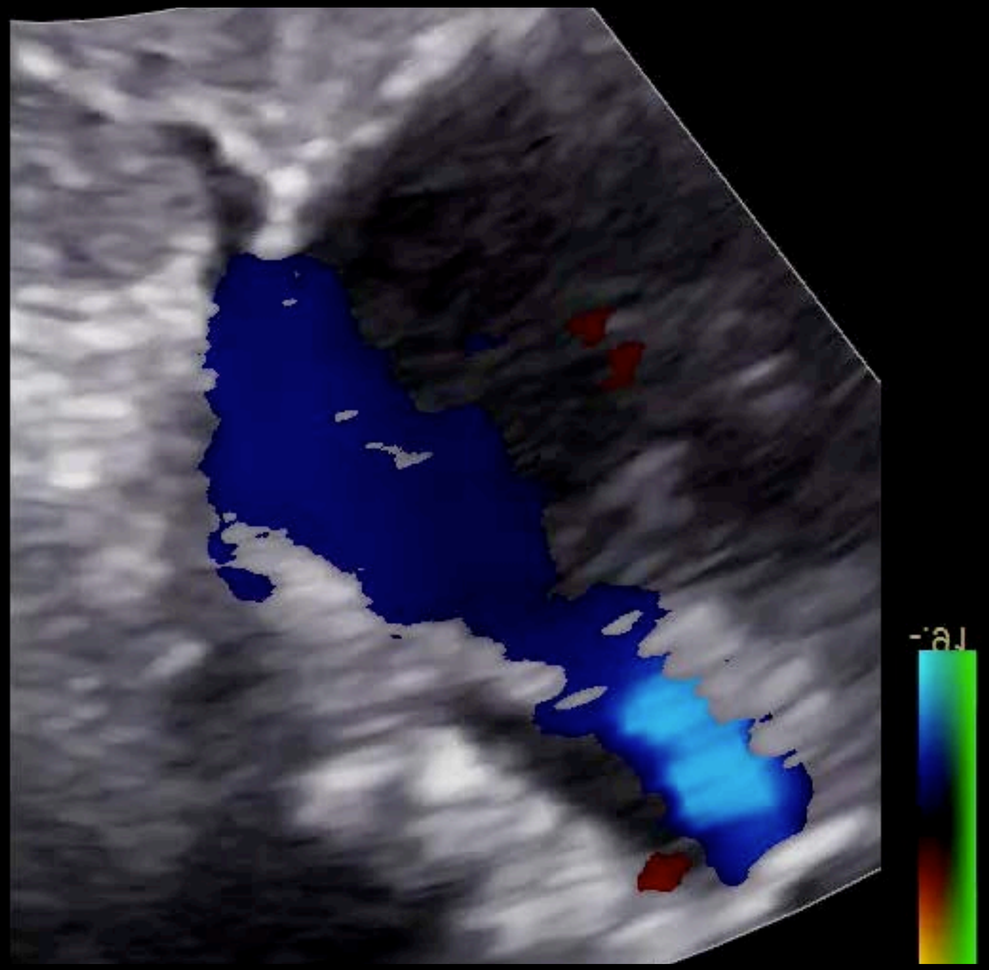




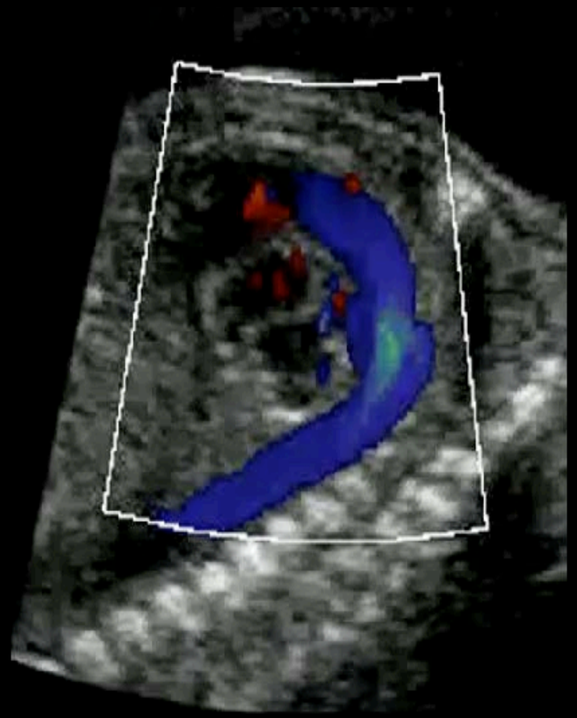

before birth



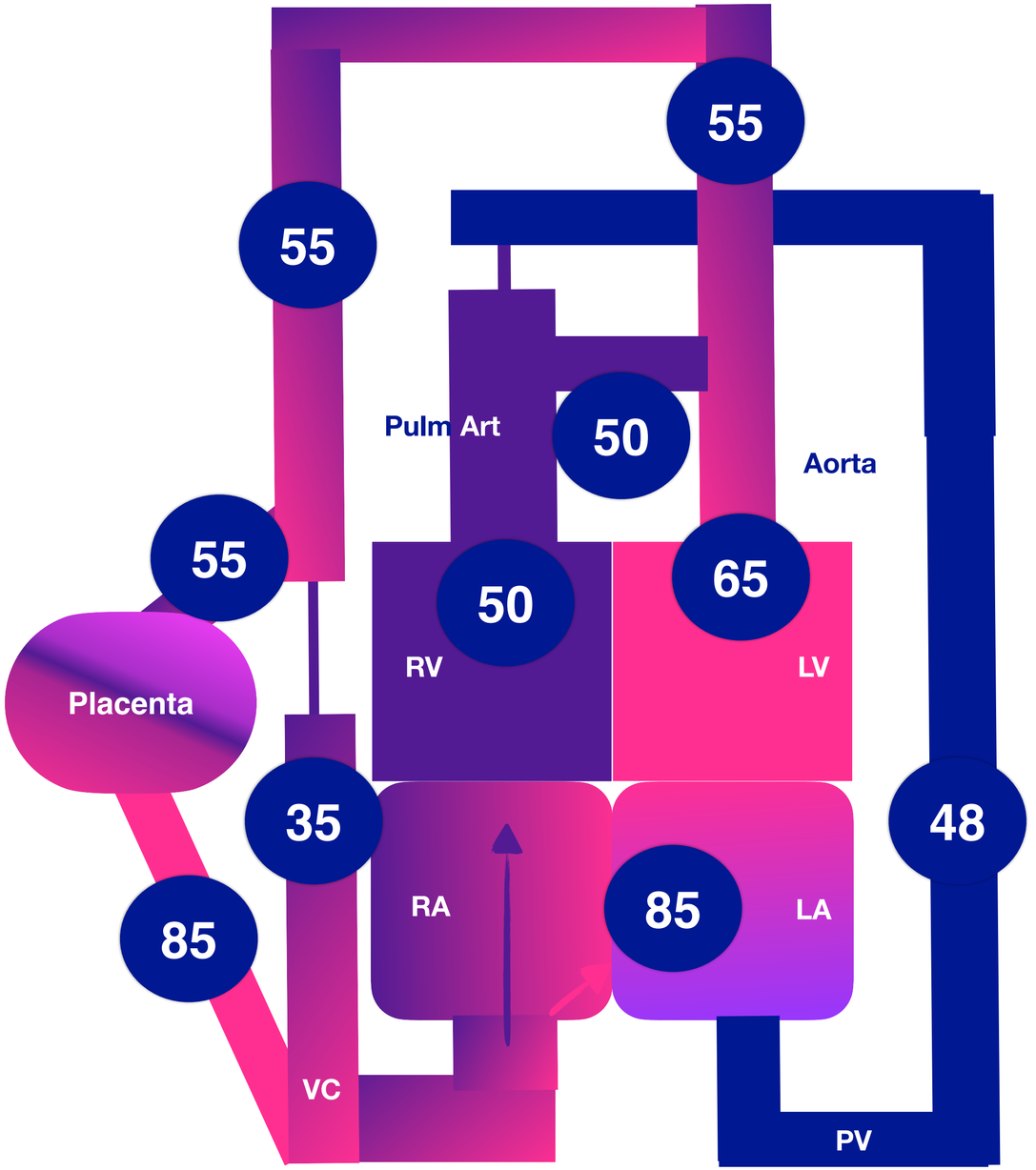

VCI
Eustachi
OD
PFO
VCS
OG



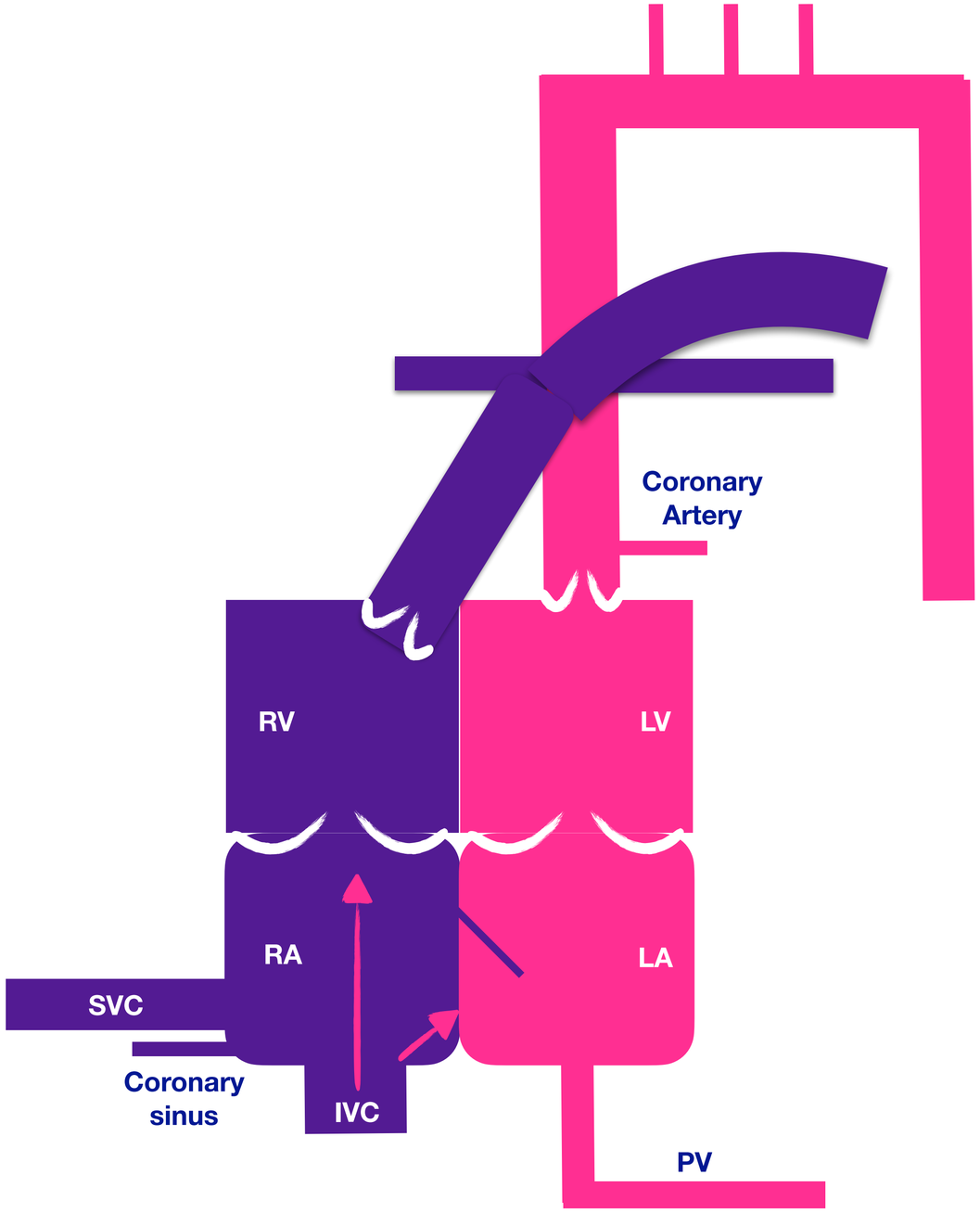
Fetal arterial duct



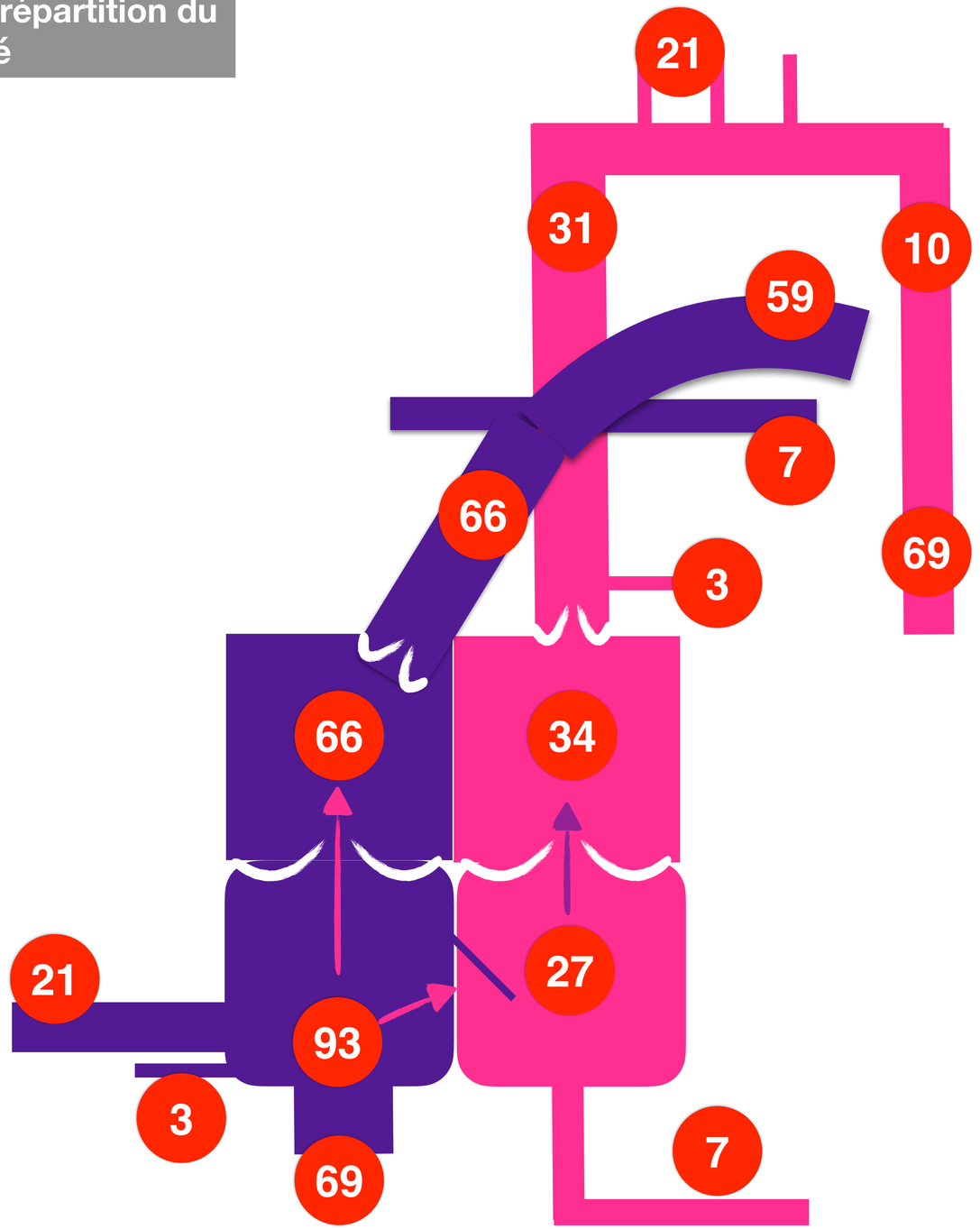
Saturations foetales



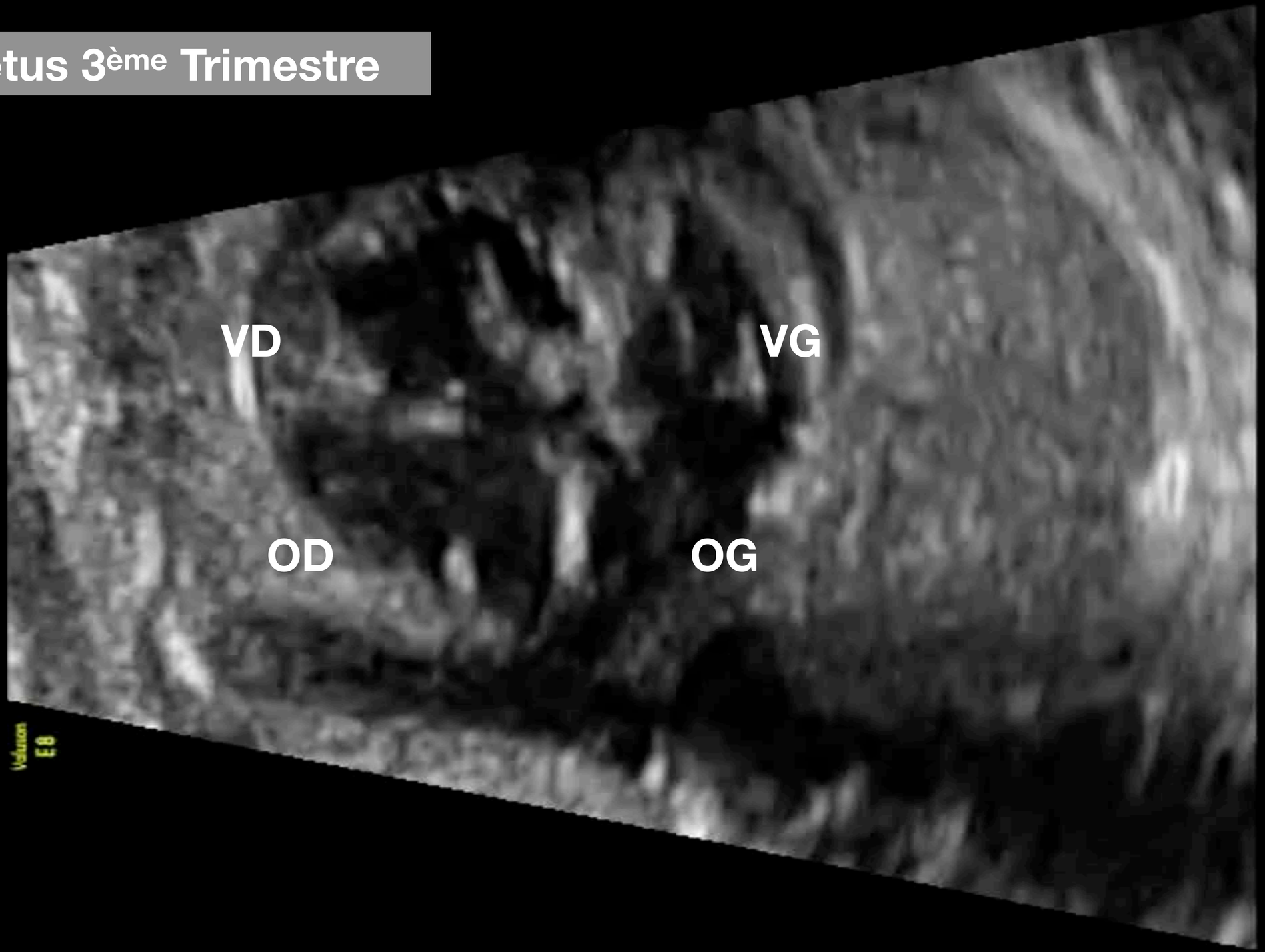
Conséquences développementales de la répartition du débit sanguin foetal combiné



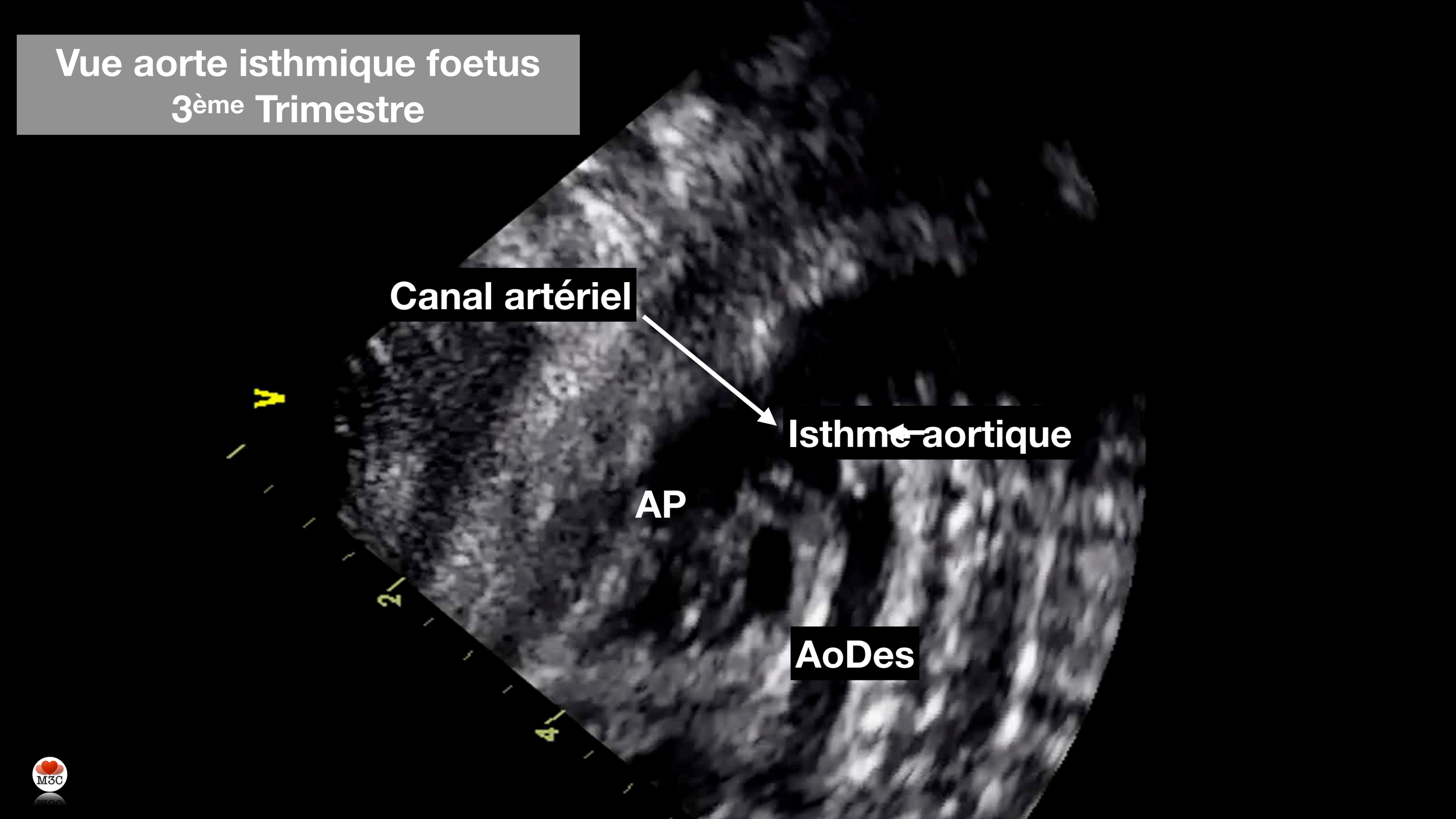
Conséquences développementales de la répartition du débit sanguin foetal combiné



4 cavités foetus 3ème Trimestre



Vue aorte isthmique foetus
3ème Trimestre

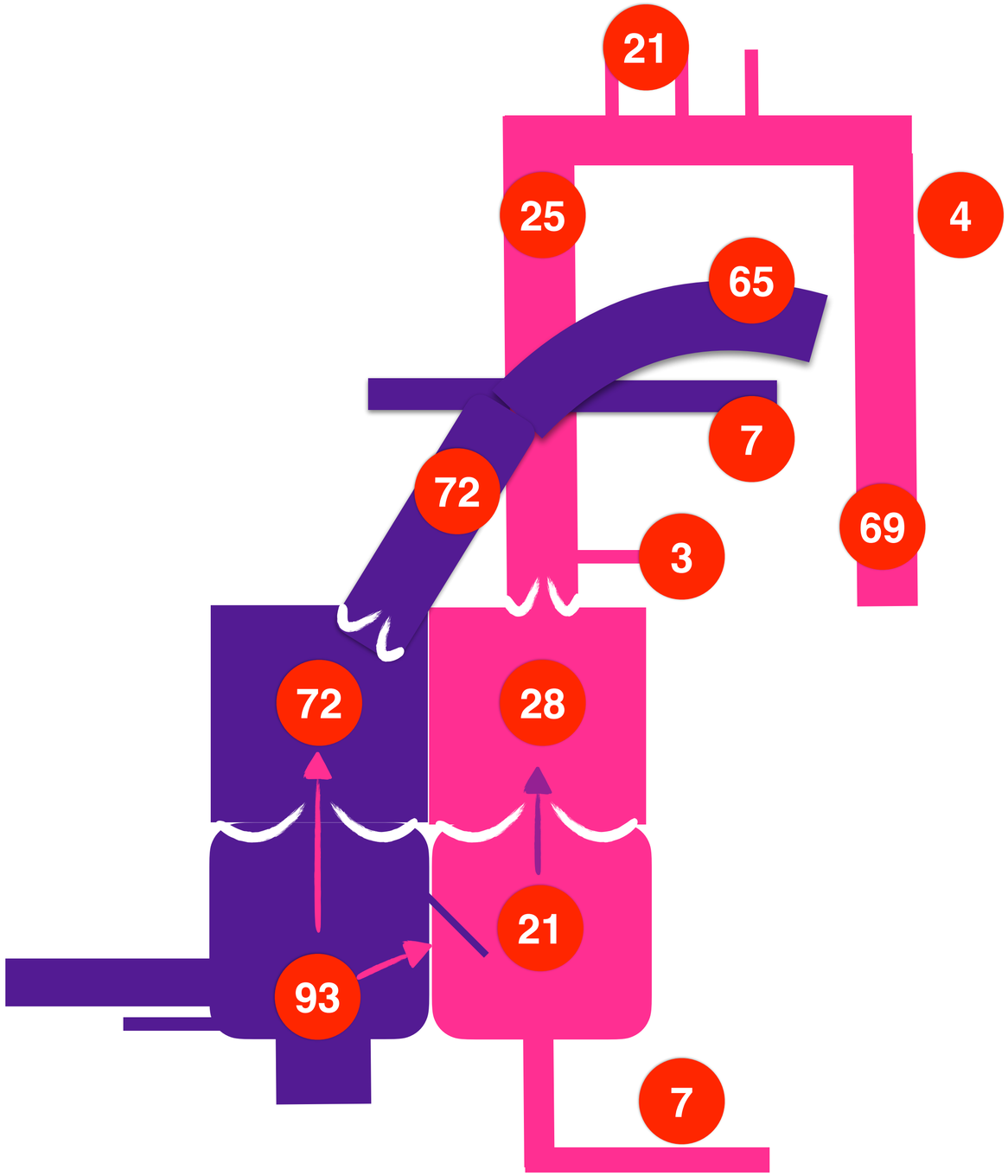


Force et Fragilité

- Force pour la perfusion du fœtus
  - Possible court-circuit d'un ventricule
  - Les discordances A-V ou V-A sont bien tolérées

- Fragilité pour la circulation post-natale
  - L'harmonie du cœur est menacée par des lésions mineures
  - Les cercles vicieux s'installent rapidement

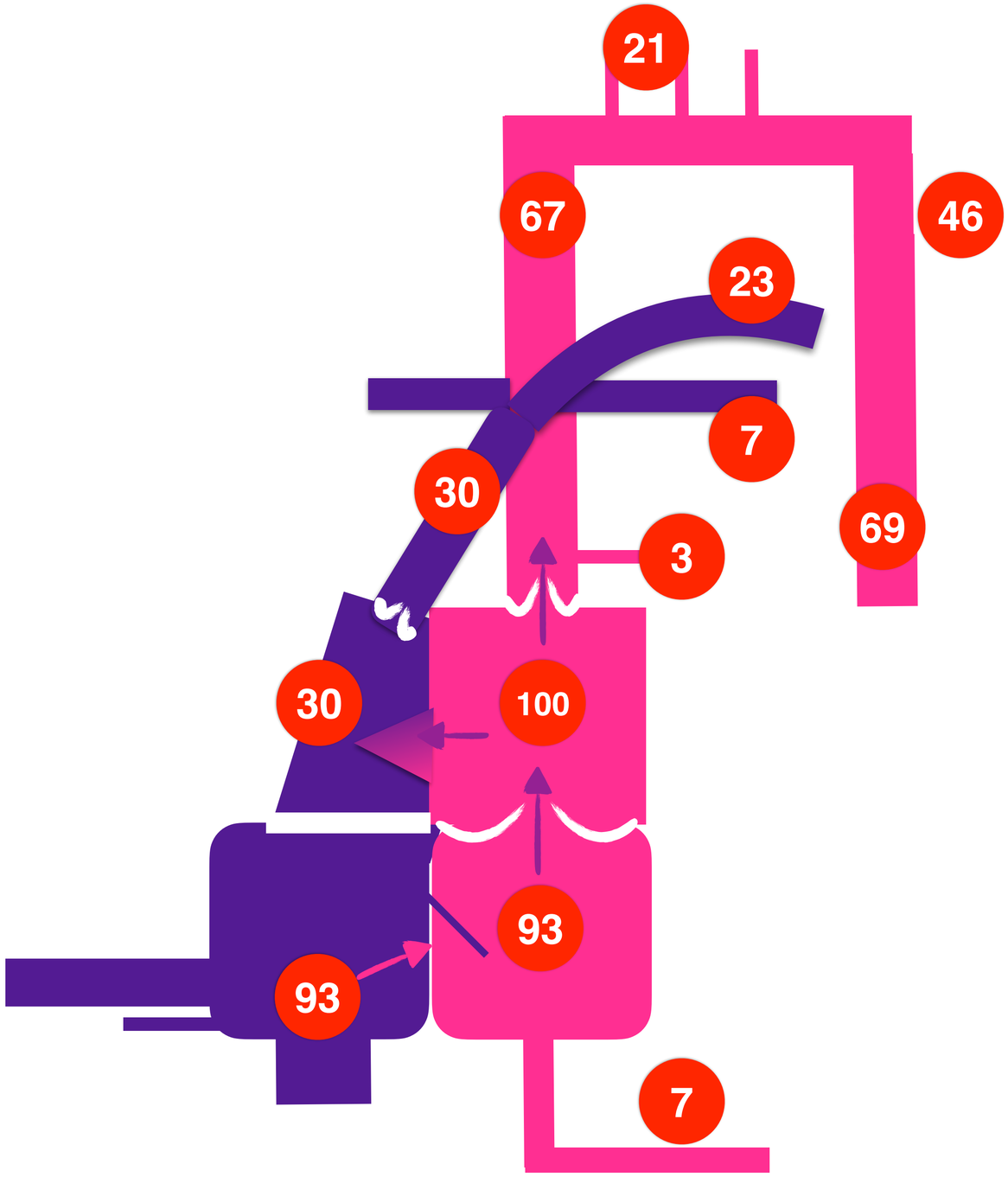
Conséquences développementales des anomalies de la répartition du débit sanguin foetal combiné



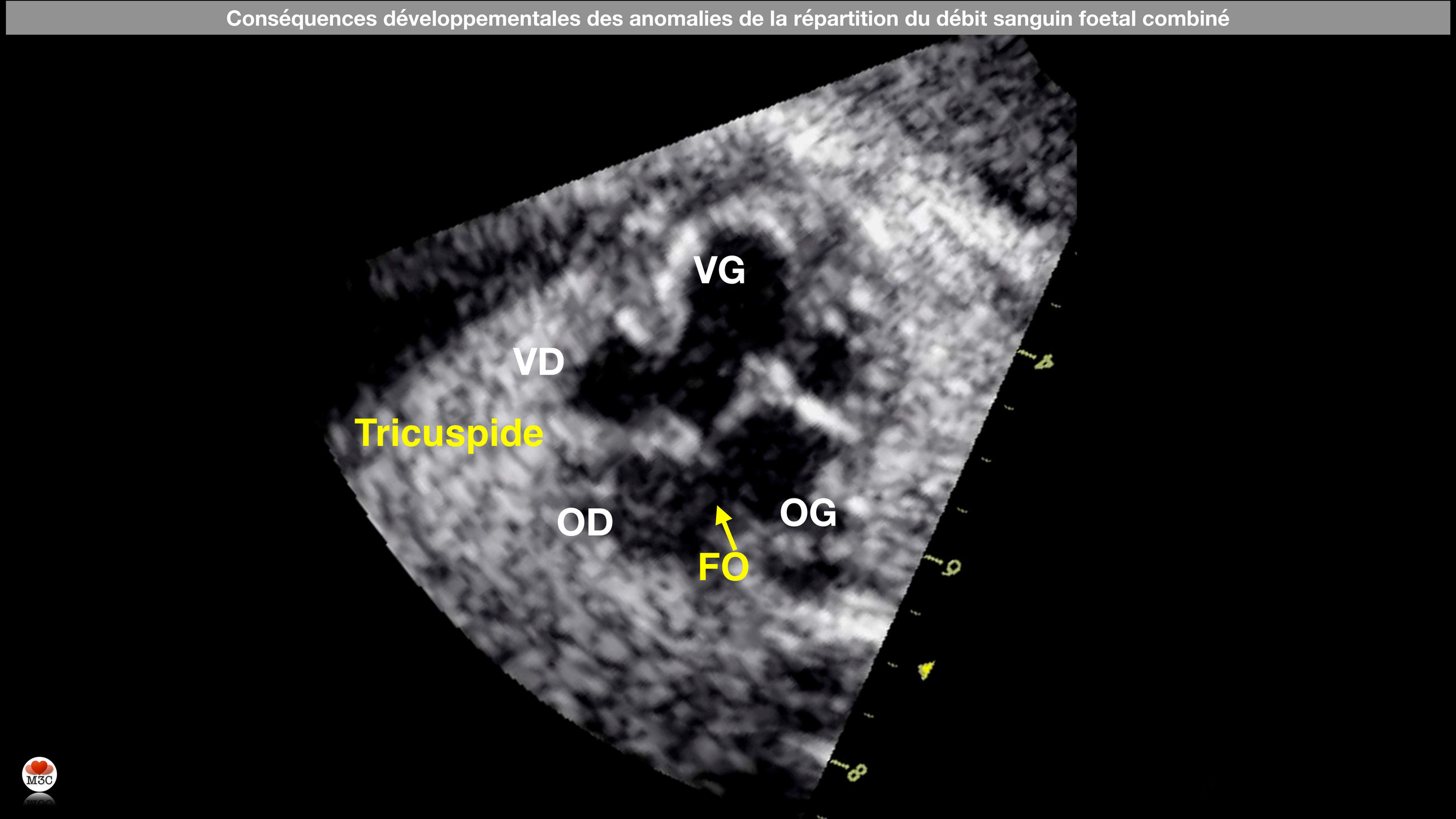
Conséquences développementales des anomalies de la répartition du débit sanguin foetal combiné

Conséquences développementales des anomalies de la répartition du débit sanguin foetal combiné

Atrésie tricuspide Ib

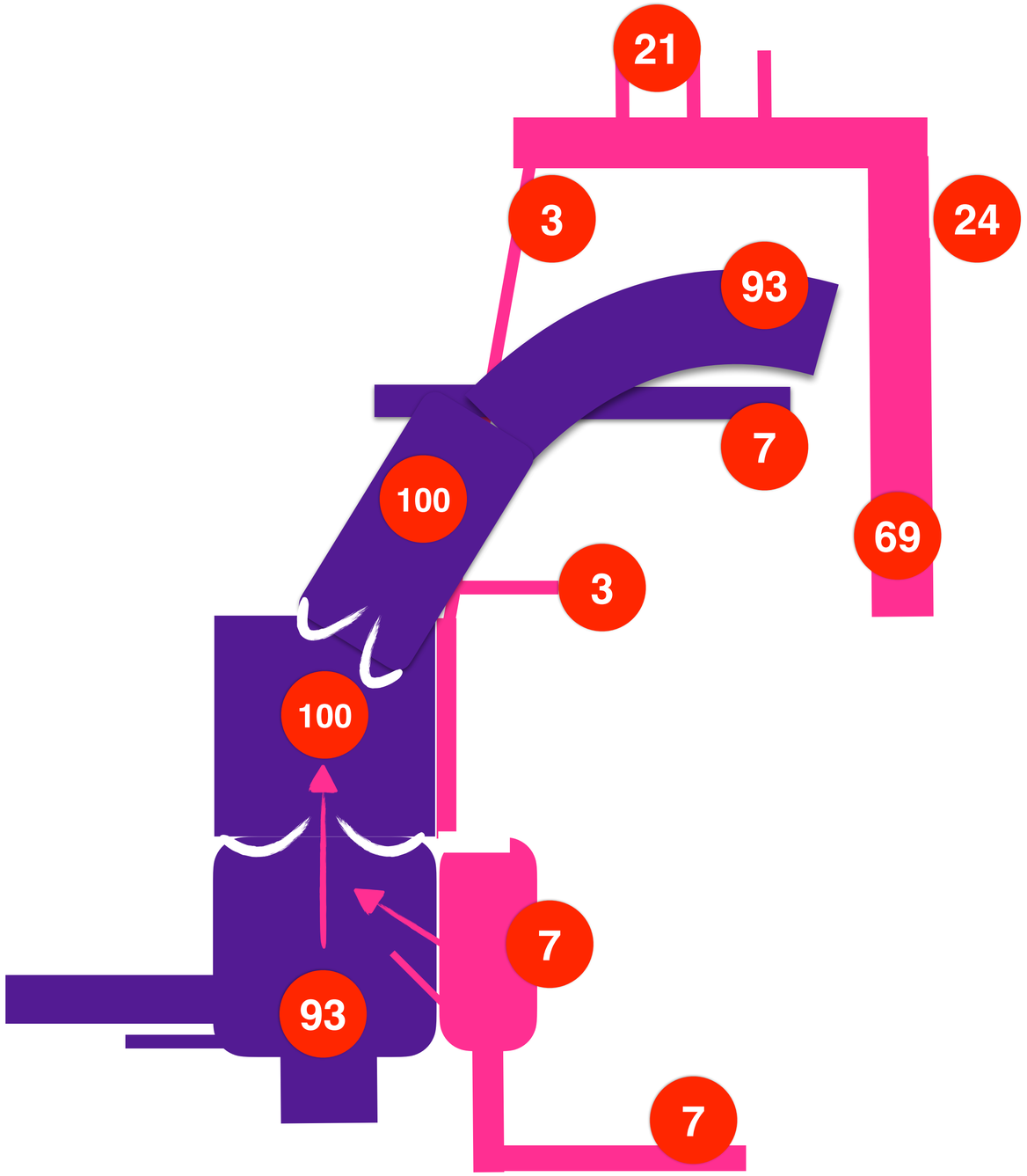


Conséquences développementales des anomalies de la répartition du débit sanguin foetal combiné



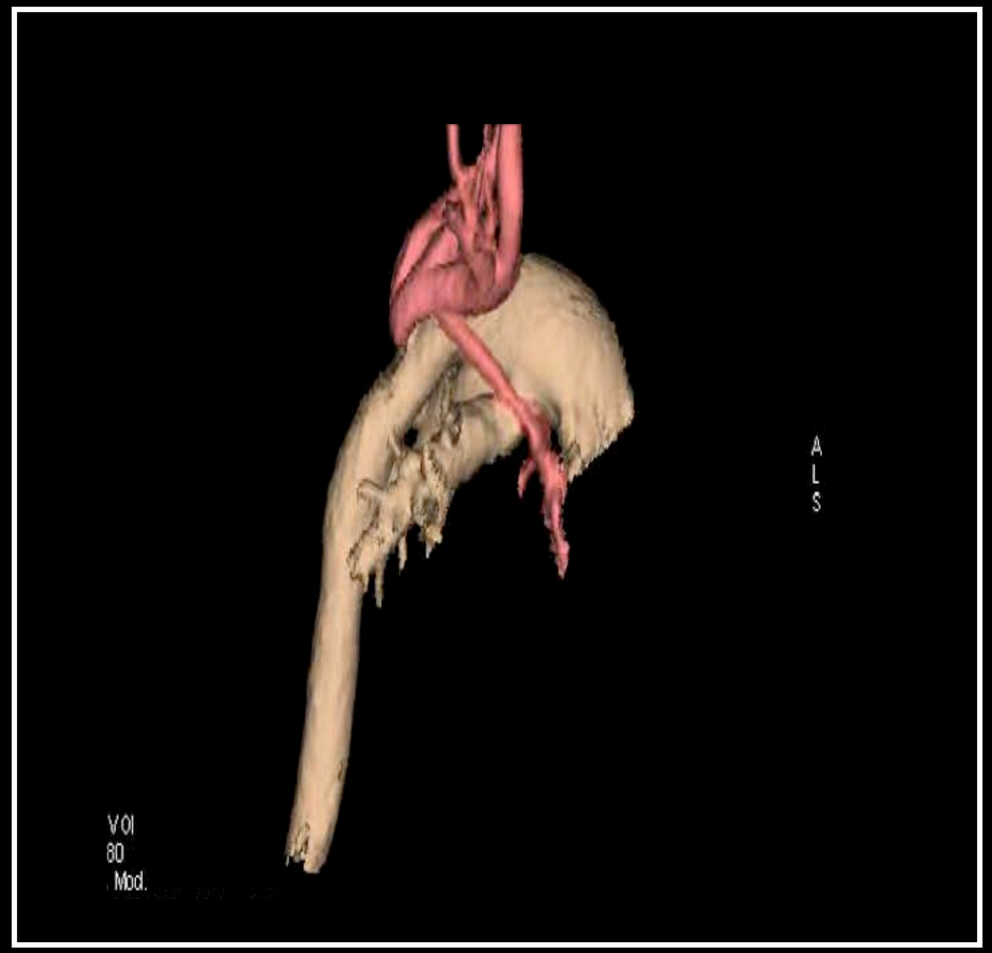
Conséquences développementales des anomalies de la répartition du débit sanguin foetal combiné

Hypoplasie du coeur gauche



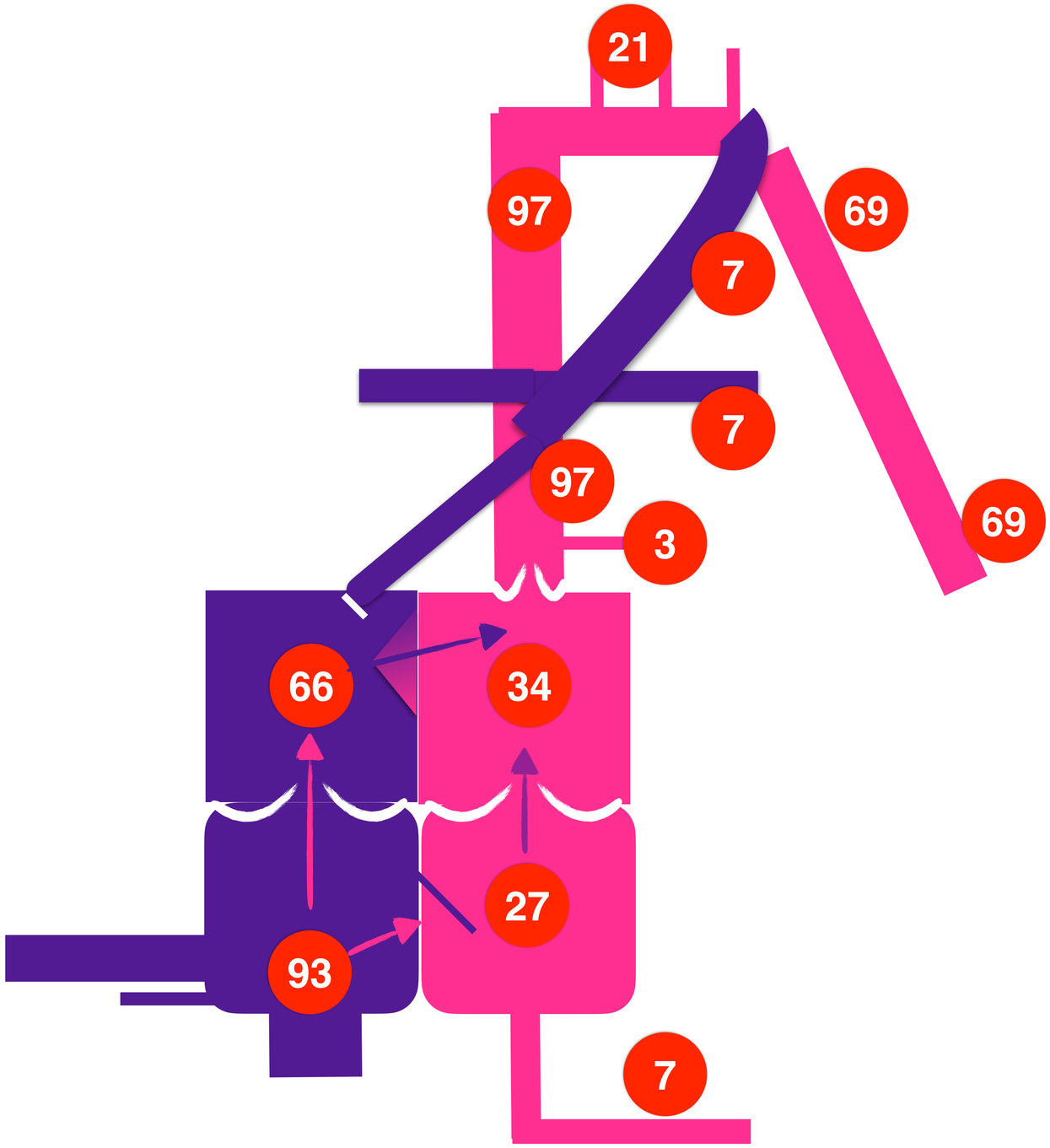
Conséquences développementales des anomalies de la répartition du débit sanguin foetal combiné

Hypoplastic left heart syndrome-Aortic atresia



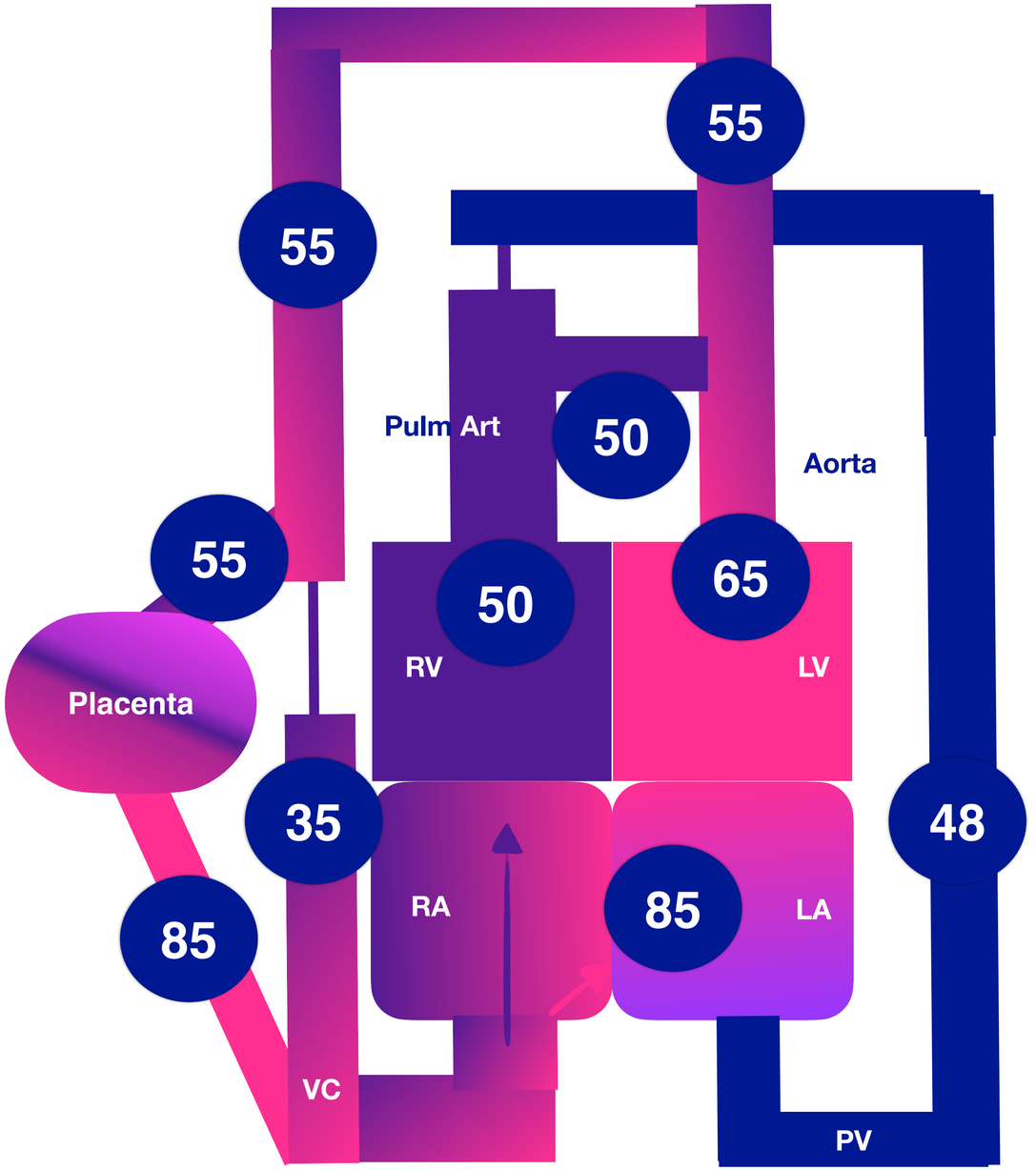
Conséquences développementales des anomalies de la répartition du débit sanguin foetal combiné

Atrésie pulmonaire avec CIV

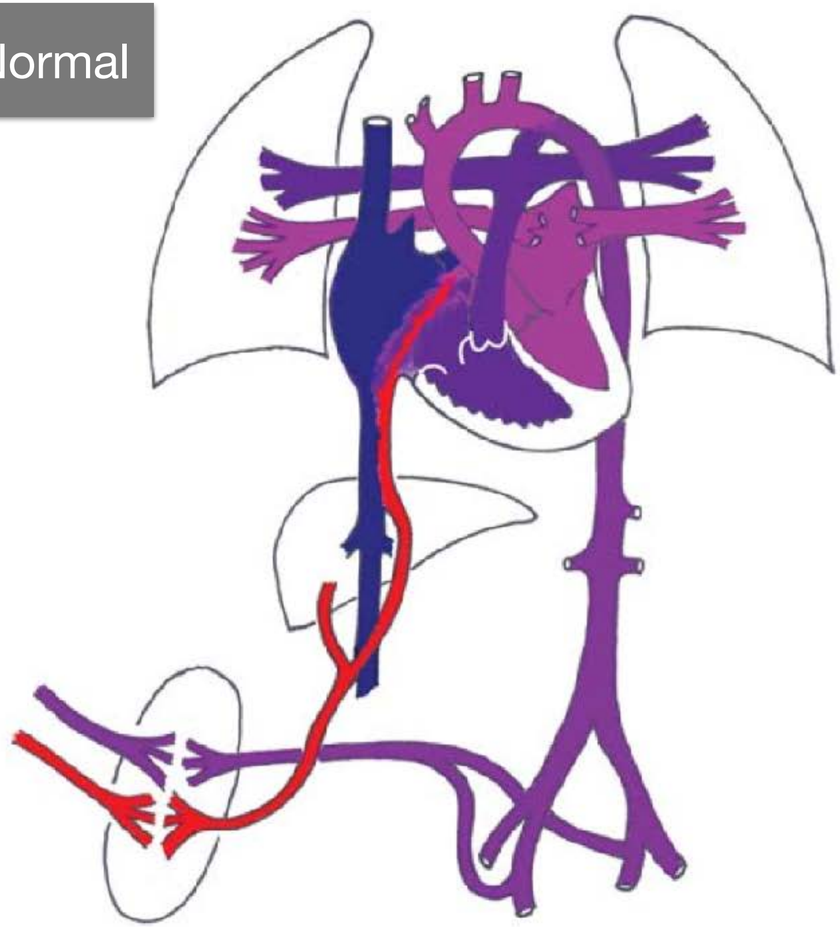


Conséquences développementales des anomalies de la répartition du débit sanguin foetal combiné

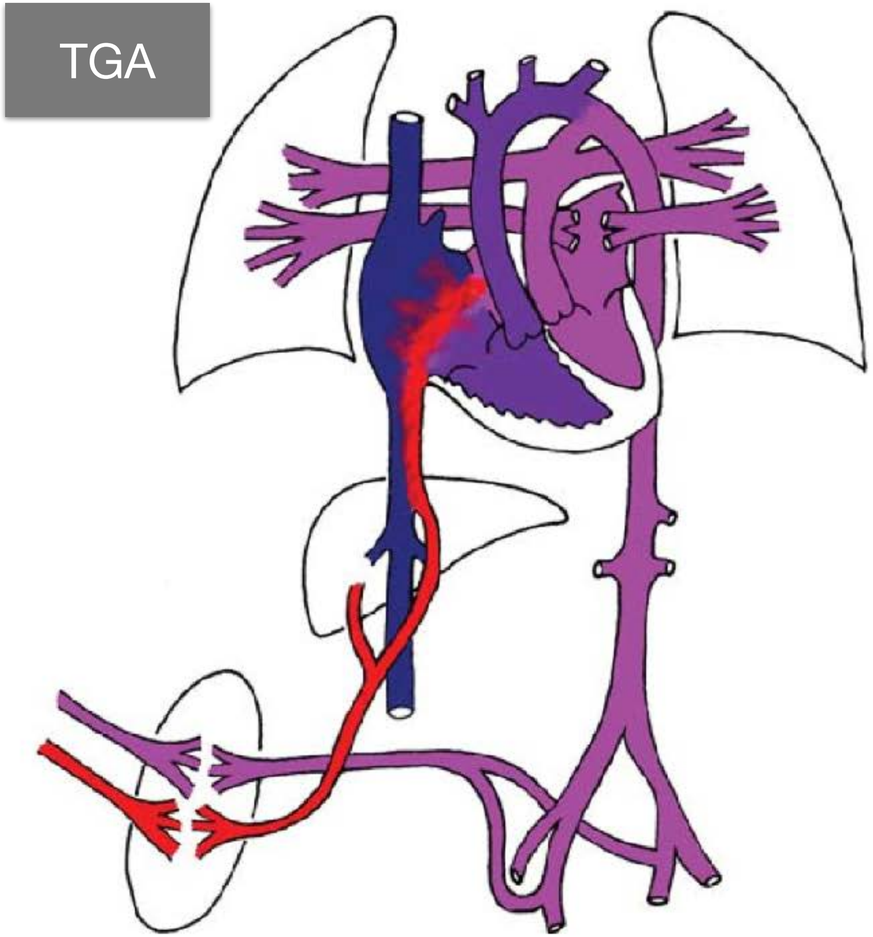
Conséquences développementales des anomalies de l’oxygénation foetale



Normal

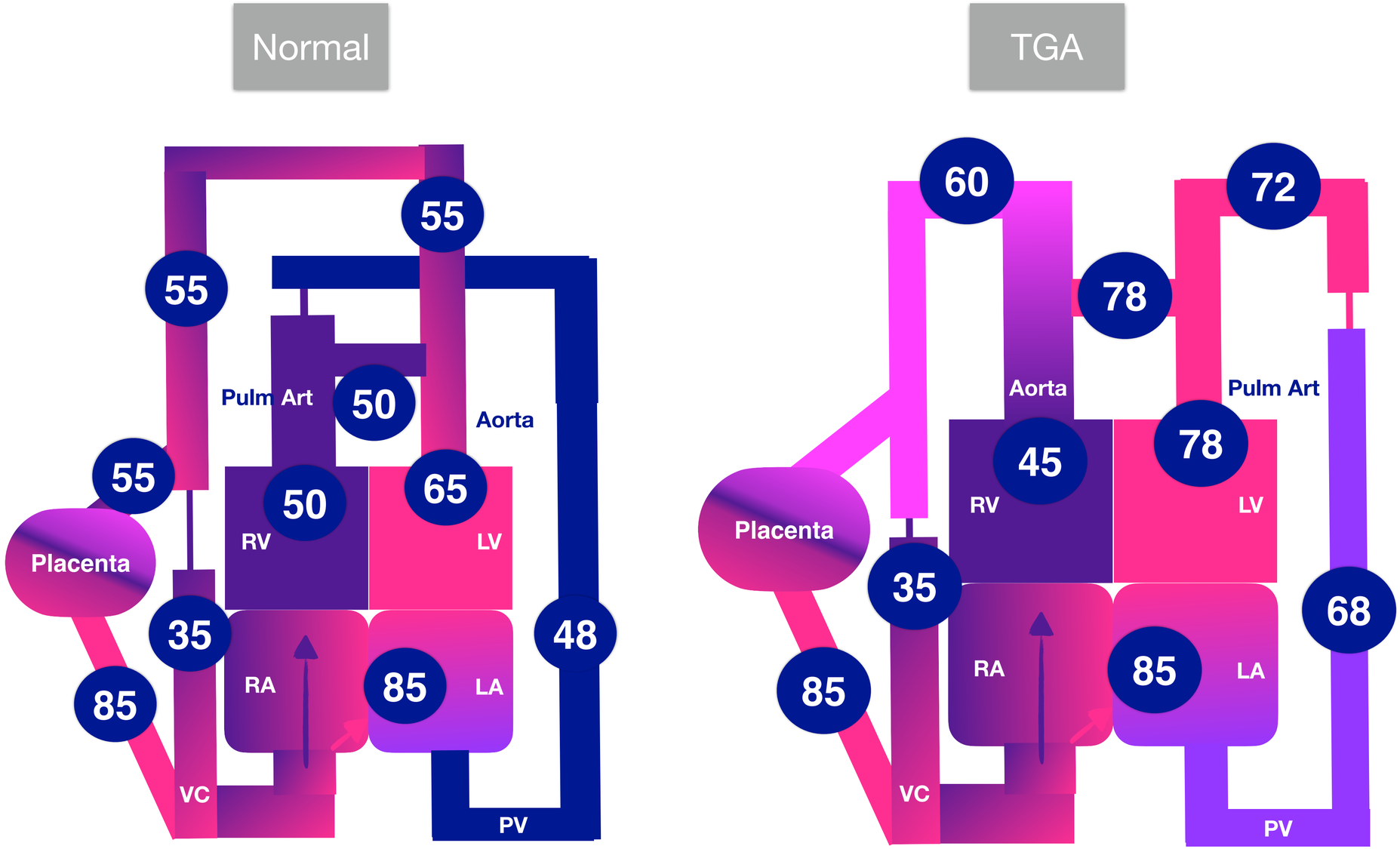


TGA



Sun L et al. Circulation. 2015;131:1313-1323.

Conséquences développementales de la répartition du débit sanguin foetal combiné

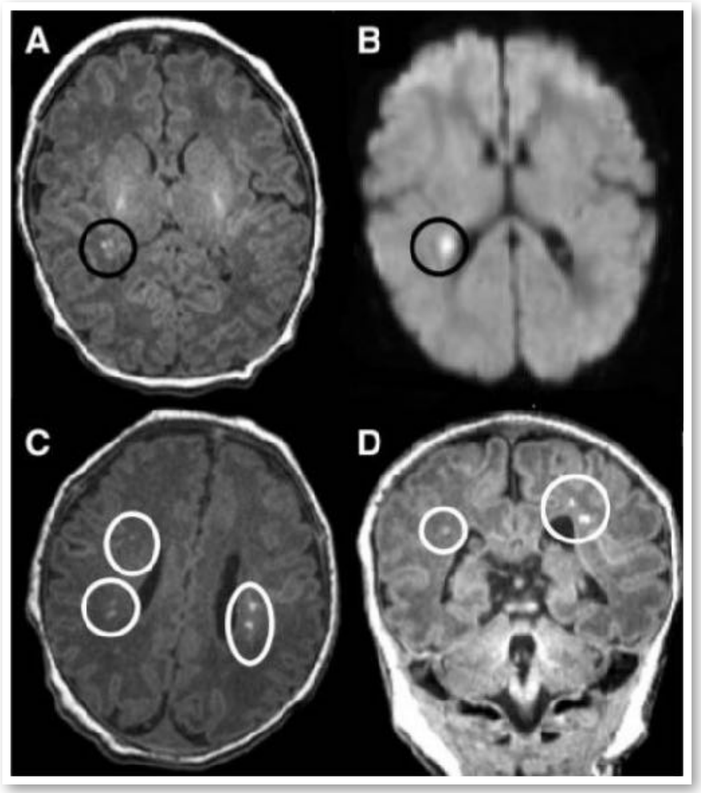


Rudolph A. Ped Res 2007;61:375-80
Prsa M et al. Circ Cardiovas Imaging 2014;7:663-70

Prenatal white matter MRI anomalies in children with cyanotic congenital heart diseases

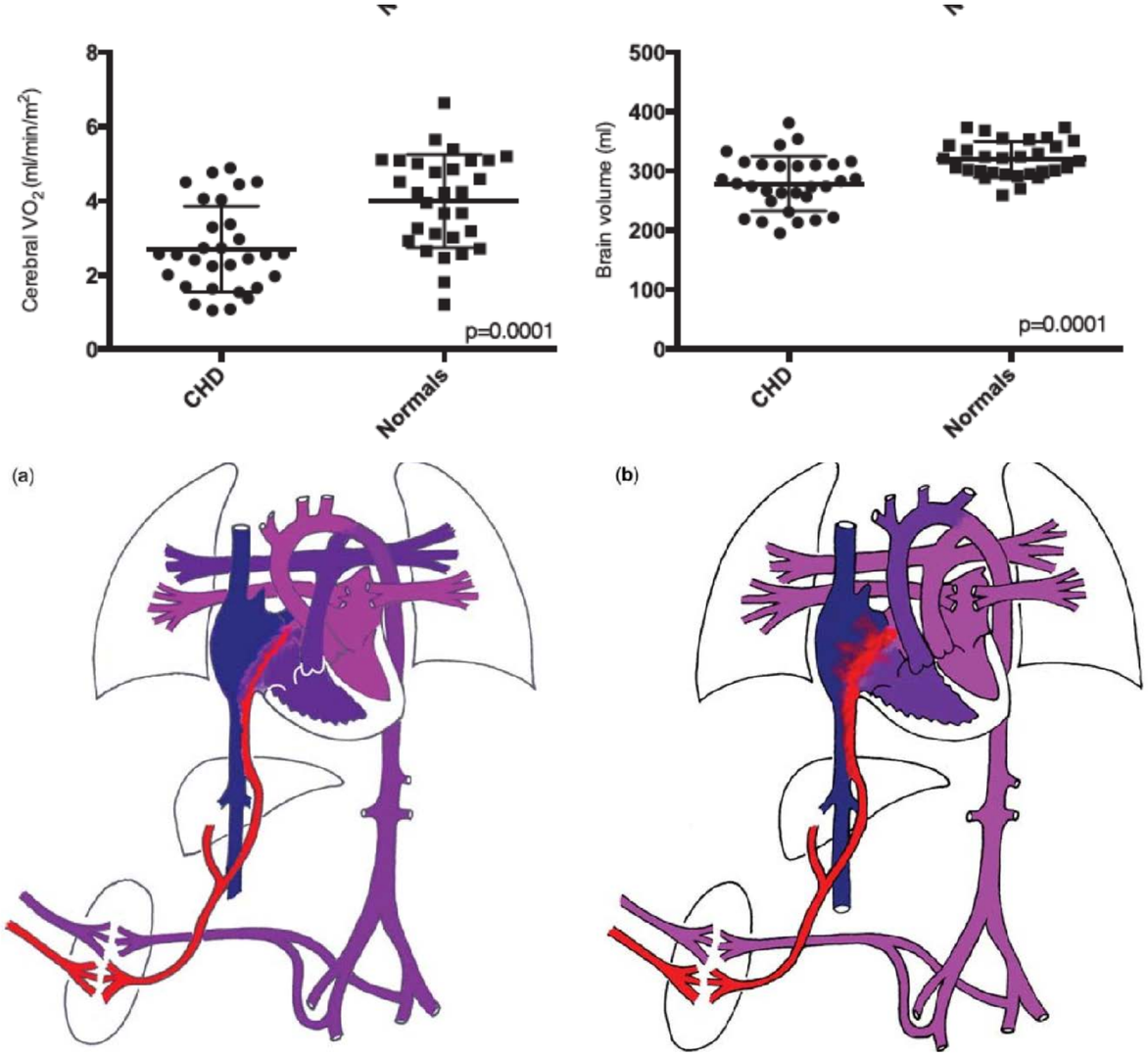
- **White matter lesions in** 30 to 40% of newborns with TGA (Miller et al., 2004; Licht et al., 2009)

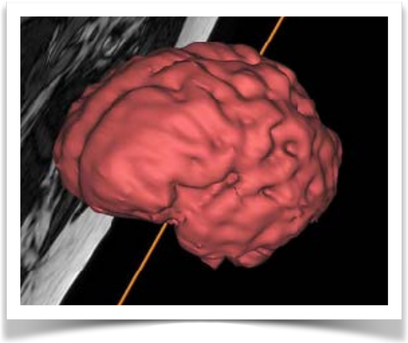
- Same type of anomalies but more severe in complex CHDs such as HLHS (Mahle et al., 2002).



Periventricular white matter lesions in a child with TGA **before** the arterial swicth.
Petit et al., 2009 *in Circulation*

Type of CHD and prenatal brain perfusion

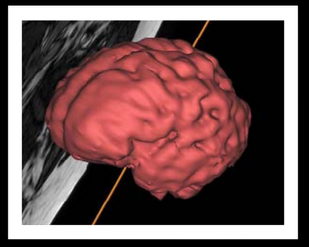




Mechanisms for reduced cerebral oxygenation and impaired brain growth in fetuses with CHD

1-In TGA, streaming results in well oxygenated blood being directed to the pulmonary circulation, whereas the blood supplied to brain is derived largely from more deoxygenated blood returning from the caval veins.
2-Reduction in Umbilical Vein Sao2, which is suggestive of abnormal placental function and results in lower fetal O2 delivery even in the setting of normal CVO and UV flow.

Sun L et al. Circulation. 2015;131:1313-1323.

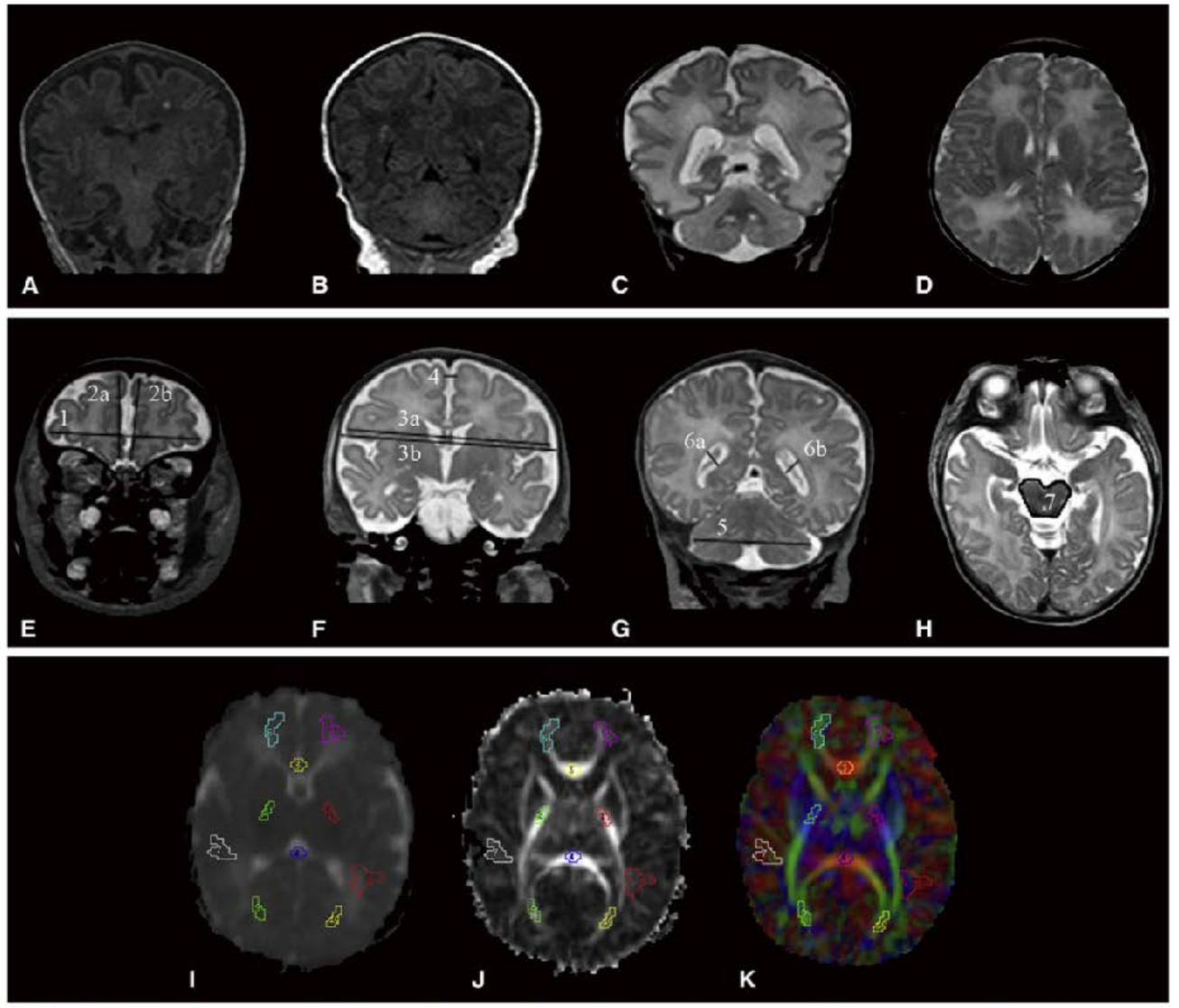


Brain dysmaturation observed in CHD appears to confer increased susceptibility to white matter injury in the perioperative period and neurodevelopmental deficits at 2 years.

The identification of fetal hypoxia as a potentially modifiable cause of delayed fetal brain development
may be clinically significant.

Oxygen saturations in the fetal sheep and human fetuses circulation can be augmented through increases in the oxygen concentration of maternal inhaled air.

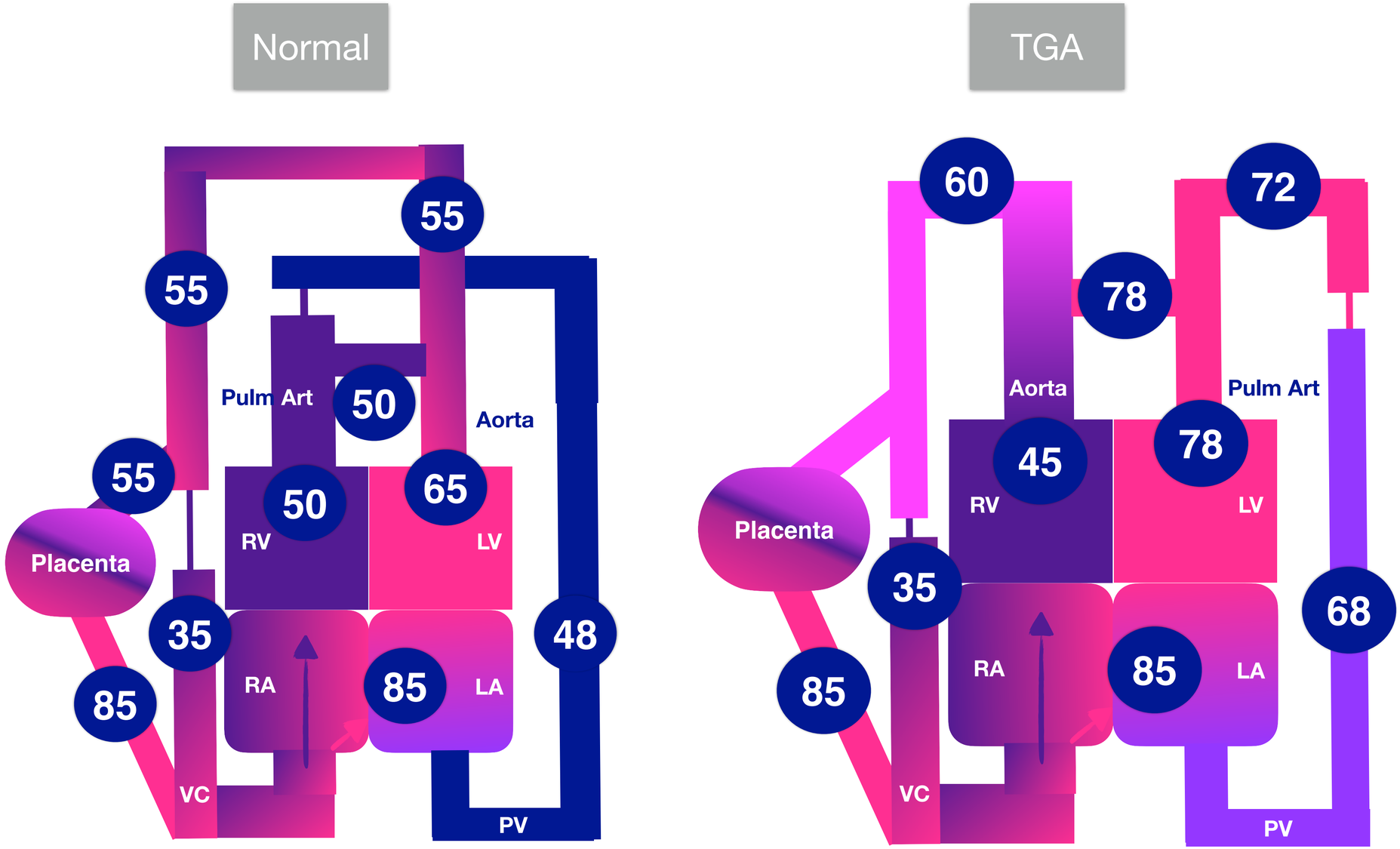
Maternal hyperoxygenation could be a method to improve brain development in utero



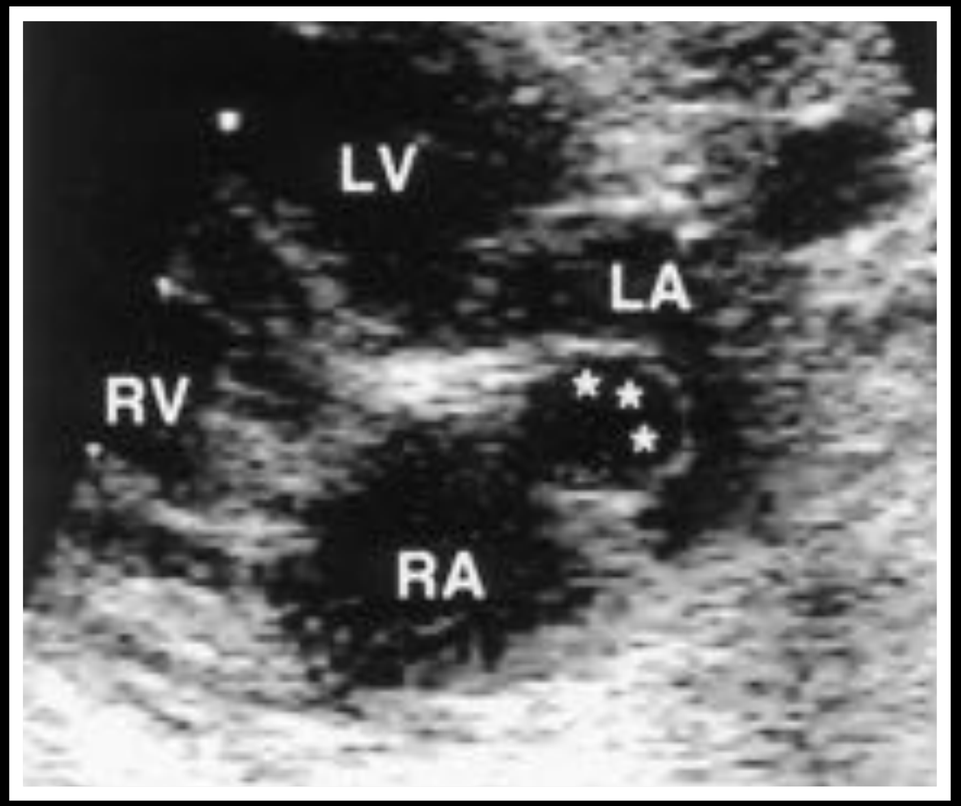
IGURE 1. A–D, Qualitative scoring abnormalities. A, T_1-weighted image with abnormalities that included focal signal abnormality, delayed myelination posterior limb of the internal capsule, increased extra-axial space, and delayed gyrification. B, T_1-weighted image with bilateral focal signal abnormalities nd delayed gyrification. C, T_2-weighed image with ventriculomegaly, diffuse excessive high-signal intensity *(DEHSI)*, increased extra-axial space, and oderate-to-severe delay in gyrification. D, T_2-weighted image with DEHSI. E–H, Subset of brain metrics. 1, Bifrontal diameter; 2a, right frontal height; b, left frontal height; 3a, brain biparietal diameter; 3b, bone biparietal diameter; 4, interhemispheric distance; 5, transverse cerebellar diameter; 6a, right entricular diameter; 6b, left ventricular diameter; and 7, brainstem area. I–K, Diffusion imaging: I, mean diffusivity; J, fractional anisotropy; K, red, green, lue color plot. Regions of interest were the same for each image (from top to bottom): left and right frontal white matter, genu of the corpus collosum, left nd right posterior limb of the internal capsule, splenium of the corpus collosum, left and right subcortical white matter, and left and right optic radiation.

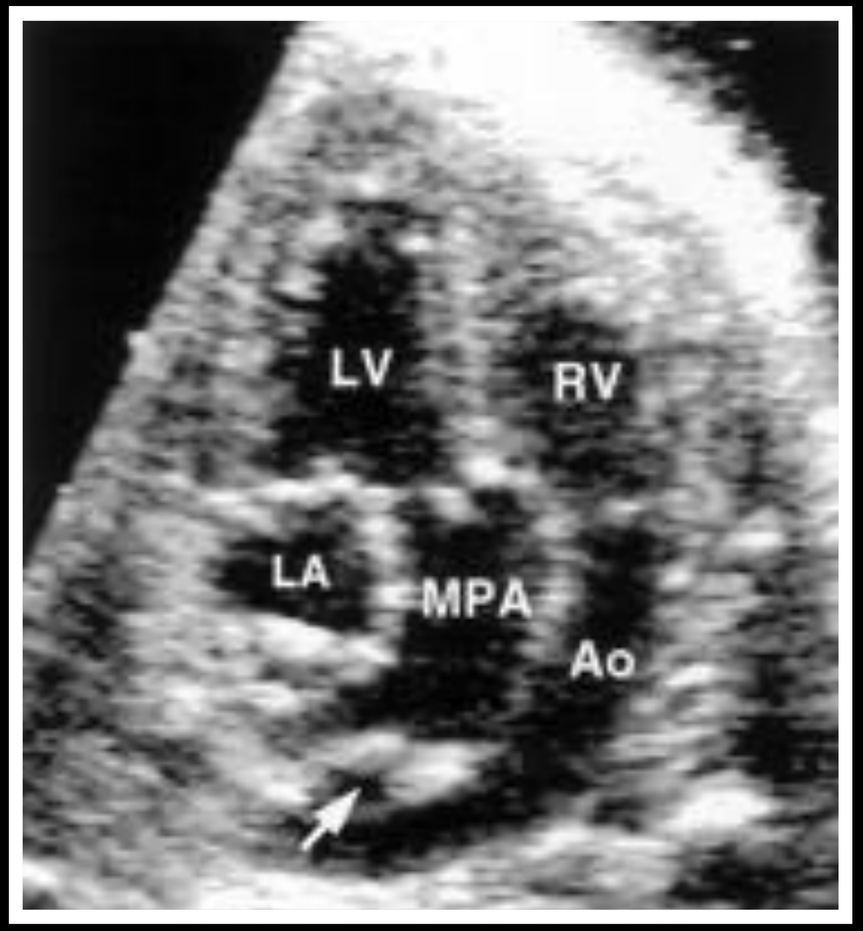
J Thorac Cardiovasc Surg 2012;143:1264-70

Conséquences développementales de la répartition du débit sanguin foetal combiné

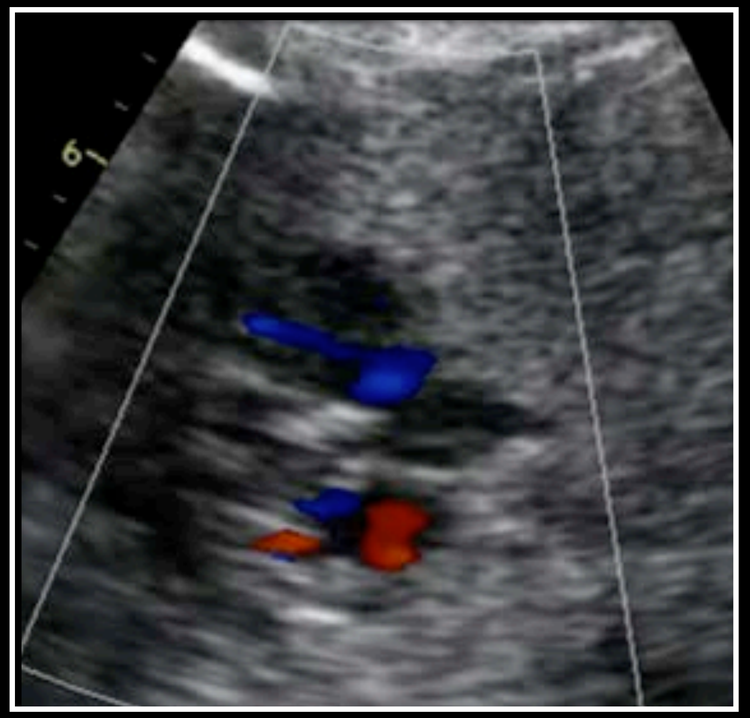


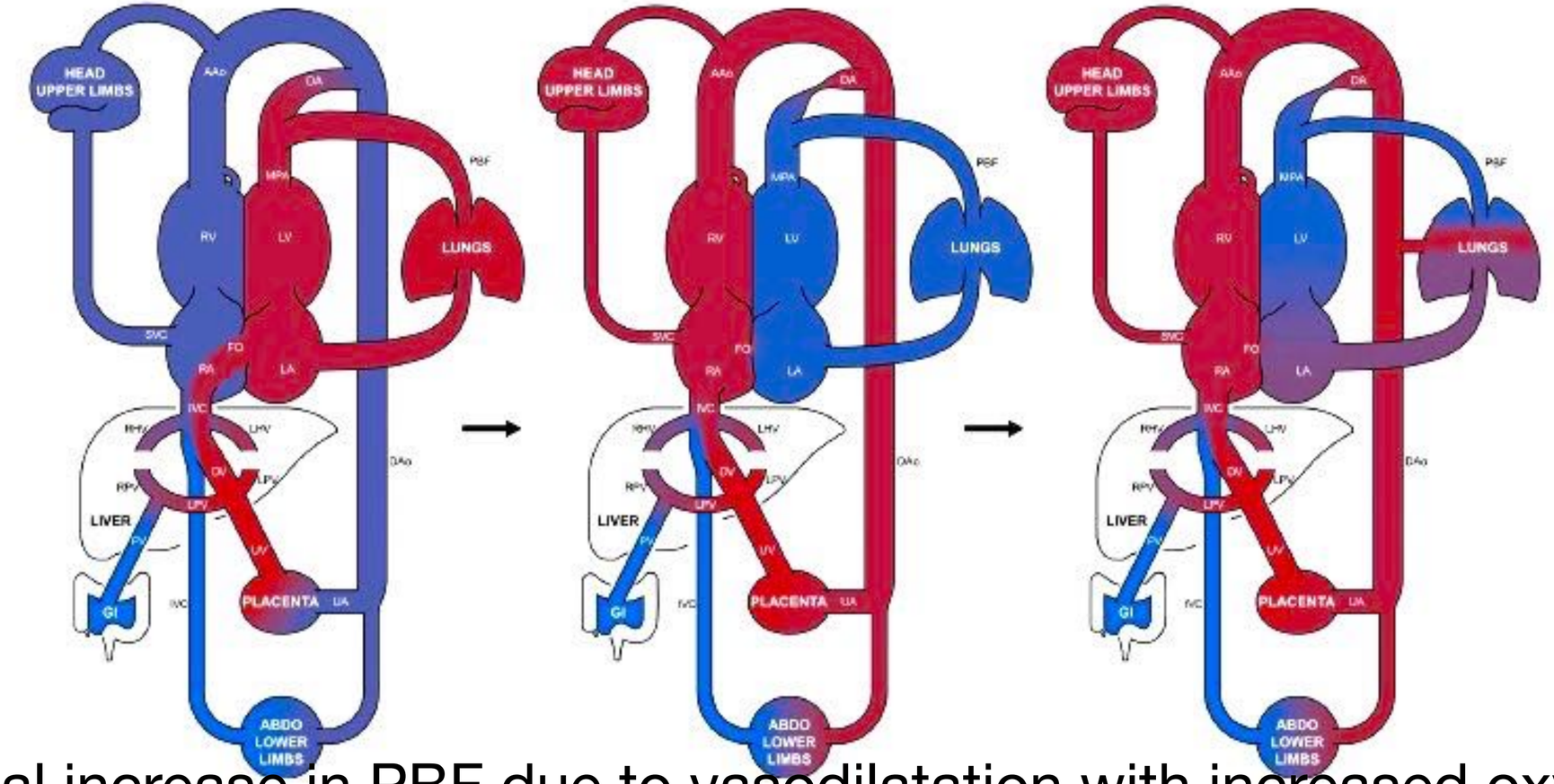
Rudolph A. Ped Res 2007;61:375-80
Prsa M et al. Circ Cardiovas Imaging 2014;7:663-70


LV
LA
RV
RA


LV
RV
LA
MPA
Ao

Conséquences développementales des anomalies de l'oxygénation foetale
Restriction du foramen ovale chez un foetus ayant une TGV





Initial increase in PBF due to vasodilatation with increased oxygen

Increased pulmonary venous return

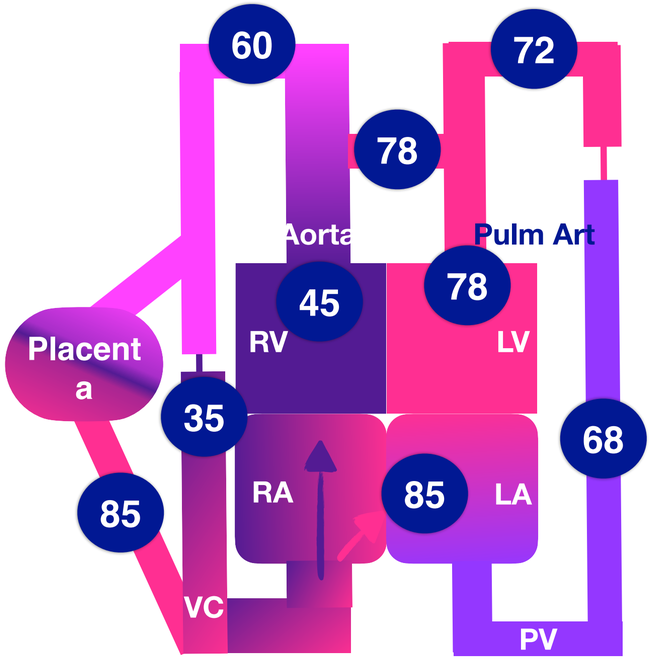
Reduced size of the FO

Ductal constriction due to oxygen

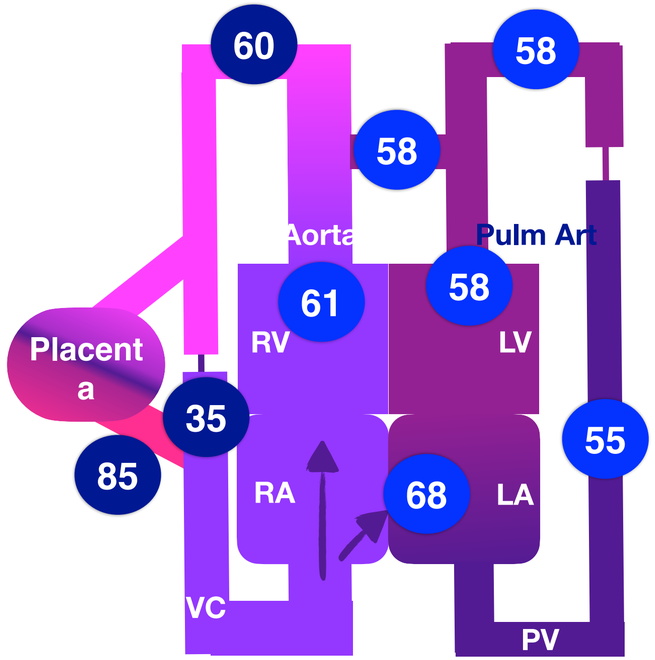
Isolation of Pulmonary circulation

Increased PVR

Development of aorta-pulmonary collaterals



TGA fetus



Closure of the ductus venous

Rudolph A. Ped Res 2007;61:375-80
Prsa M et al. Circ Cardiovas Imaging 2014;7:663-70

Physiologie cardiaque appliquée aux cardiopathies congénitales

Partie 2

Approche de la fonction myocardique

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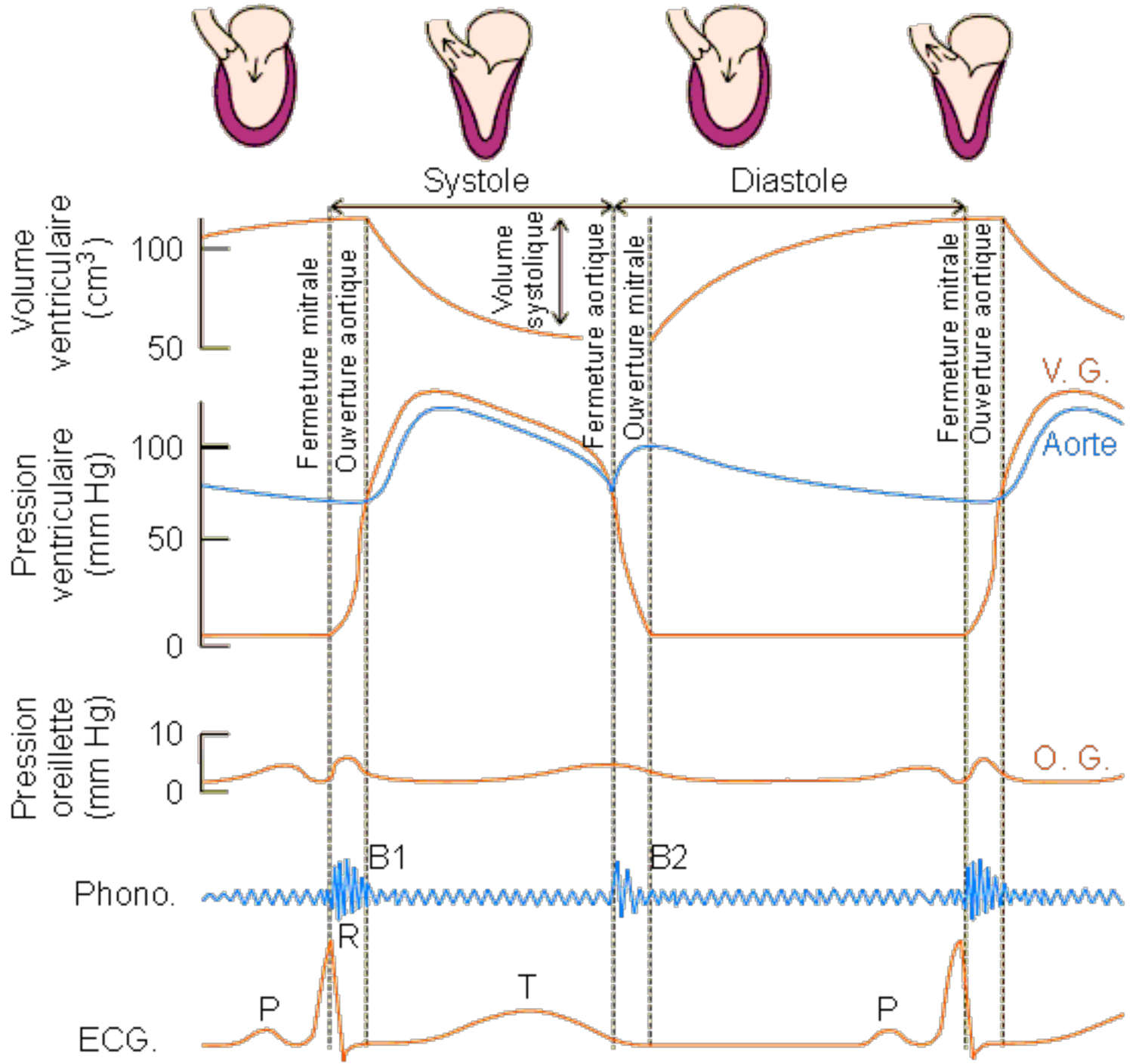
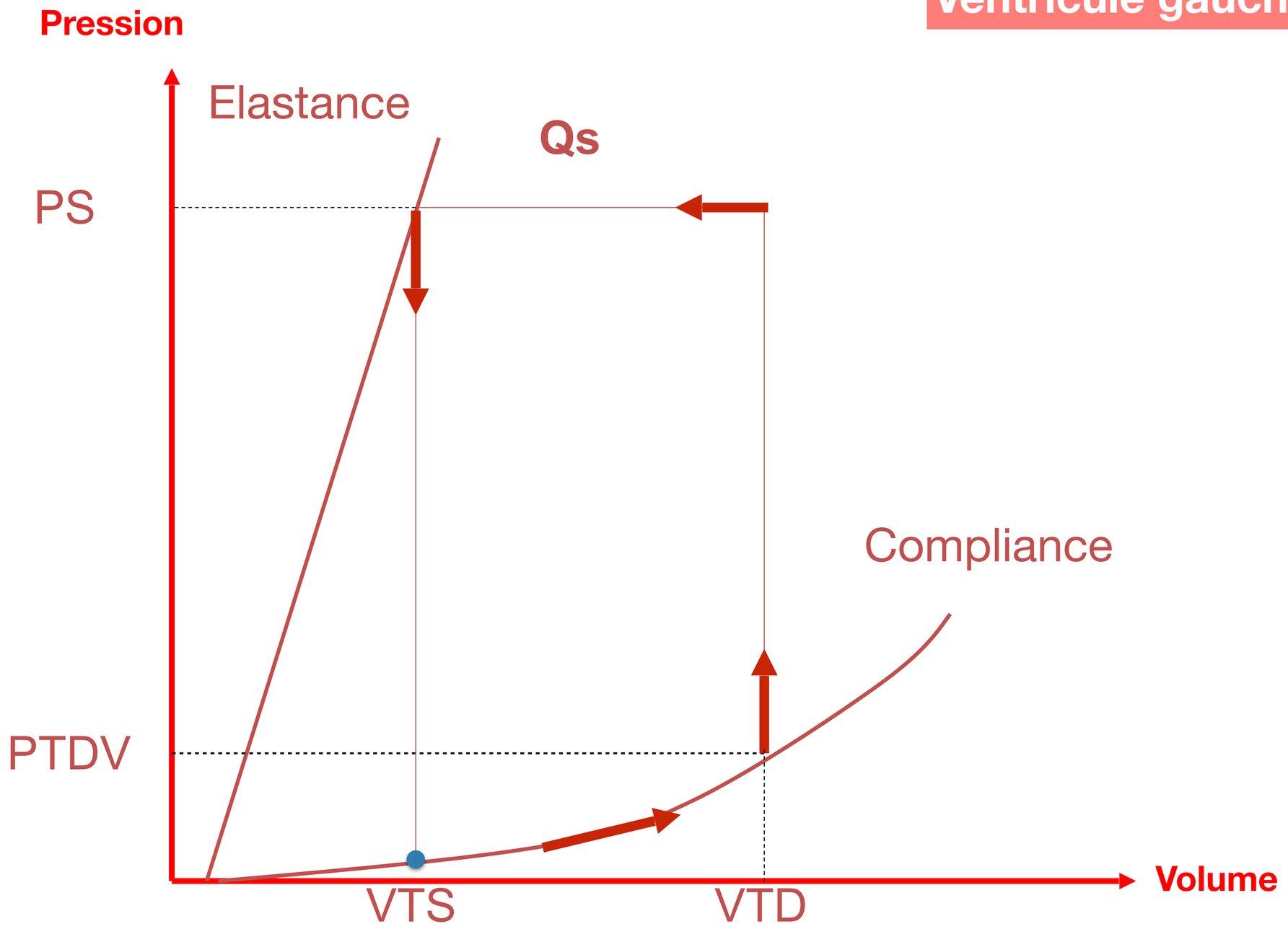
Centre de Référence Maladies Rares
Malformations Cardiaques Congénitales Complexes-M3C
Centre de Référence Maladies Rares
Maladies Cardiaques Héréditaires- CARDIOGEN

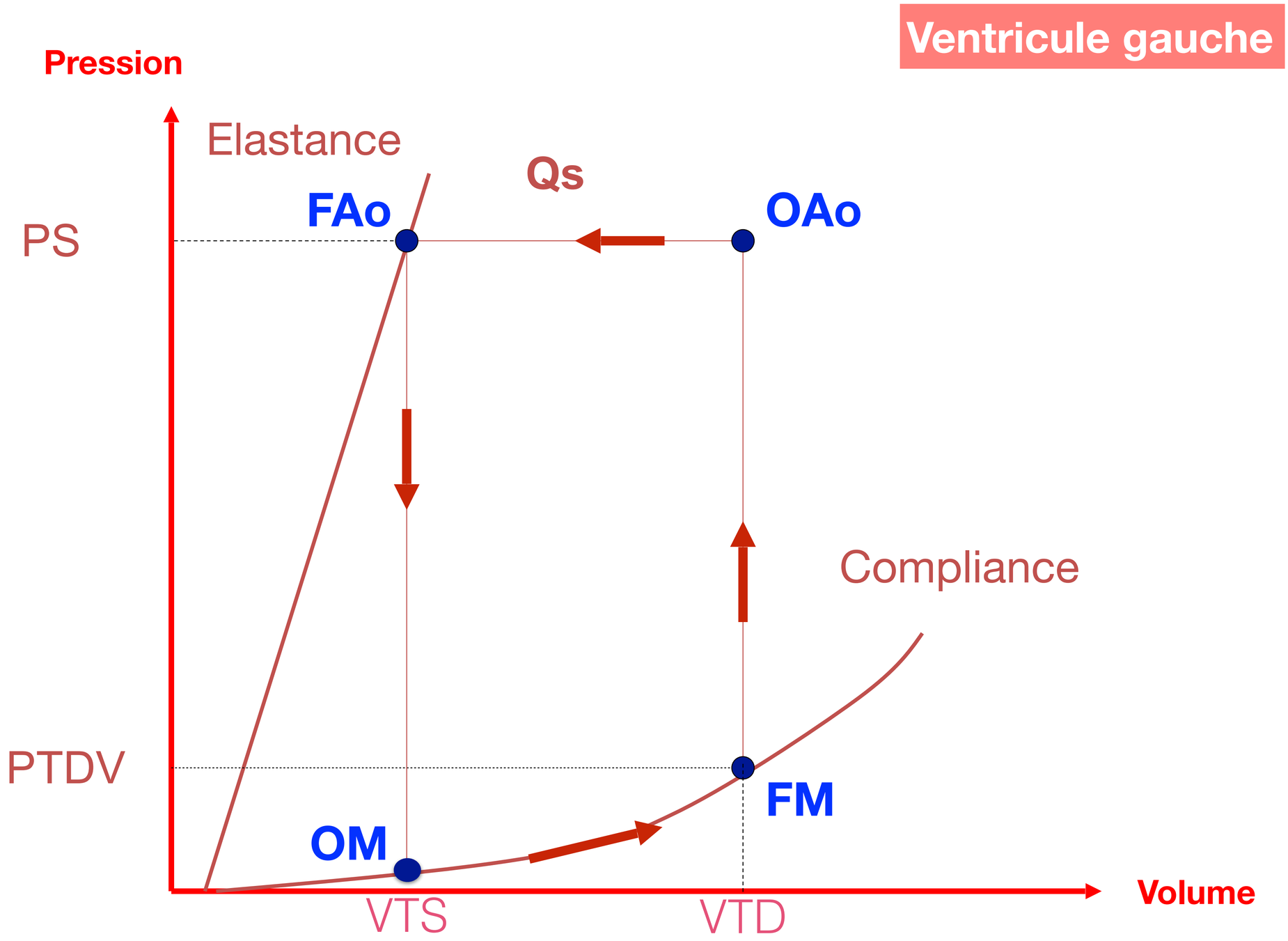

Systole
Diastole
Volume ventriculaire (cm3)
100
50
Volume systolique
Fermeture mitrale
Ouverture aortique
Fermeture aortique
Ouverture mitrale
Fermeture mitrale
Ouverture aortique
Pression ventriculaire (mm Hg)
100
50
0
V. G.
Aorte
Pression oreillette (mm Hg)
10
0
O. G.
B1
B2
Phono.
R
T
P
P
ECG.

Approche de la fonction myocardique par les courbes pression-volume

Ventricule gauche

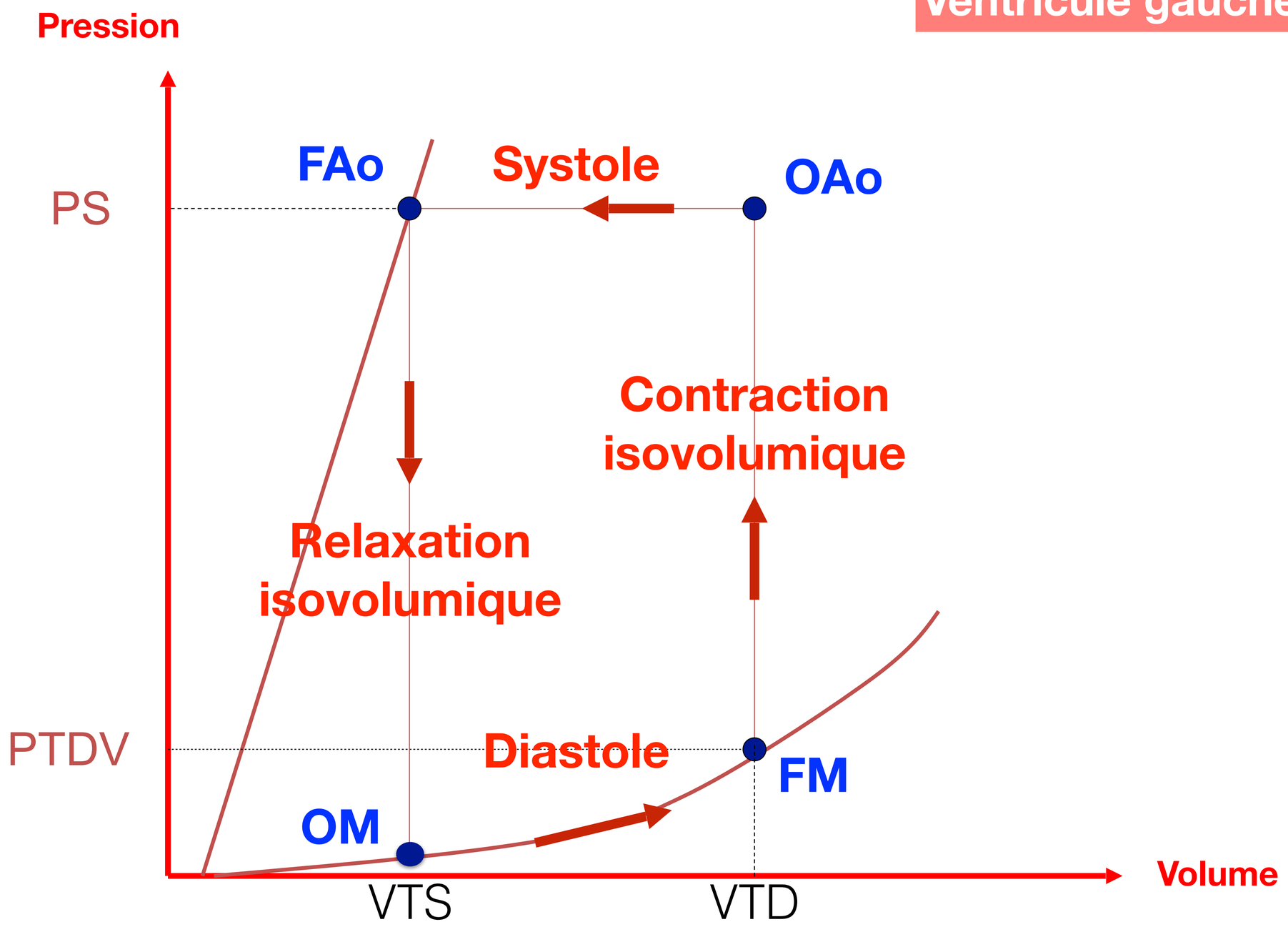


Approche de la fonction myocardique par les courbes pression-volume



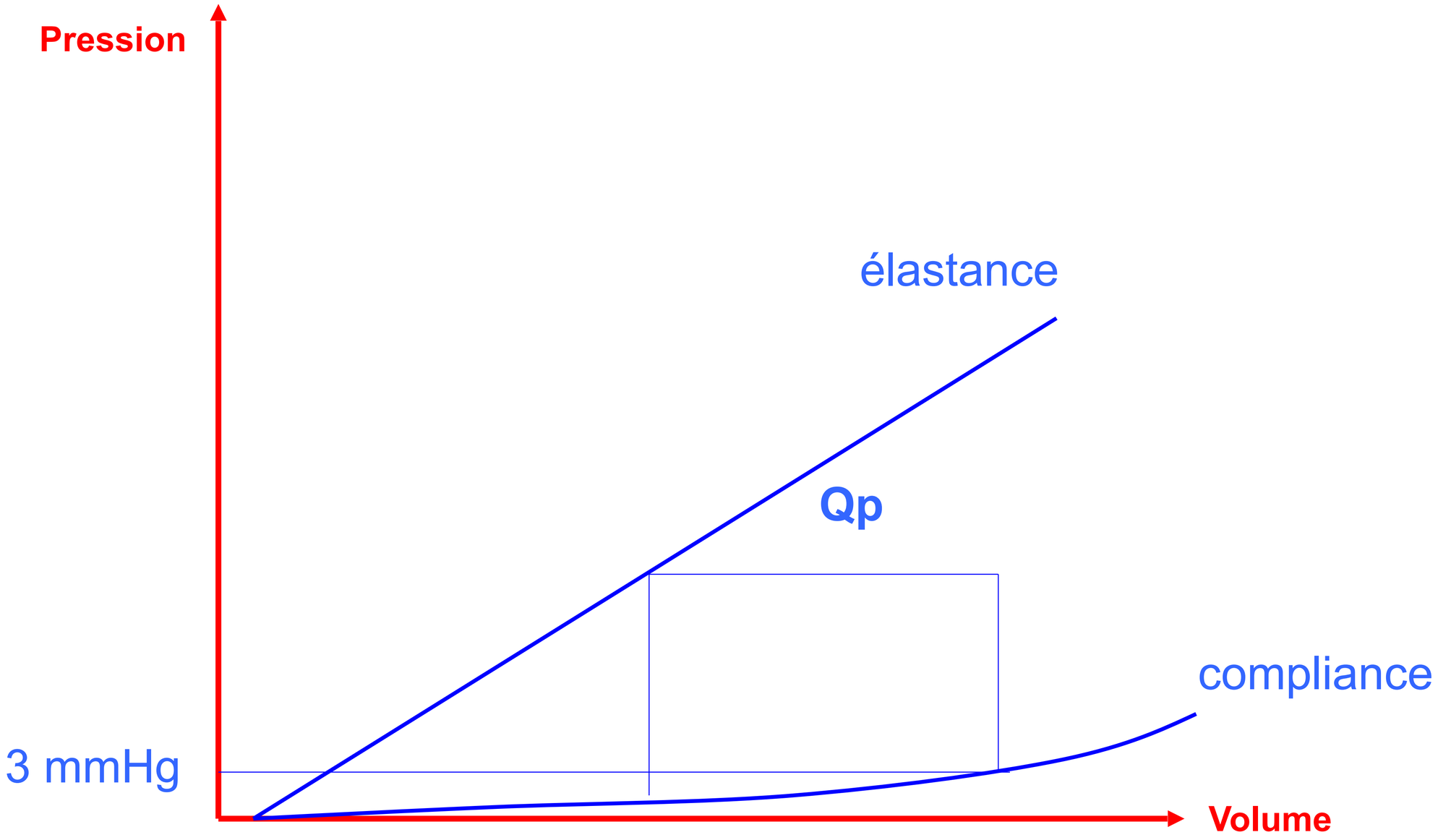
Approche de la fonction myocardique par les courbes pression-volume

Ventricule gauche

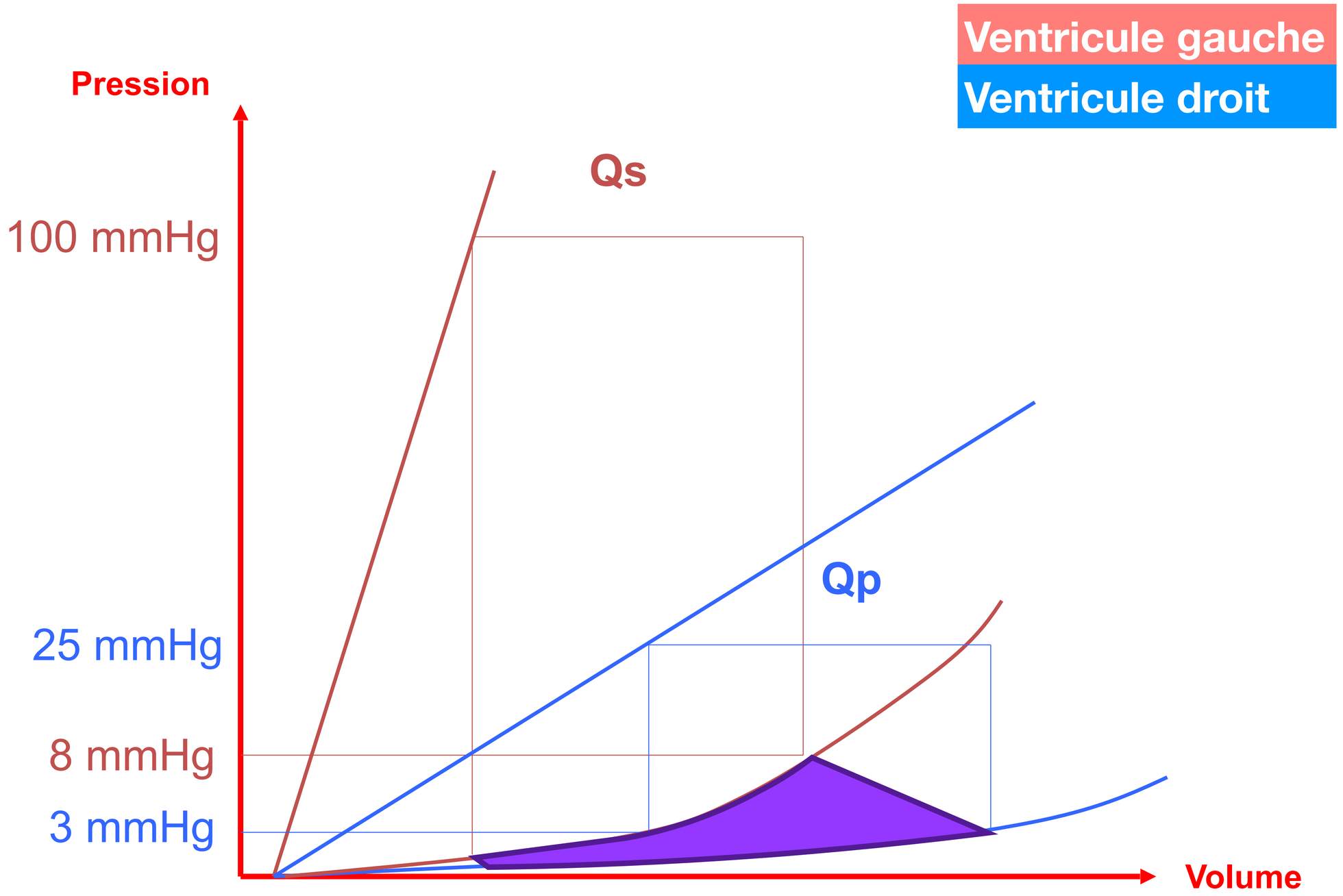


Approche de la fonction myocardique par les courbes pression-volume

Ventricule droit

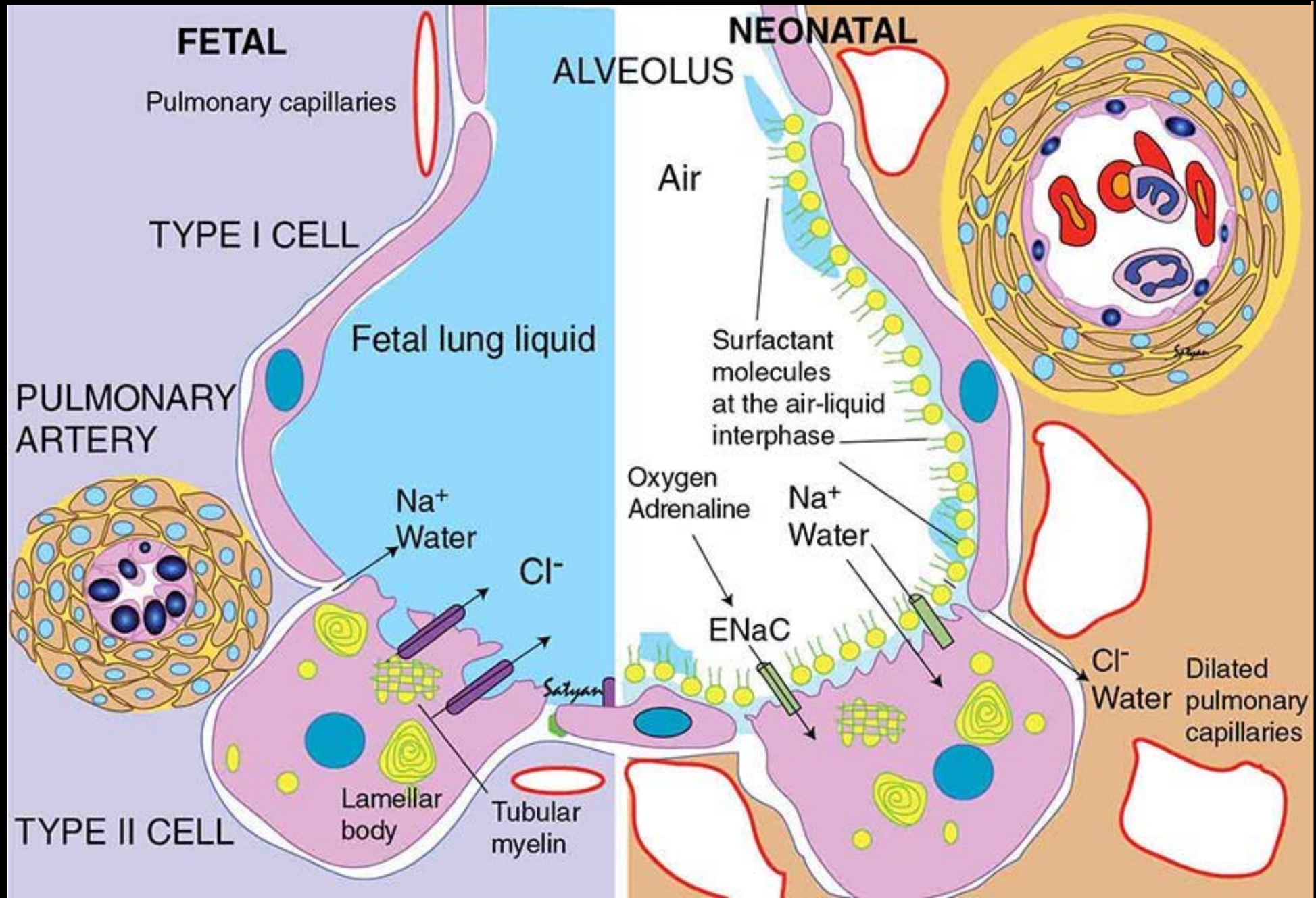


Approche de la fonction myocardique par les courbes pression-volume

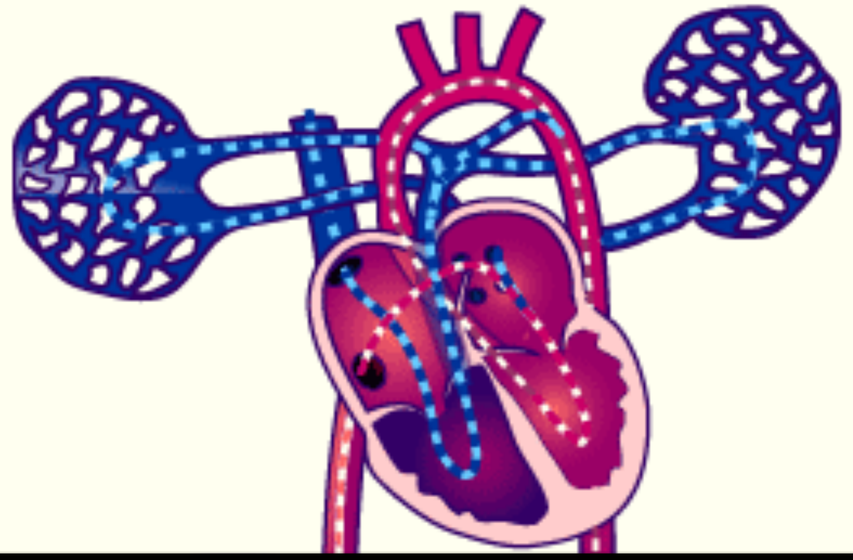


Transition foetus/nouveau-né

Transition foetus/nouveau-né

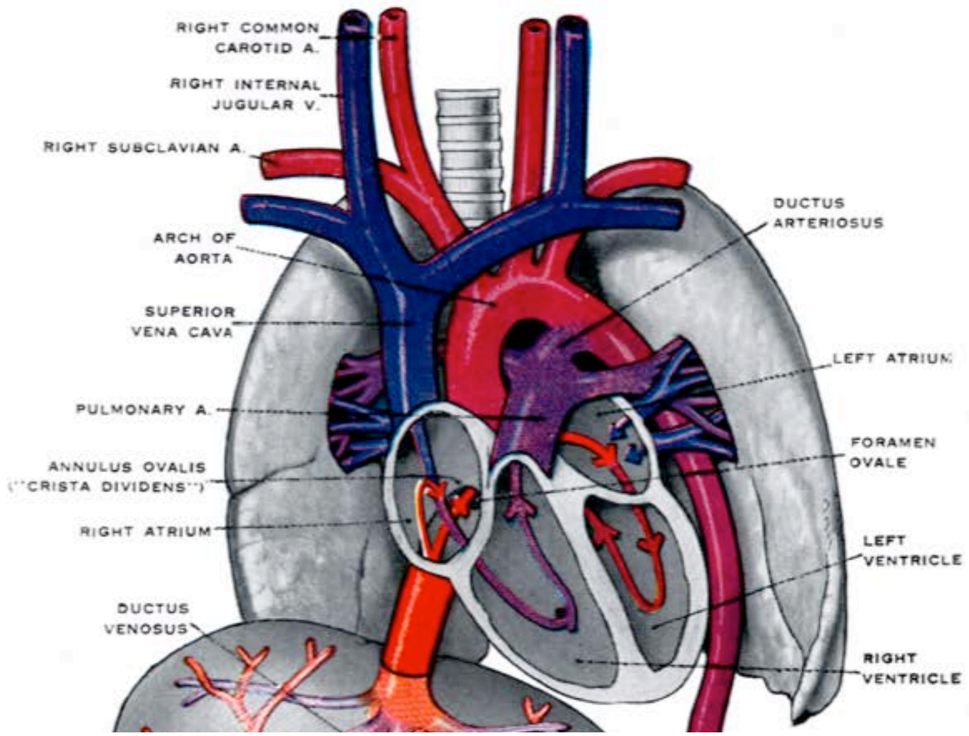


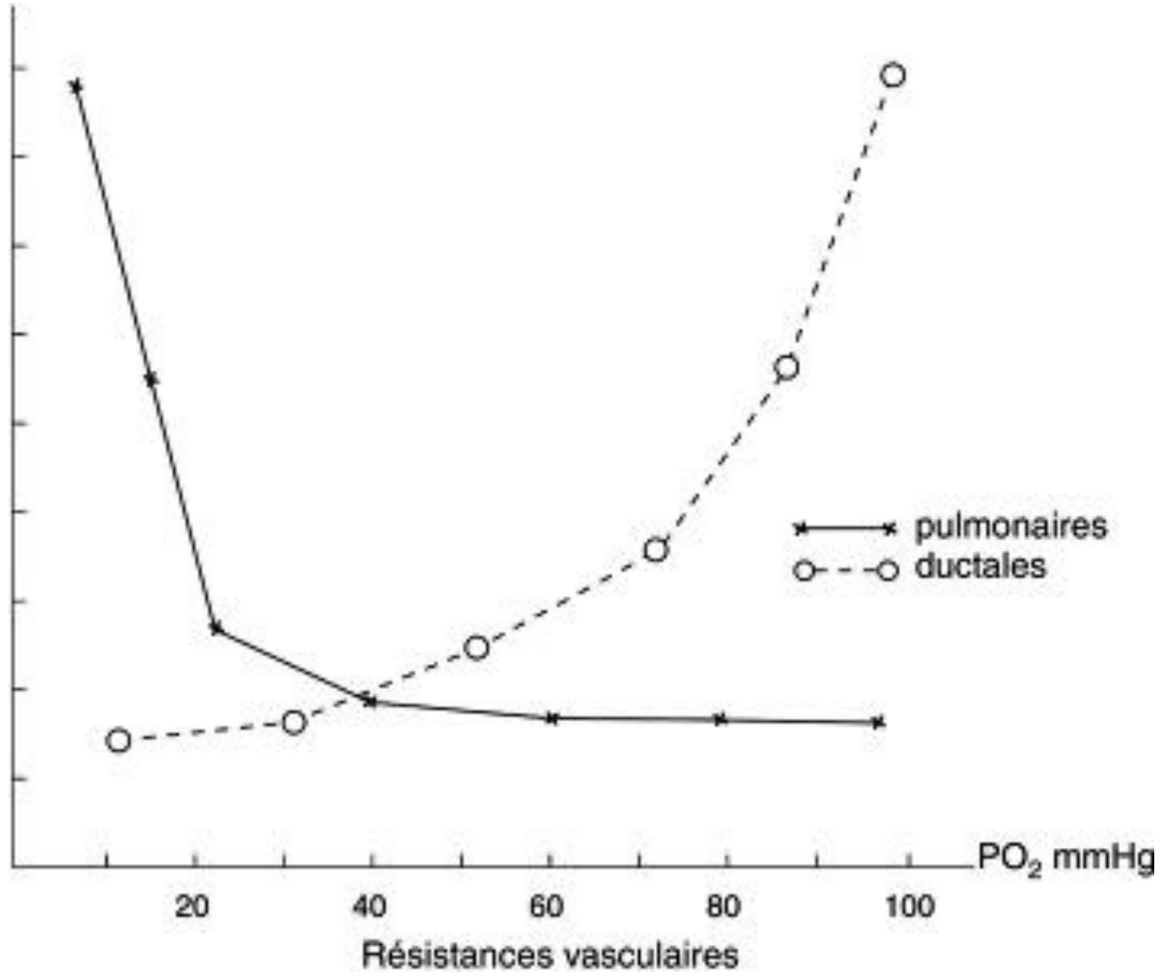
before birth



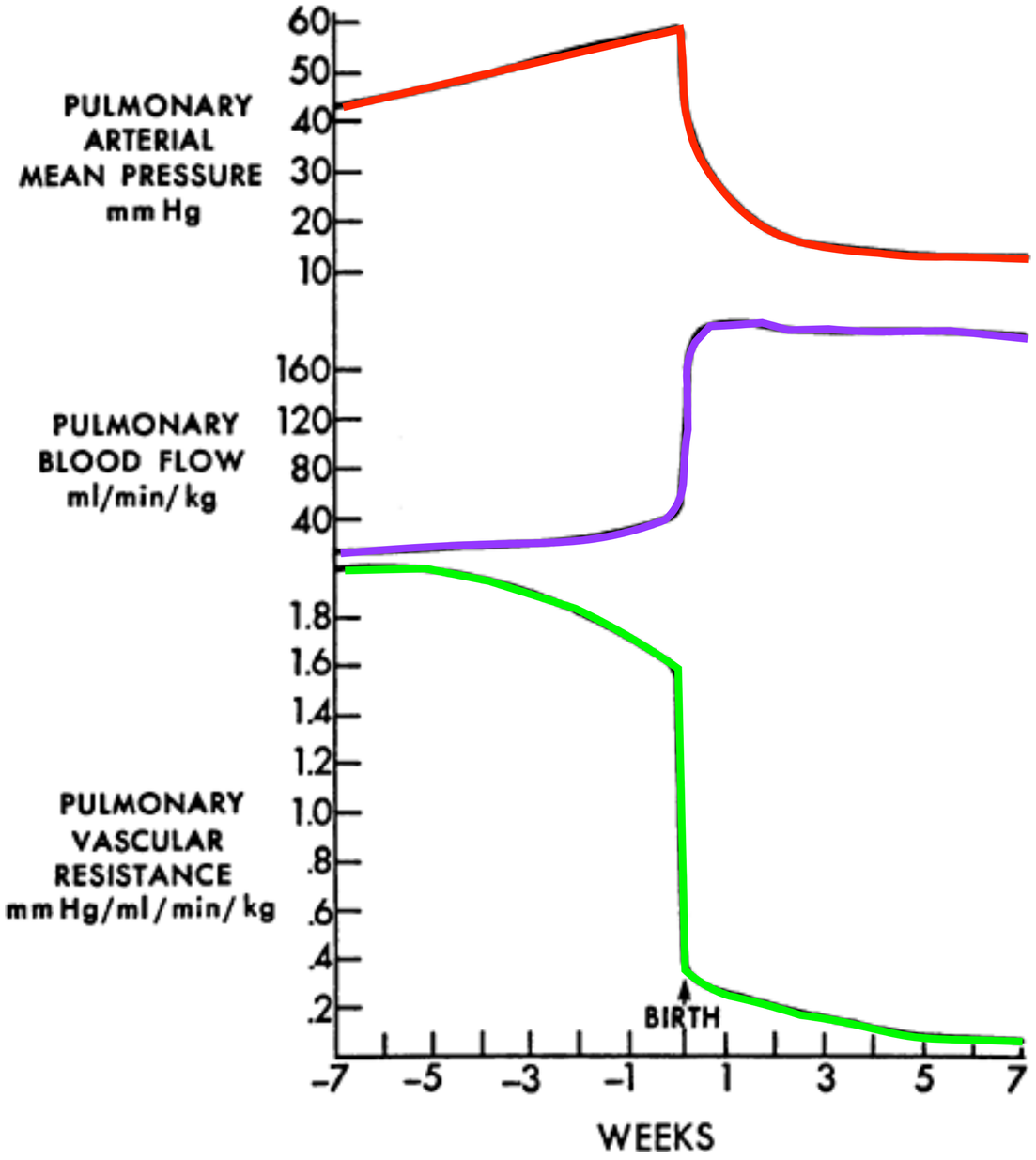
after birth

Evolution des résistances vasculaires pulmonaires à la naissance

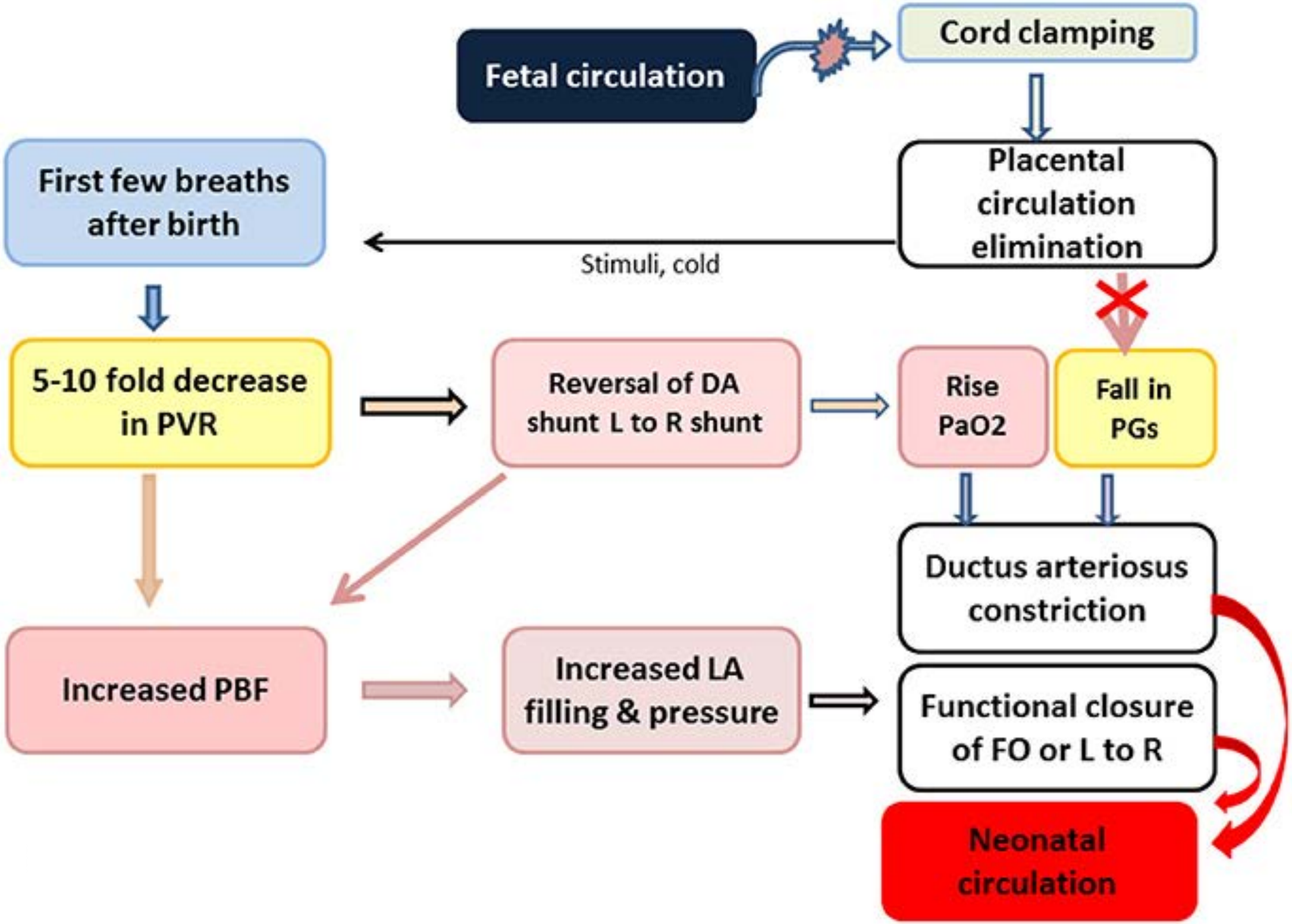




Transition foetus/nouveau-né



Transition foetus/nouveau-né



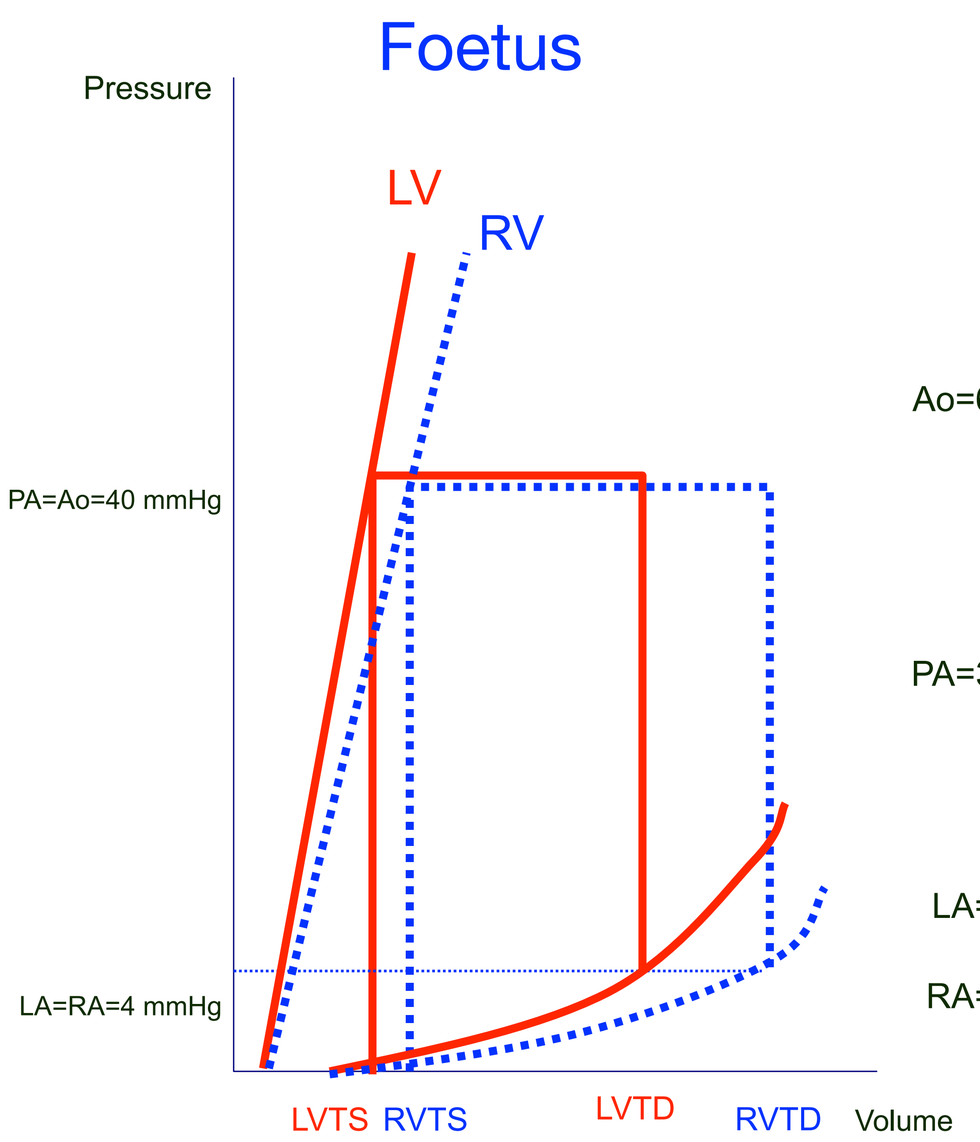
Adaptation Post-natale

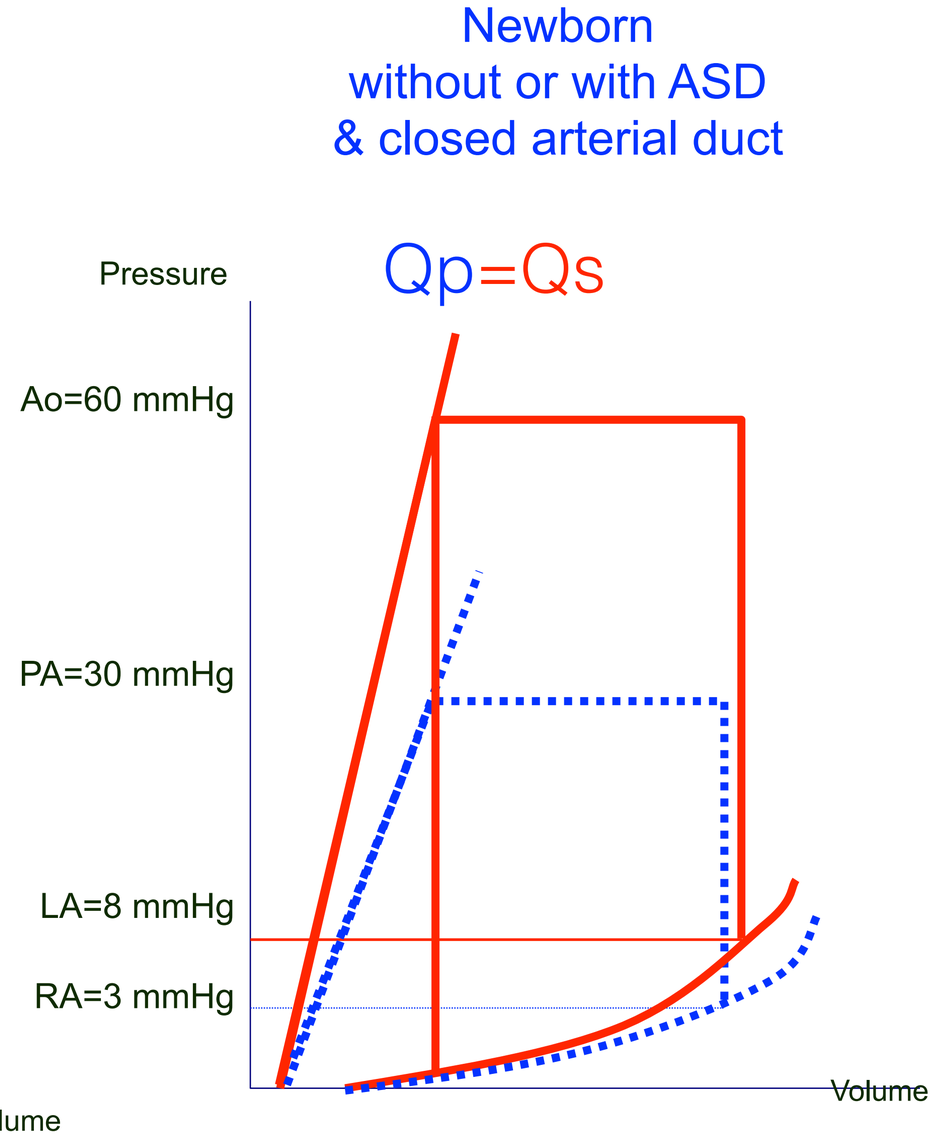
- Disparition du placenta et Vasodilatation des artérioles pulmonaires
  - Le rapport RVP/RVS passe de 10 à 1/3 en quelques minutes
- Fermeture du canal artériel et de la CIA
  - Cœur en série avec Pressions Gauches > Droites en systole et diastole

Il n ’y a pas d ’HTAP physiologique du nouveau-né

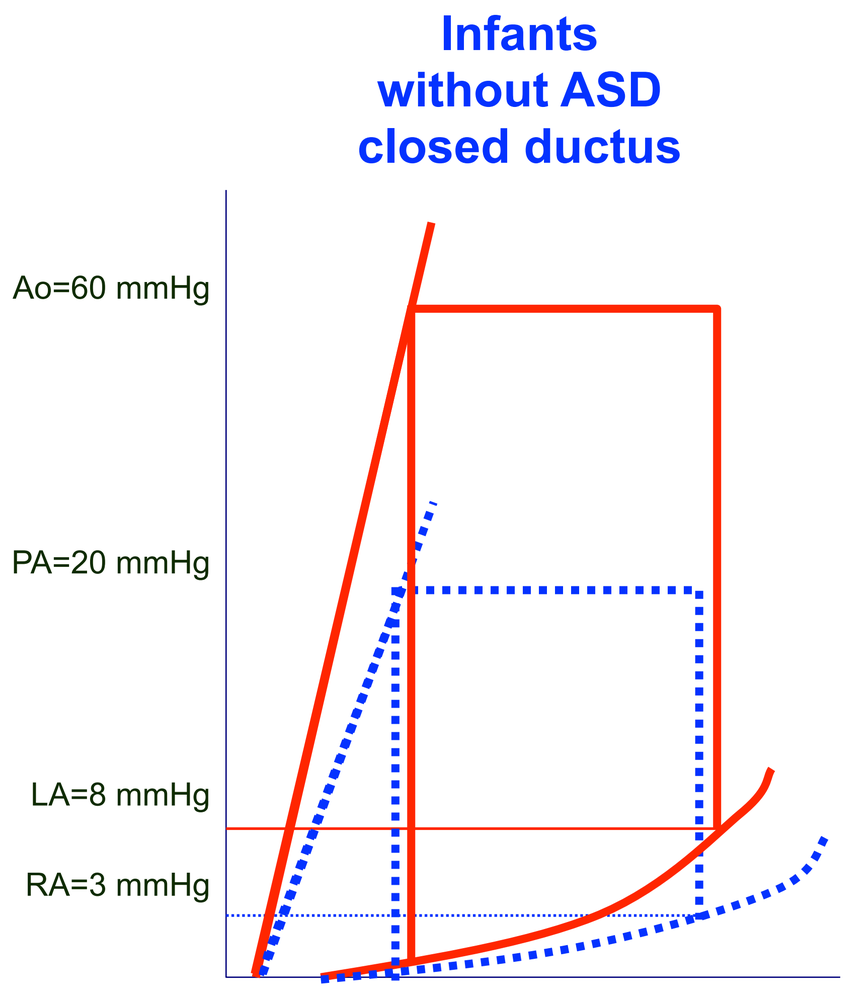
- Augmentation de la VO2 et du Débit Systémique (Qs)
  - Le travail du VG quadruple la première semaine; il s’hypertrophie et se spécialise dans la contractilité
  - Le VD fonctionne à basse pression, se démuscle et se spécialise dans la compliance

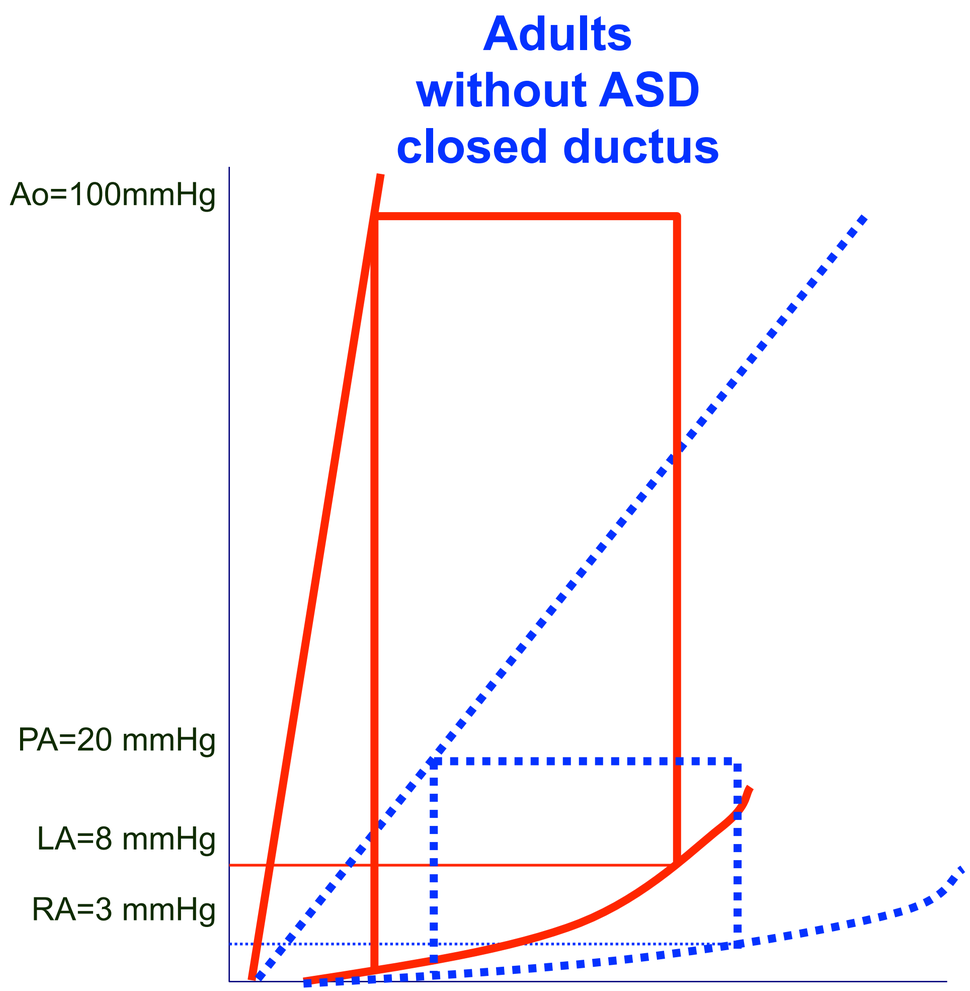
Approche de la fonction myocardique par les courbes pression-volume



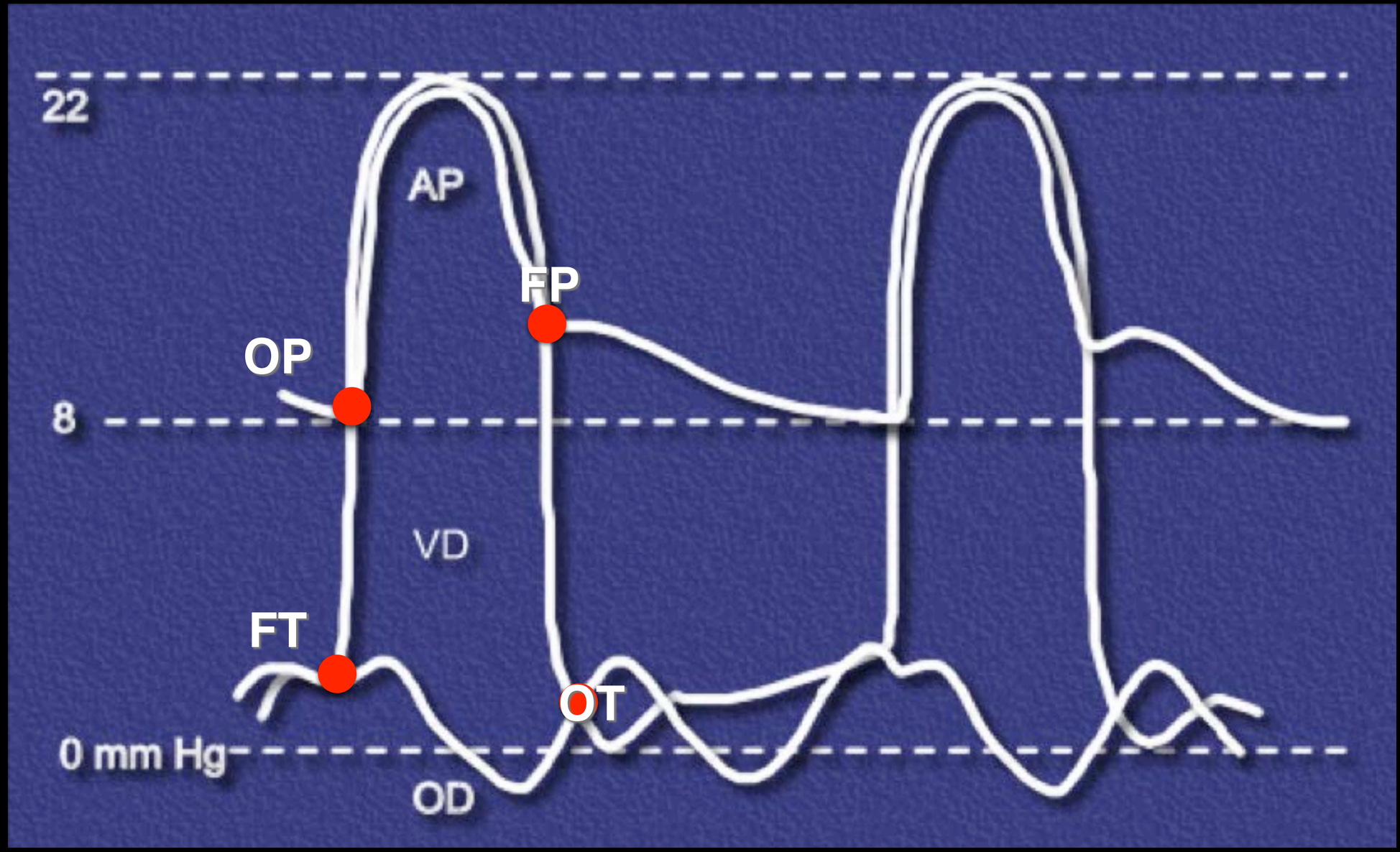


Approche de la fonction myocardique par les courbes pression-volume

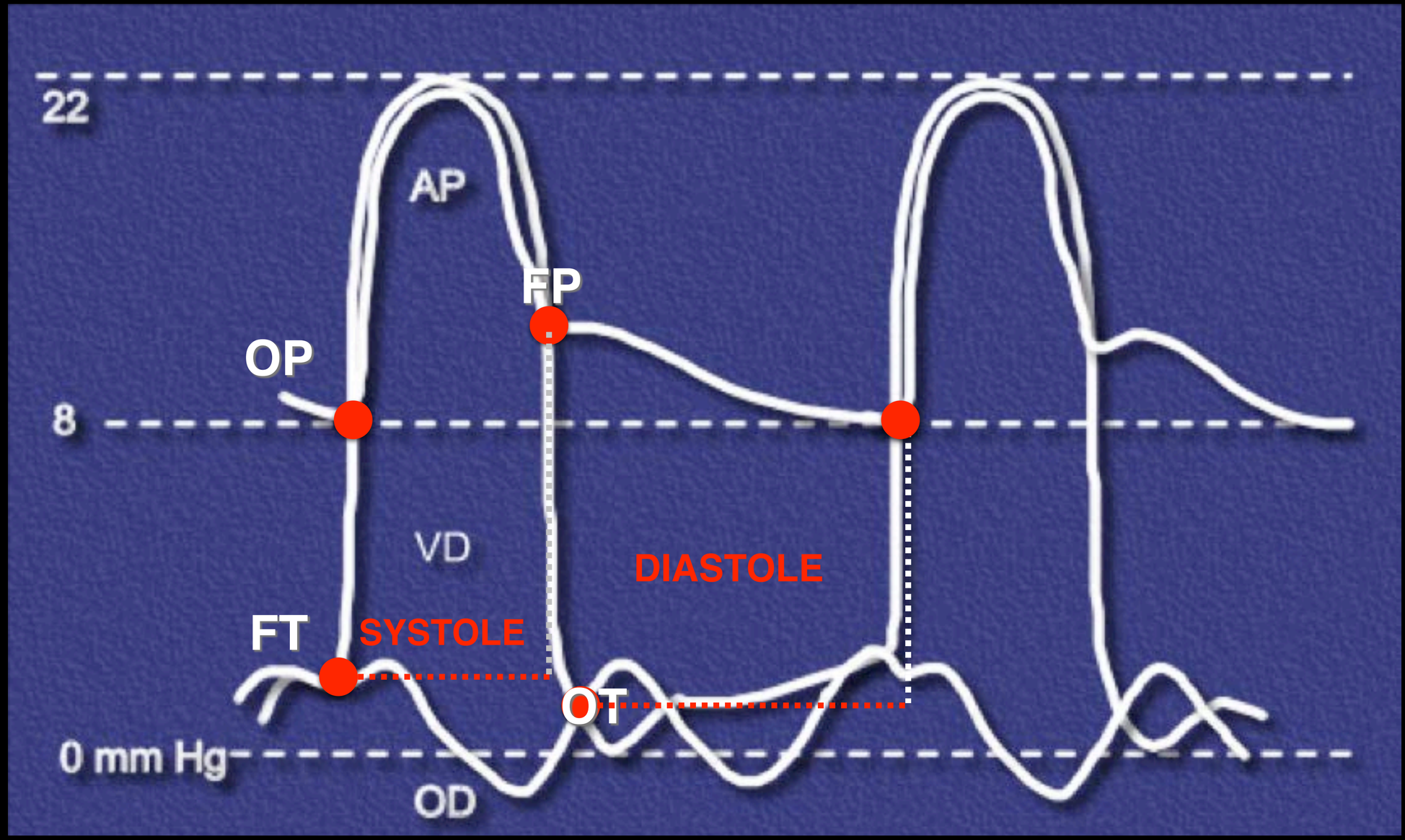




Courbes de pression en fonction du temps



Courbes de pression en fonction du temps



Courbes de pression en fonction du temps

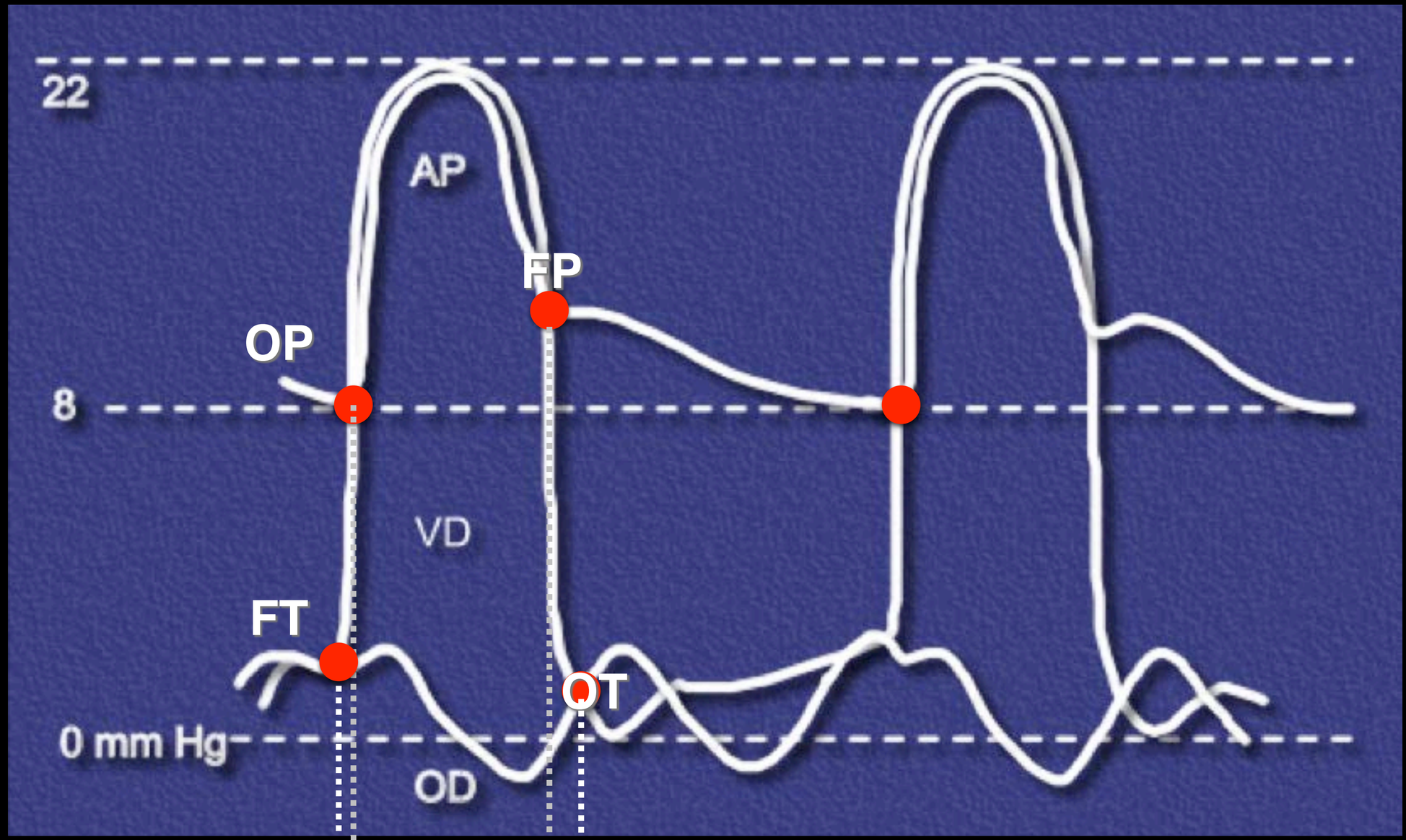
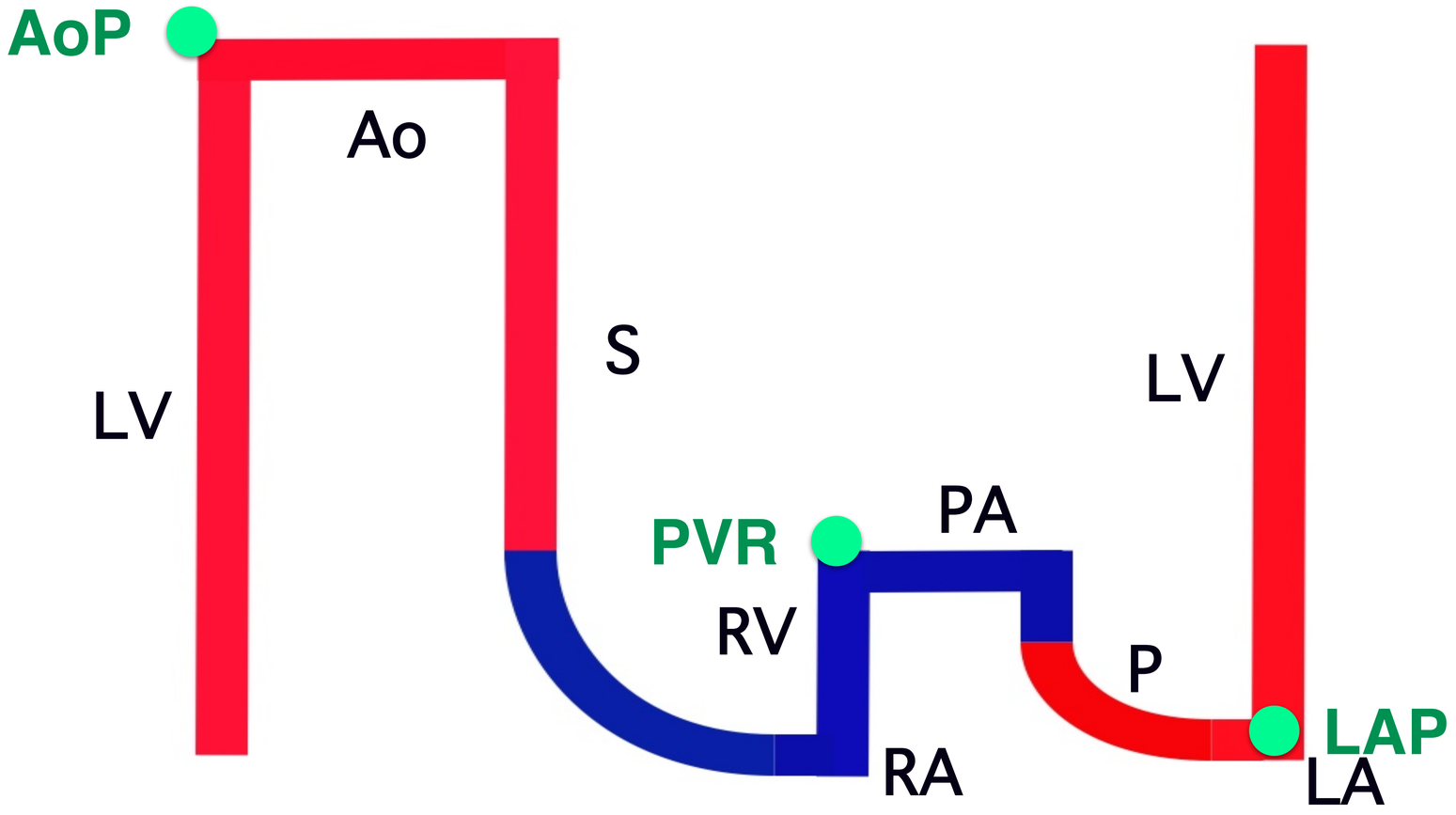


Schéma hémodynamique du Cœur en Série



Physiologie cardiaque appliquée aux cardiopathies congénitales

Partie 3

Physiologie des shunts

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Malformations Cardiaques Congénitales Complexes-M3C
Centre de Référence Maladies Rares
Maladies Cardiaques Héréditaires- CARDIOGEN

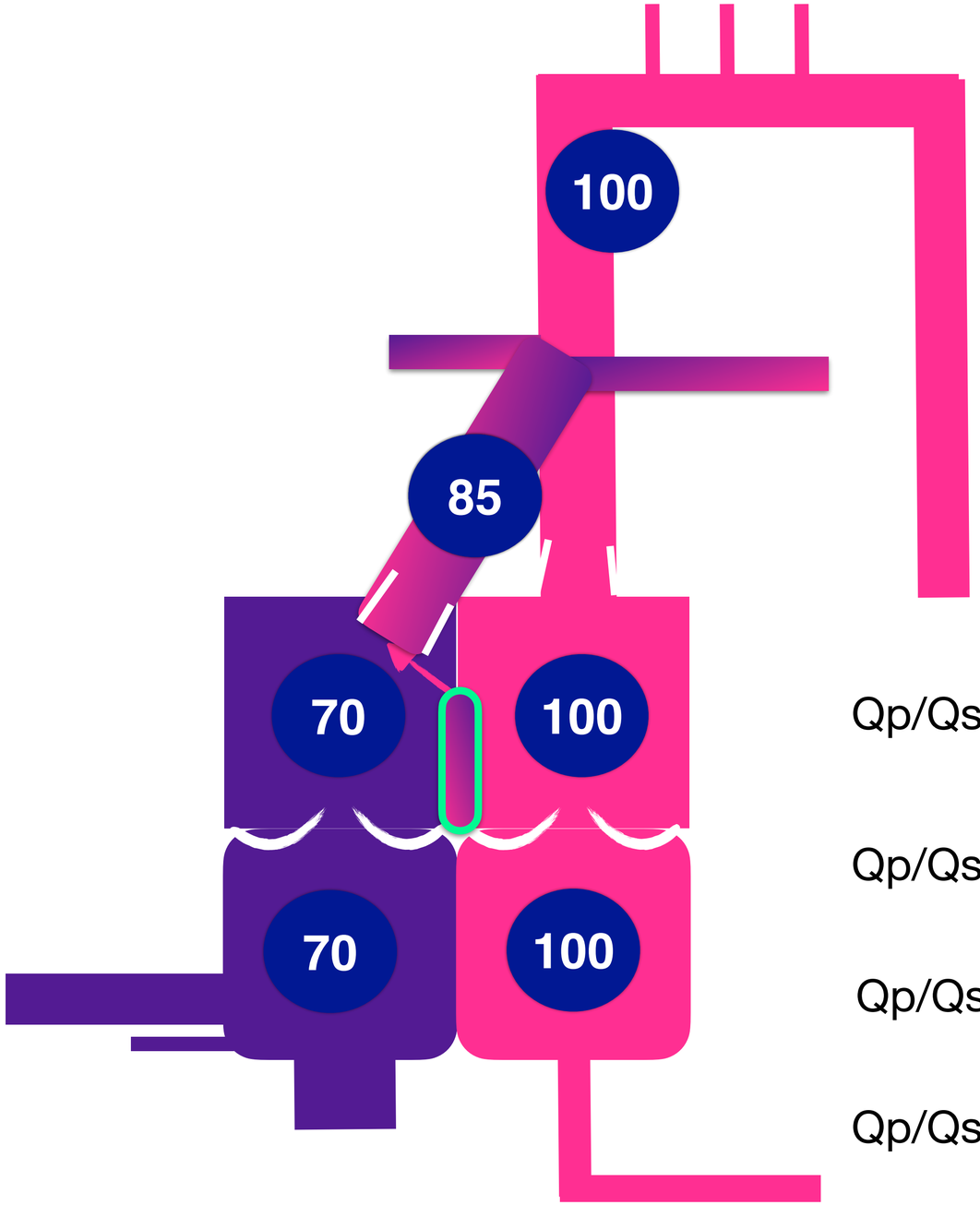
Qp/Qs
Le principe de Fick

- Q= VES x FC
- VO_2=Q x DAV
- Qp/Qs=[SaAo-SaVC]/[SaVP-SaAP]
- Résistances=ΔP/Q - Loi de Poiseuille
  - Rp/Rs= ΔPp . Qs/ ΔPs . Qp
  - Rp/Rs= (ΔPp/ΔPs) . (Qs/Qp)

Q: débit
VO2: Consommation d'oxygène
FC: Fréquence cardiaque
DAV: Différence artério-veineuse
Sa: Saturation

Ao: Aorte
VC: Veine cave
VP: Veine pulmonaire
AP: Artère pulmonaire
p: pulmonaire
s: systémique

CIV



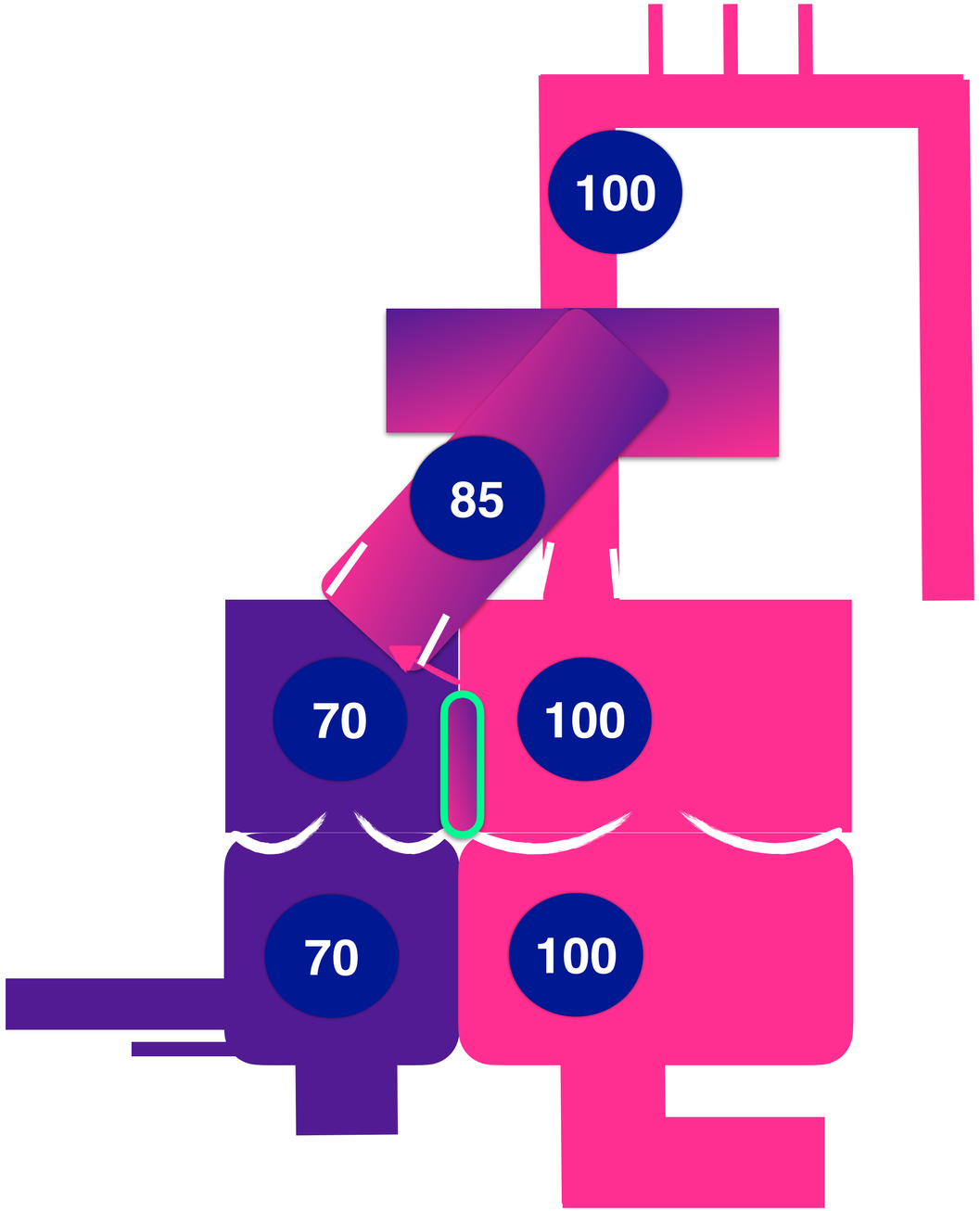
Qp/Qs=[SaAo-SaVC]/[SaVP-SaAP]

Qp/Qs=[100-70]/[100-X]

Qp/Qs=[100-70]/[100-85]

Qp/Qs=[30]/[15] = 2/1 = 2

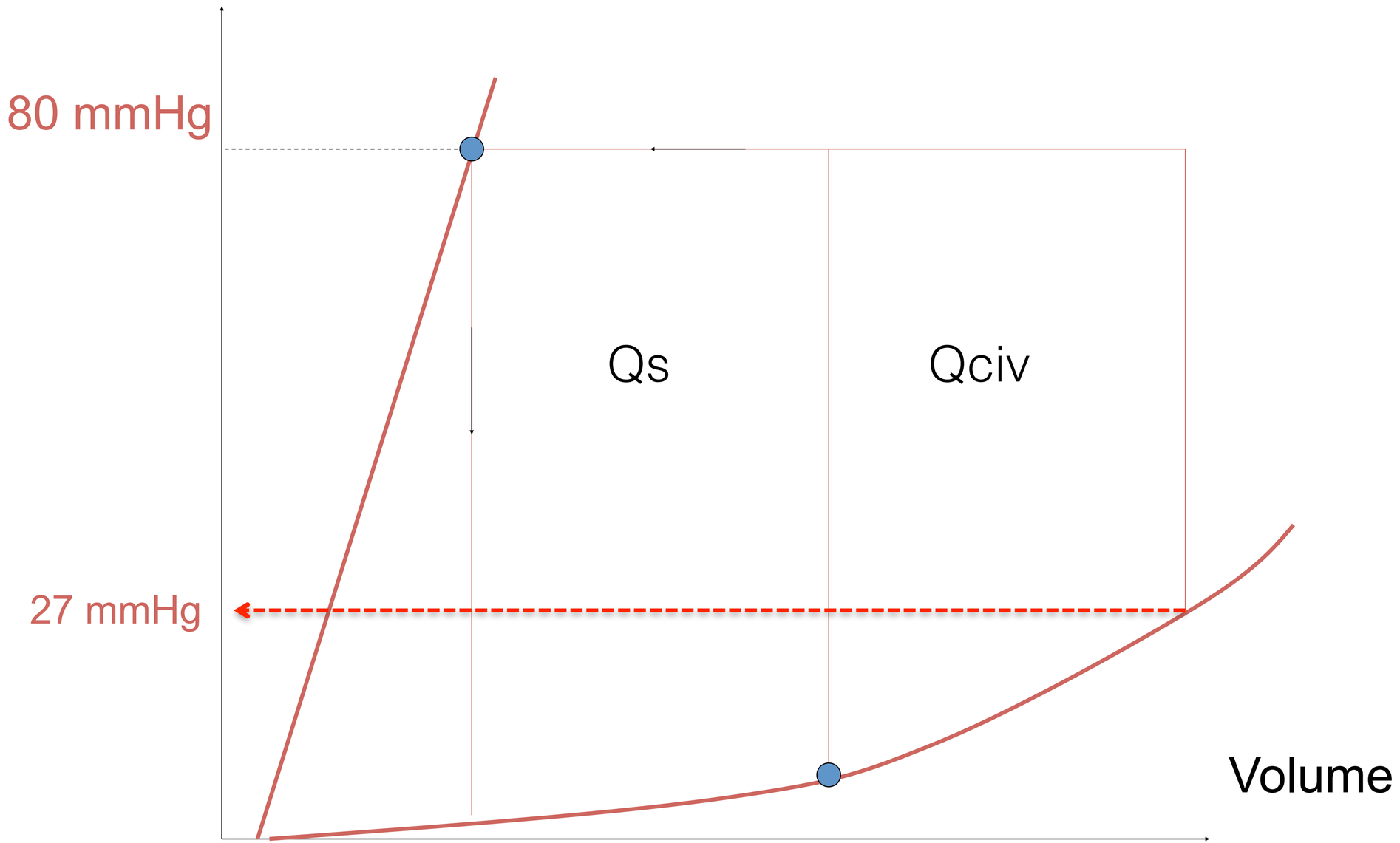
CIV



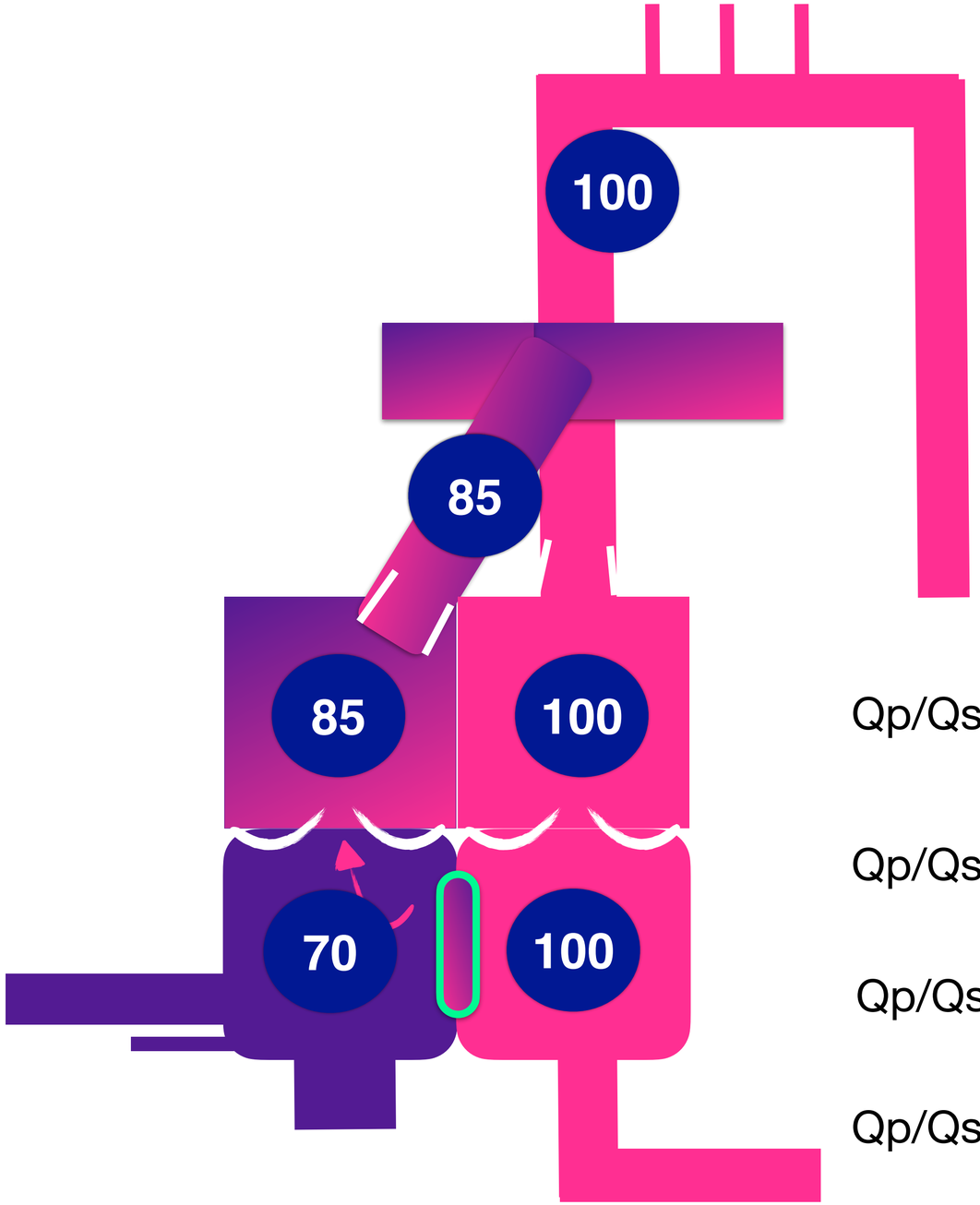
CIV

VG

Qp/Qs = 2 / VESvg= 2 VESvd



CIA



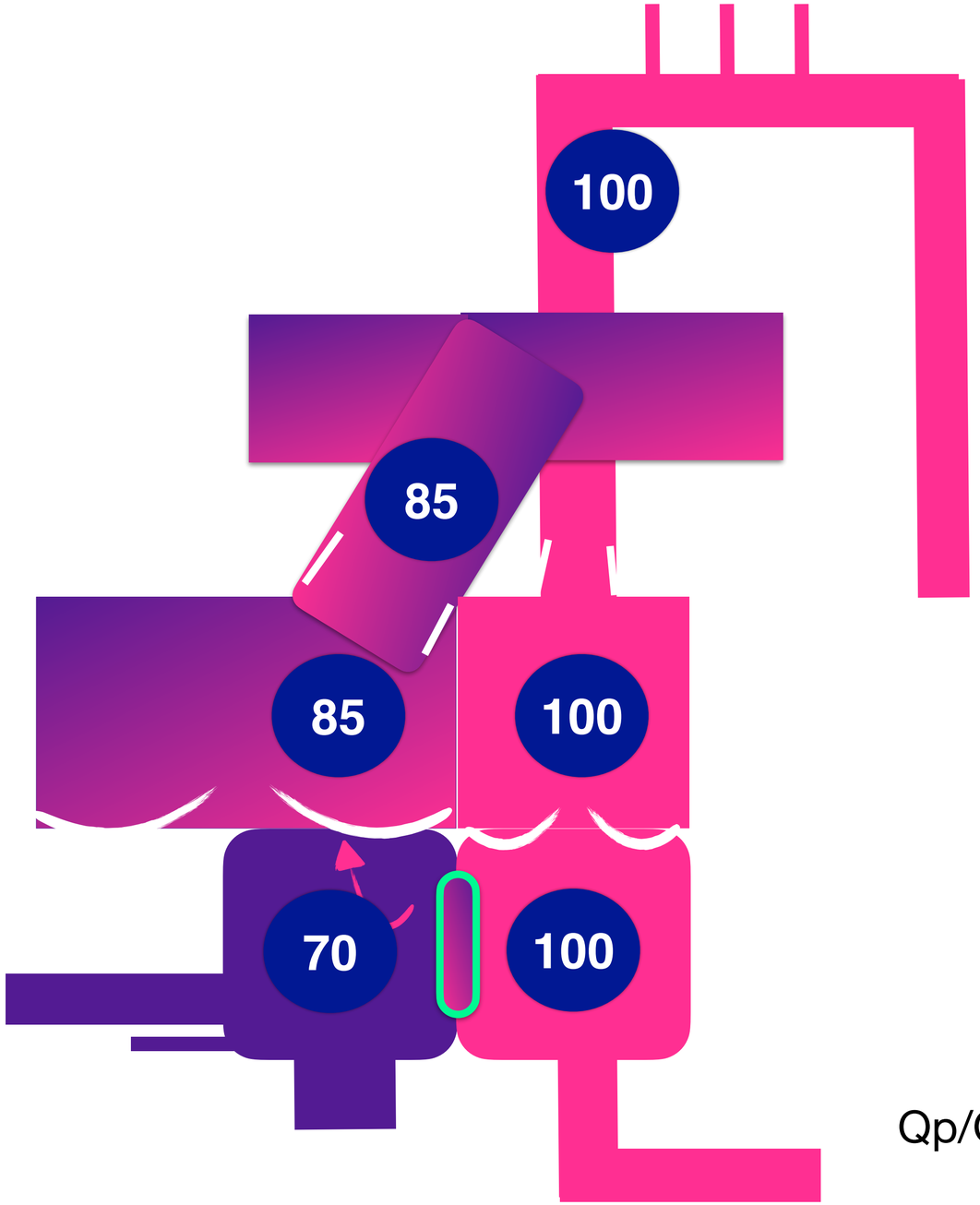
Qp/Qs=[SaAo-SaVC]/[SaVP-SaAP]

Qp/Qs=[100-70]/[100-X]

Qp/Qs=[100-70]/[100-85]

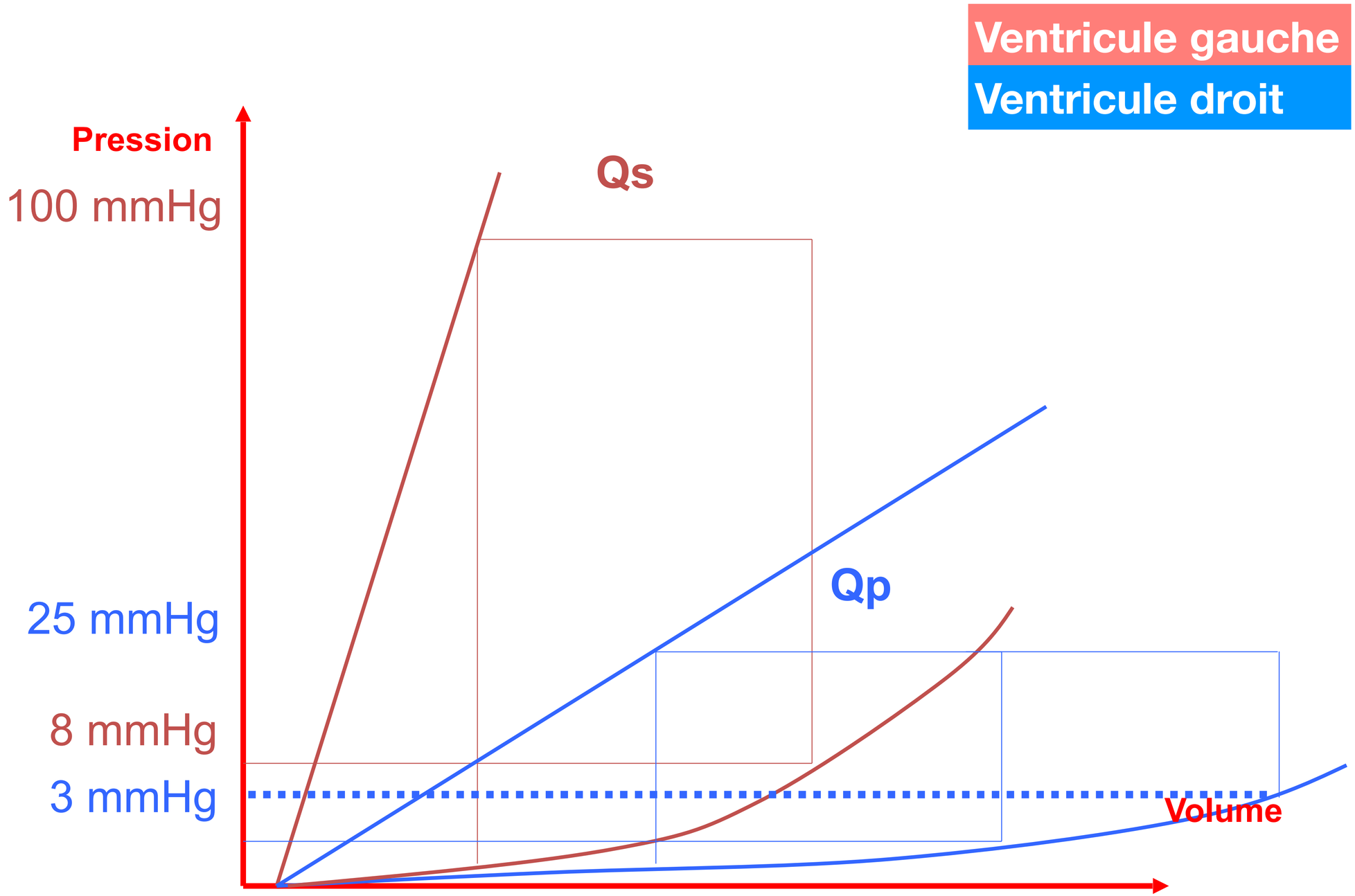
Qp/Qs=[30]/[15] = 2/1 = 2

CIA

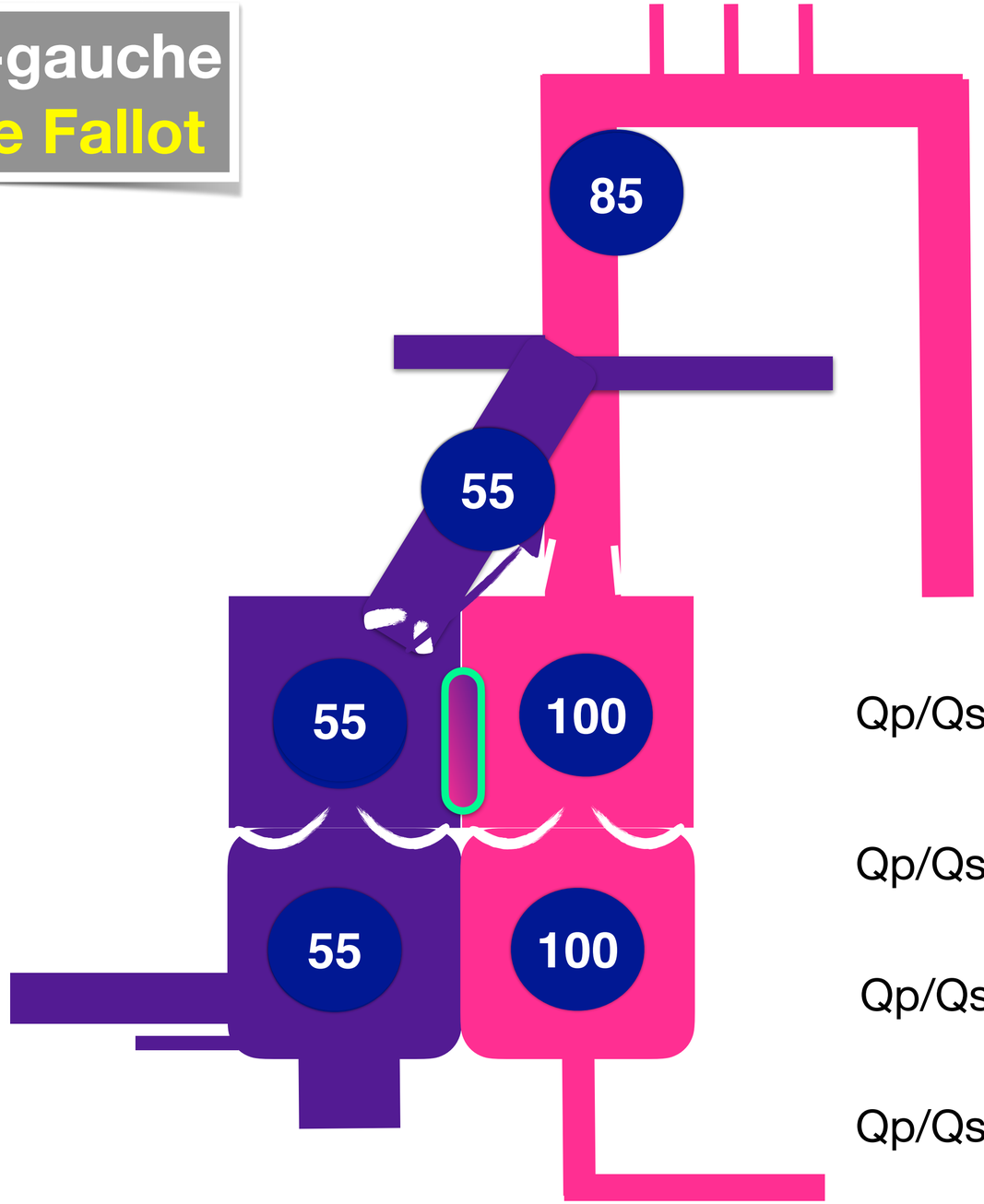


Qp/Qs=[30]/[15] = 2/1 = 2

Approche de la fonction myocardique par les courbes pression-volume



Shunt droite-gauche
Tétralogie de Fallot



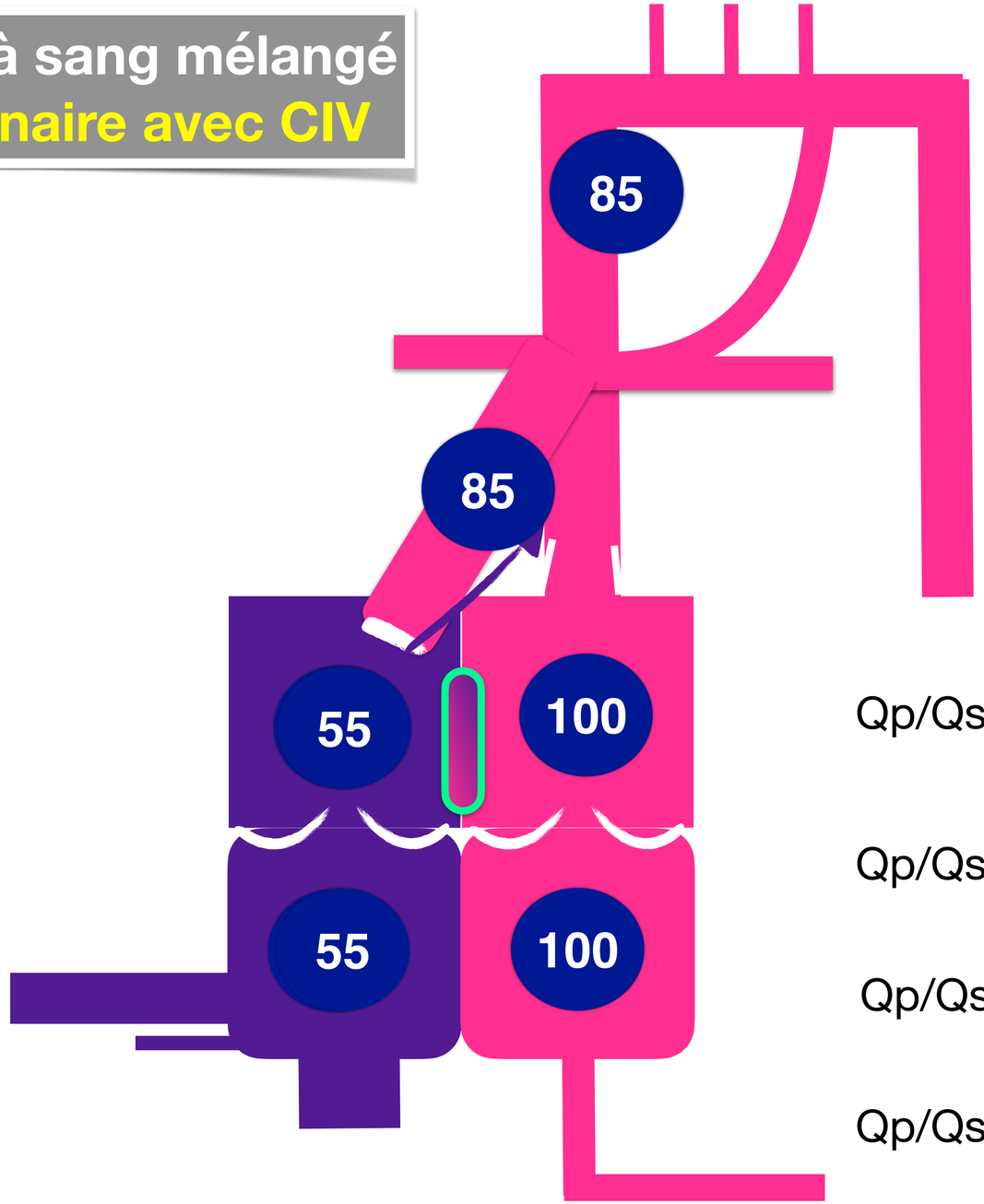
Qp/Qs=[SaAo-SaVC]/[SaVP-SaAP]

Qp/Qs=[X-(X-30)]/[100-(X-30)]

Qp/Qs=[85-55]/[100-55]

Qp/Qs=[30]/[45] = 2/3

Cardiopathies à sang mélangé
Atrésie pulmonaire avec CIV



Qp/Qs=[SaAo-SaVC]/[SaVP-SaAP]

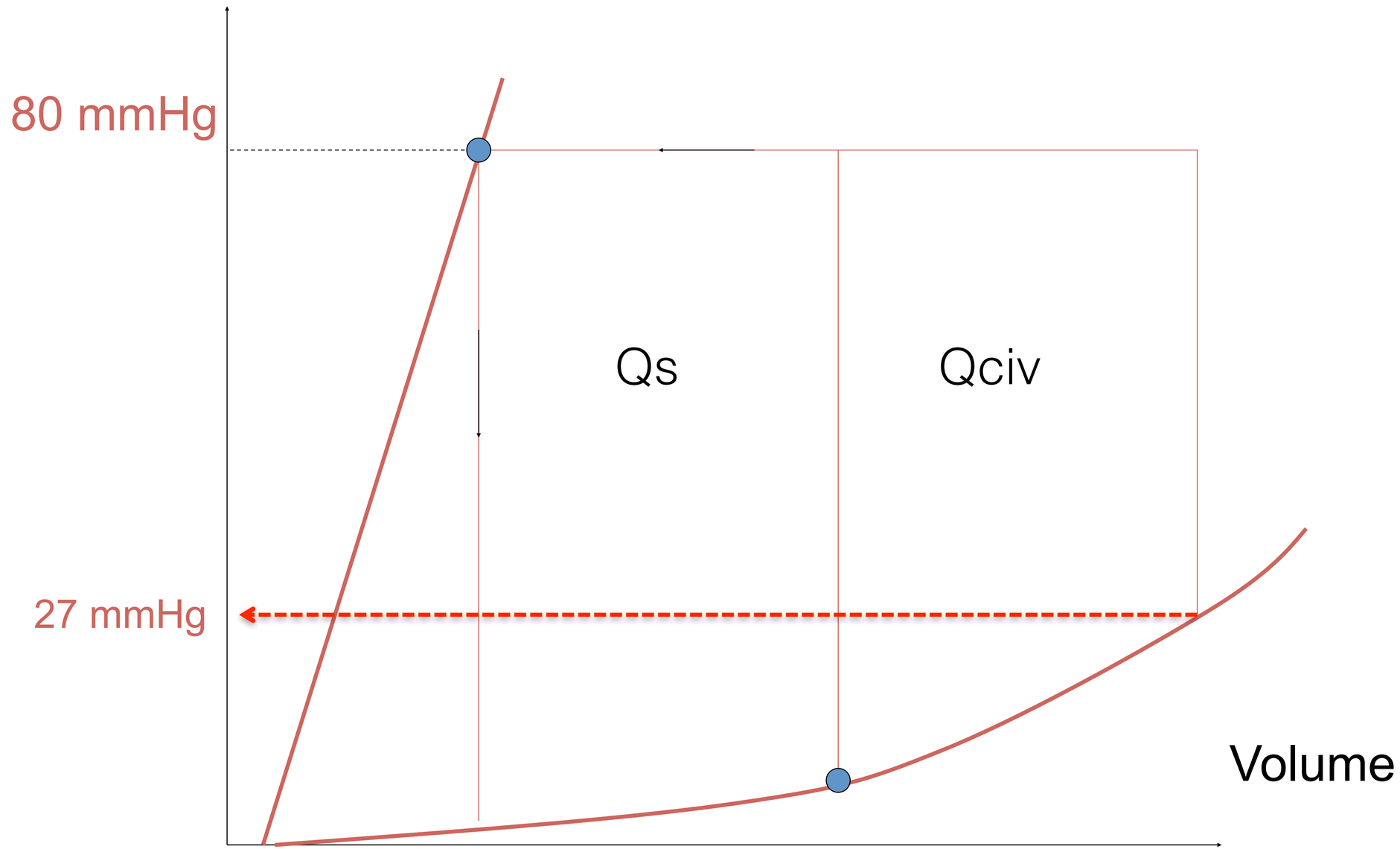
Qp/Qs=[X-(X-30)]/[100-(X-30)]

Qp/Qs=[85-55]/[100-85]

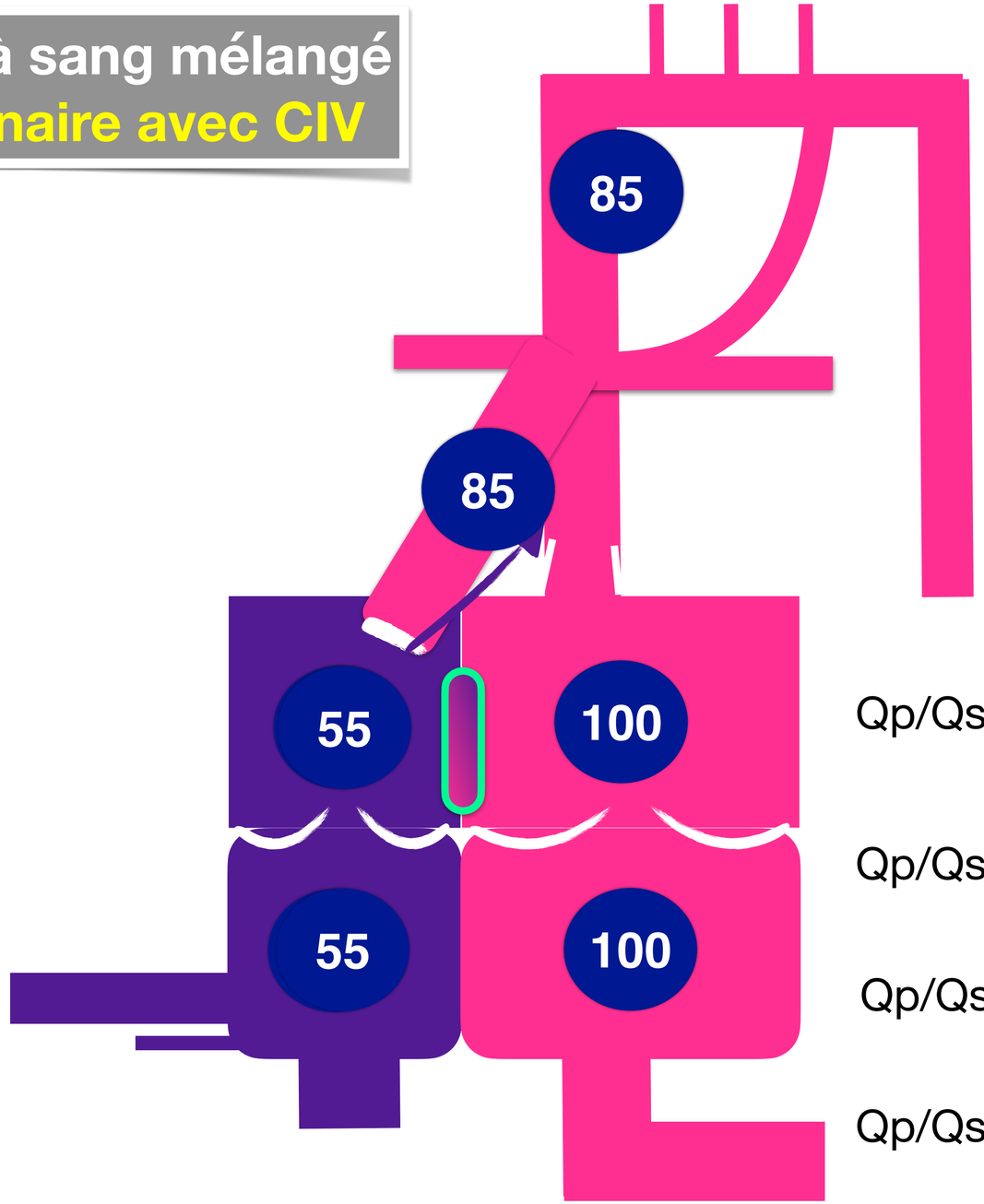
Qp/Qs=[30]/[15] = 2

VG

Qp/Qs = 2 / VESvg= 2 VESvd



Cardiopathies à sang mélangé
Atrésie pulmonaire avec CIV



Qp/Qs=[SaAo-SaVC]/[SaVP-SaAP]

Qp/Qs=[X-(X-30)]/[100-(X-30)]

Qp/Qs=[85-55]/[100-85]

Qp/Qs=[30]/[15] = 2

Qp/Qs

Estimation du Qp/Qs dans les cardiopathies congénitales

Shunt gauche-droite : l'inconnue est la saturation dans l'artère pulmonaire

Shunt droite-gauche : la saturation dans l'aorte donne le Qp/Qs car la saturation dans les veines caves et dans l'artère pulmonaire sont égales à Ao-DAV

Cardiopathies à sang mélangé : la saturation dans l'aorte donne le Qp/Qs car la saturation dans l'AP est égale à celle de l'aorte et la saturation dans la VC est égale à Ao-DAV

Estimation des résistances pulmonaires

Résistances=$\Delta P/Q$

Résistances pulmonaires

Rp= ΔP (PAP moyenne-POG moyenne) / Qp

Résistances systémiques

Rs= ΔP (PAo moyenne -POD moyenne) / Qs

Rapport des résistances pulmonaires/systémiques

Rp/Rs= ΔPp . Qs/ ΔPs . Qp

Rp/Rs= (ΔPp/ΔPs) . (Qs/Qp)

Les différents types d'hypertensions pulmonaires associées aux cardiopathies congénitales

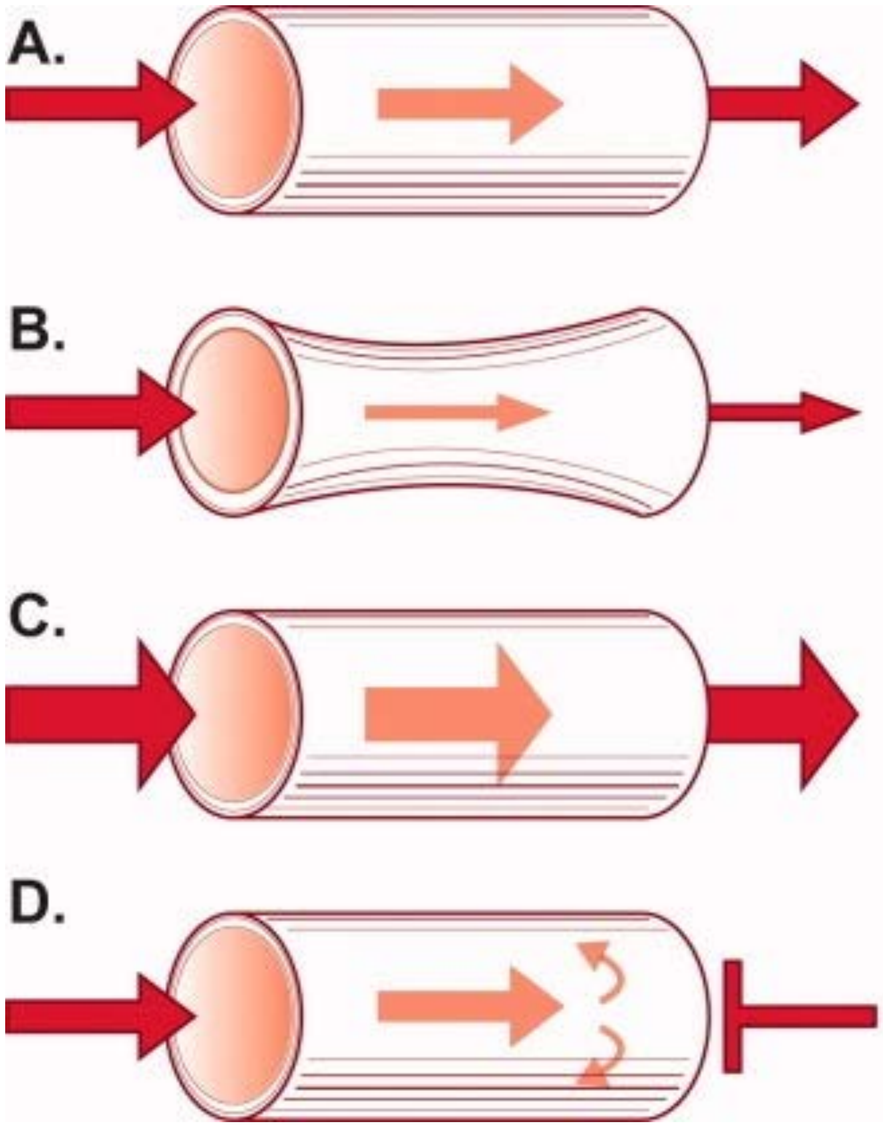
$$Rp = \Delta P / Qp$$

mPAP-PWP=Qp x Rp

mean PAP = Flow x Resistance + PWP

- Flow-associated pulmonary hypertension (hyperkinetic)
  - congenital systemic pulmonary shunt
- Increased pulmonary vascular resistance
  - pulmonary arteriopathy ("Eisenmenger")
- Pulmonary venous congestion

Current hemodynamic classification of PH

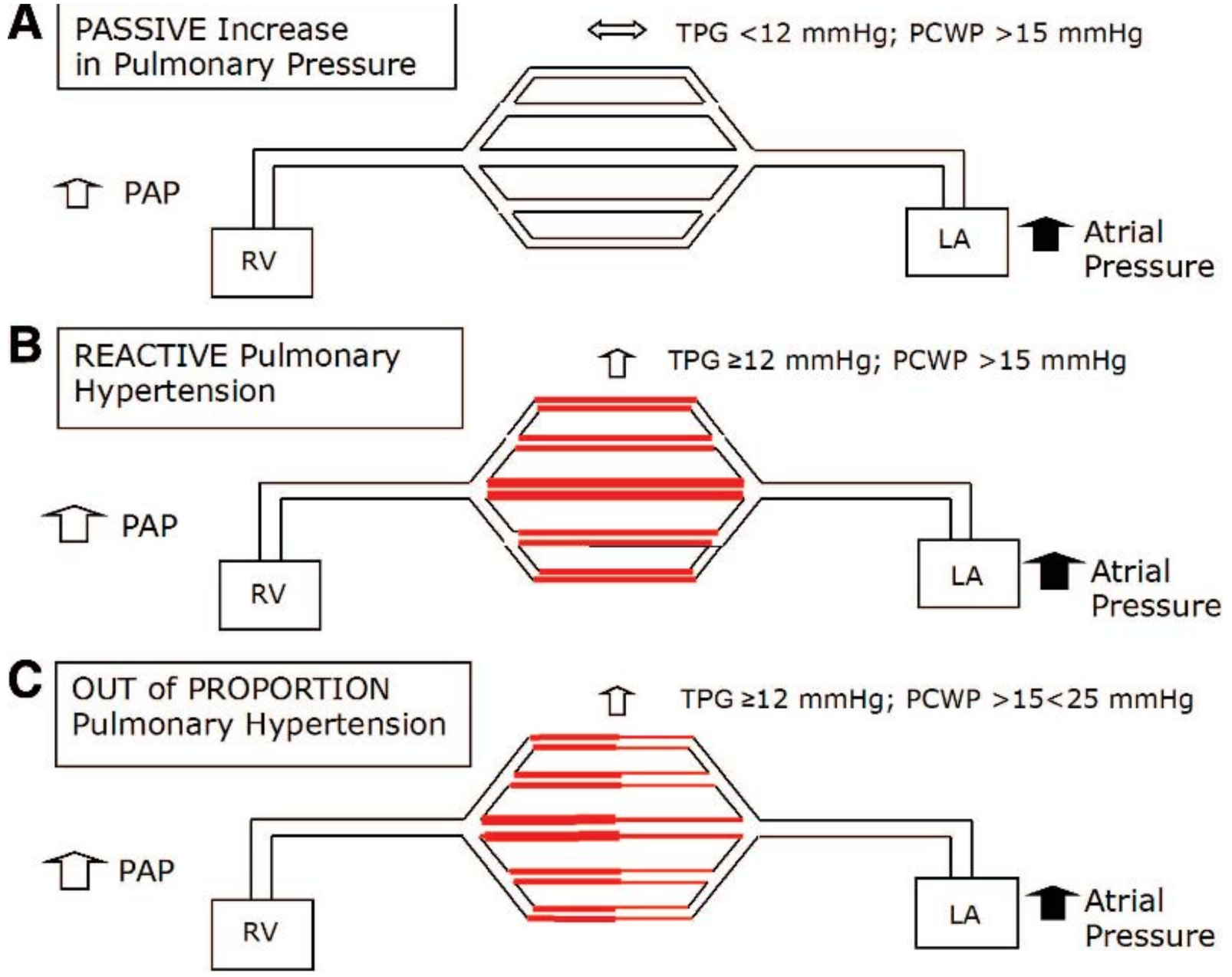


Normal Hemodynamics. Pulmonary artery pressure, cardiac output, PVR, and PCWP are normal.

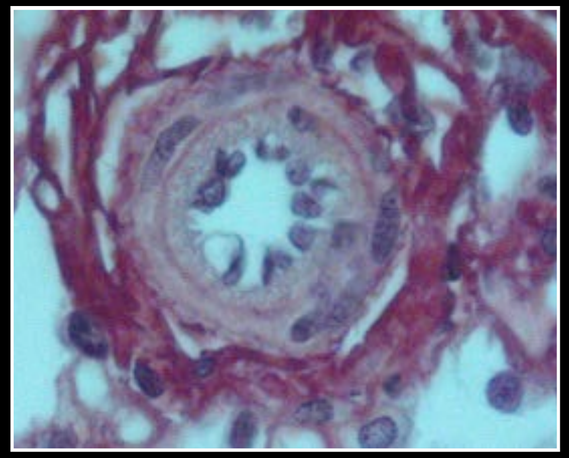
PAH. Elevated pulmonary artery pressure caused by increased PVR with normal cardiac output and PCWP.

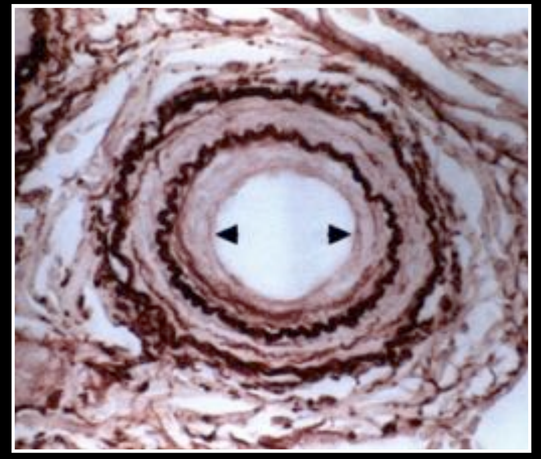
High-flow condition. Elevated pulmonary artery pressure caused by increased cardiac output but normal PVR and PCWP.

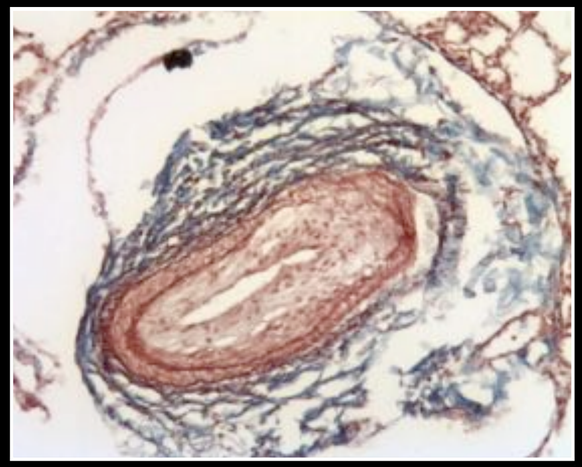
Pulmonary venous hypertension. Elevated pulmonary artery pressure caused by increased PCWP, normal cardiac output and PVR.


A
PASSIVE Increase in Pulmonary Pressure
⇔ TPG <12 mmHg; PCWP >15 mmHg
PAP
RV
LA
Atrial Pressure
B
REACTIVE Pulmonary Hypertension
⇧ TPG ≥12 mmHg; PCWP >15 mmHg
PAP
RV
LA
Atrial Pressure
C
OUT of PROPORTION Pulmonary Hypertension
⇧ TPG ≥12 mmHg; PCWP >15<25 mmHg
PAP
RV
LA
Atrial Pressure

Natural history of L-R shunts

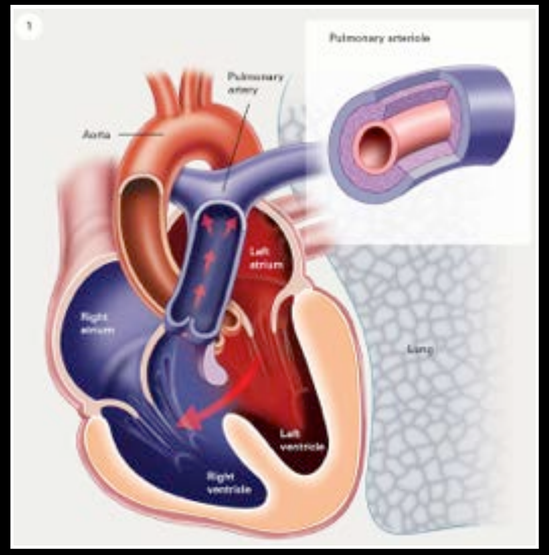


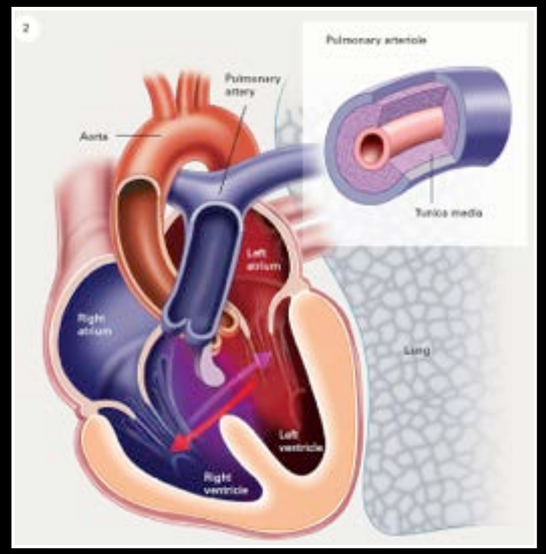


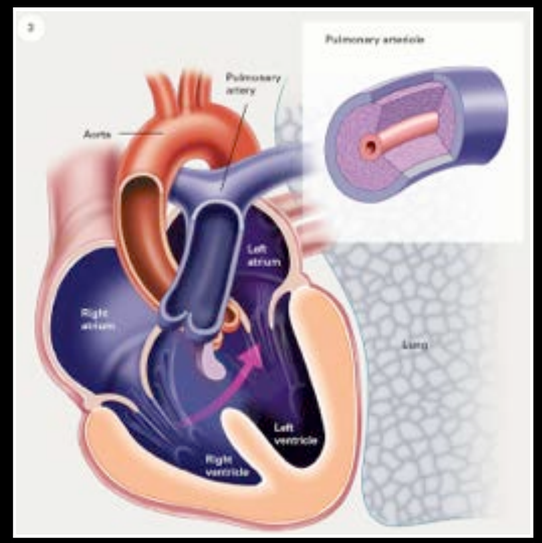


Reversible

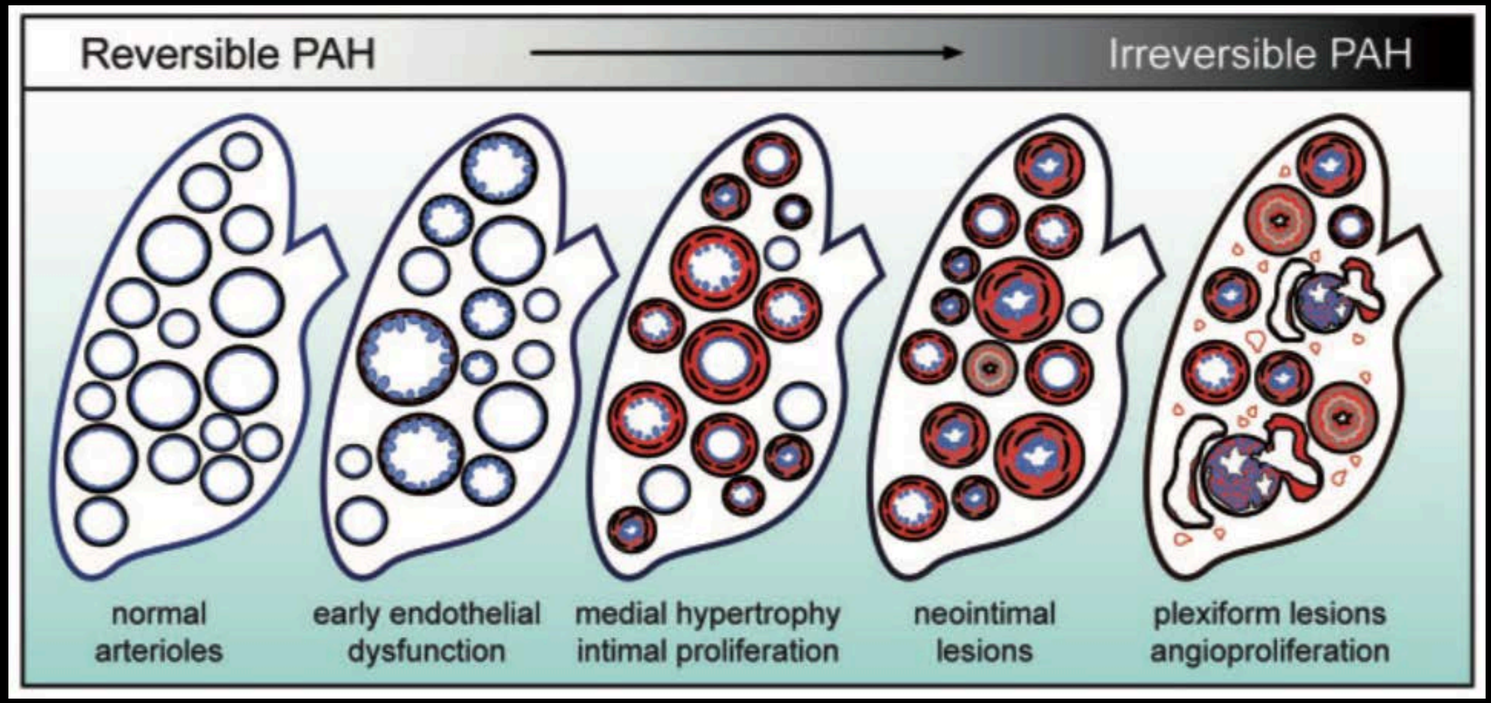
Non reversible

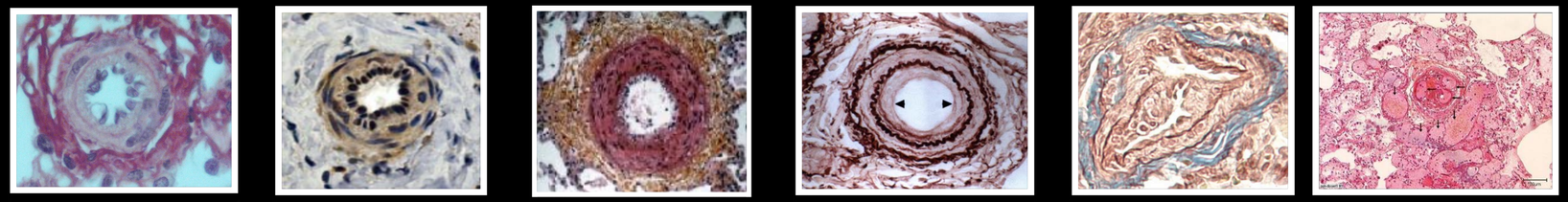


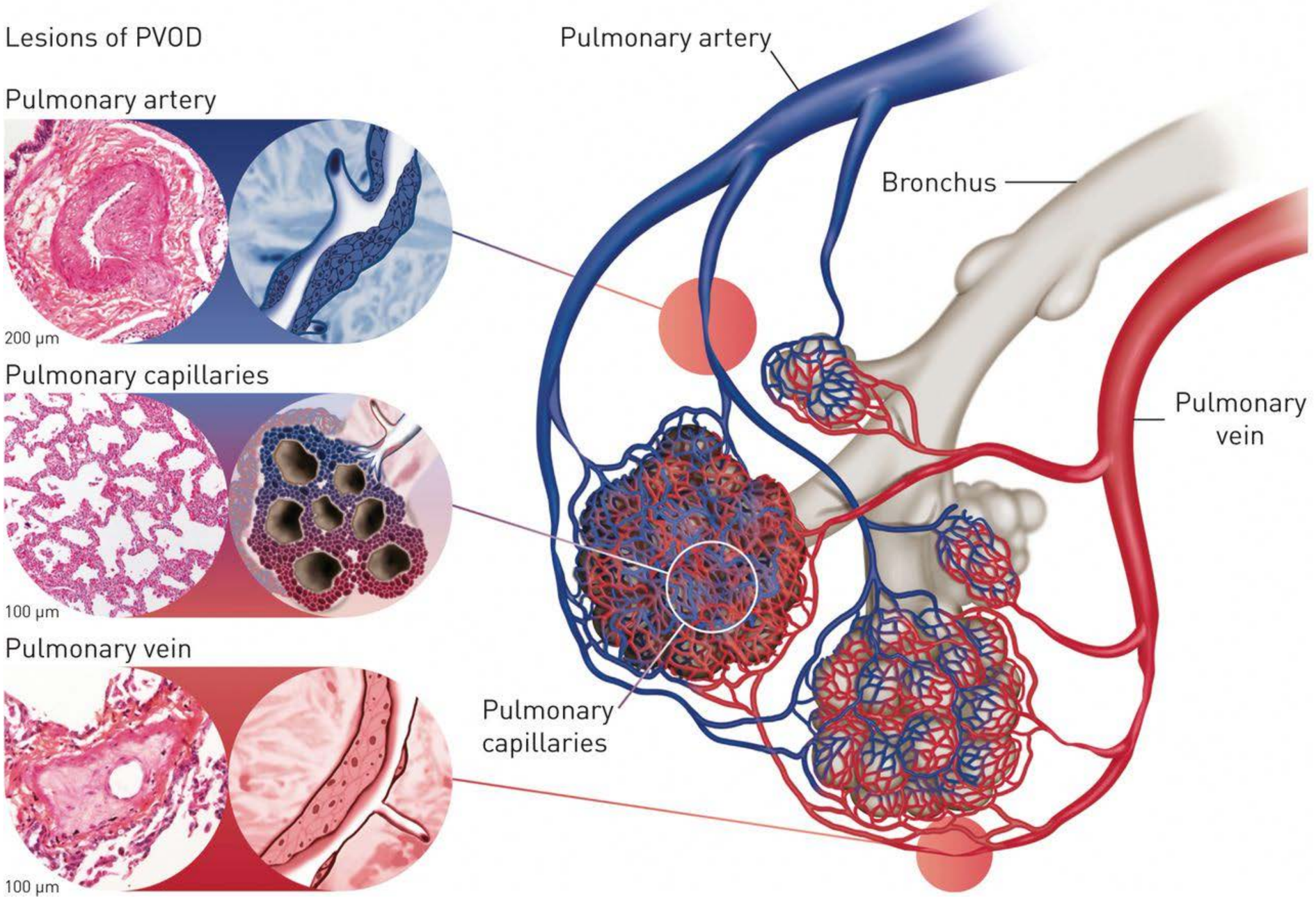




Left-to-right shunt: natural history/*pathology*






Lesions of PVOD
Pulmonary artery
200 µm
Pulmonary capillaries
100 µm
Pulmonary vein
100 µm
Pulmonary artery
Bronchus
Pulmonary vein
Pulmonary capillaries

Estimation des résistances pulmonaires

Résistances=$\Delta P/Q$

Résistances pulmonaires

Rp= ΔP (PAP moyenne-POG moyenne) / Qp

Résistances systémiques

Rs= ΔP (PAo moyenne -POD moyenne) / Qs

Rapport des résistances pulmonaires/systémiques

Rp/Rs= ΔPp . Qs/ ΔPs . Qp

Rp/Rs= (ΔPp/ΔPs) . (Qs/Qp)

Hypertension pulmonaire des cardiopathies congénitales
Exemple d'une CIV large

CIV large chez un enfant de 6 mois
Pression aortique = 90/60 moyenne 70 mmHg
POG estimée à 15 mmHg sur un PFO et l'absence d'hépatomégalie
Quelle est la pression systolique dans l'artère pulmonaire ?

90 mmHg

Le ventricule gauche est dilaté avec un z-score à 3.5.
Quelle est votre estimation du Qp/Qs ?

Augmenté > 1 par le shunt gauche-droite

Si le Qp/Qs est à 3, quelle est votre estimation des résistances pulmonaires ?

Rp/Rs= (ΔPp/ΔPs) . (Qs/Qp) soit (PAPmoyenne-15)/(70-POD) x 1/3
Rp/Rs= (X-15)/(70-8) x 1/3 => (30-15)/62 x 1/3= 1/12

Hypertension pulmonaire des cardiopathies congénitales
Exemple d'une CIV large

CIV large chez un enfant de 6 ans
Pression aortique = 110/60 moyenne 85 mmHg
Quelle est la pression systolique dans l'artère pulmonaire ?

110 mmHg

Le cathétérisme cardiaque note une saturation dans l'AP à 70% et dans l'aorte à 100%. Quelle est votre mesure du Qp/Qs ?

Egal à 1

Quelle est votre estimation des résistances pulmonaires si la pression artérielle pulmonaire est à 110/20 moyenne 45 ?

$Rp/Rs = (\Delta Pp/\Delta Ps) \cdot (Qs/Qp)$ soit $(PAPmoyenne-POG)/(85-POD) \times 1$
$Rp/Rs = (45-10)/(85-8) \times 1 = 35/73 \times 1 = 1/2$

Hypertension pulmonaire des cardiopathies congénitales
Exemple d'une CIV large

CIV large chez un adolescent de 16 ans
Pression aortique = 110/60 moyenne 85 mmHg
Quelle est la pression systolique dans l'artère pulmonaire ?

110 mmHg

Le cathétérisme cardiaque note une saturation dans l'AP à 55% et dans l'aorte à 85%. Quelle est votre mesure du Qp/Qs ?

Egal à 2/3

Quelle est votre estimation des résistances pulmonaires si la pression artérielle pulmonaire est à 110/38 moyenne 70 ?

Rp/Rs= (ΔPp/ΔPs) . (Qs/Qp) soit (PAPmoyenne-POG)/(85-POD) x 1
Rp/Rs= (70-10)/(85-8) x 3/2 = 60/77 x 3/2= 18/15 > 1

Physiologie cardiaque appliquée aux cardiopathies congénitales

Partie 4

Insuffisance cardiaque appliquée aux cardiopathies congénitales

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Institut Hospitalo-Universitaire IMAGINE

Centre de Référence Maladies Rares
Malformations Cardiaques Congénitales Complexes-M3C
Centre de Référence Maladies Rares
Maladies Cardiaques Héréditaires- CARDIOGEN

Schéma hémodynamique du Cœur en Série

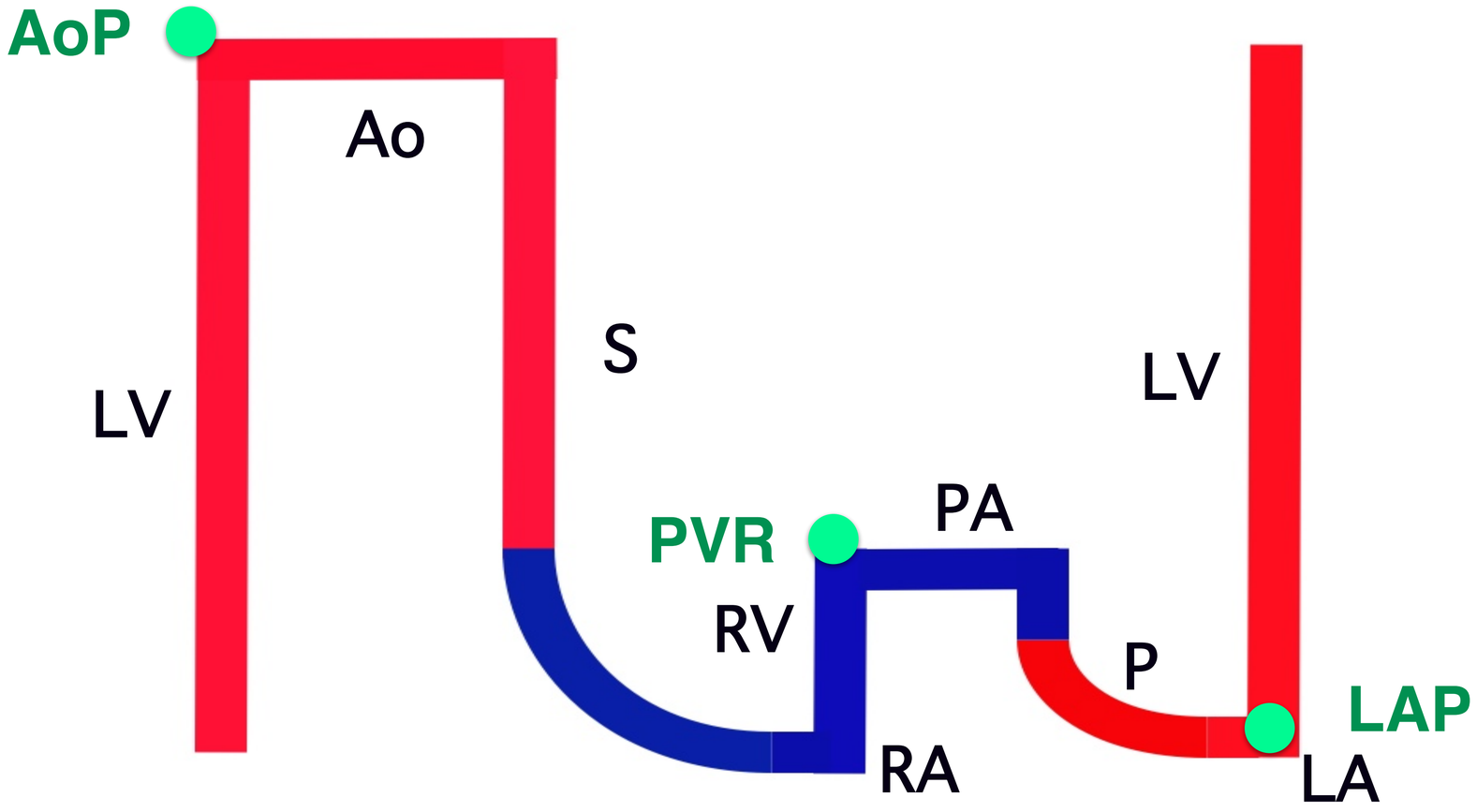
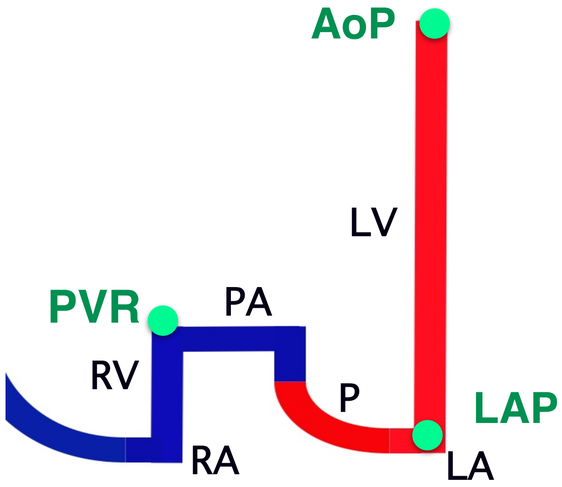


Schéma hémodynamique du Cœur en Série



- On descend de l'Ao aux Veines caves en passant par le cœur gauche, les RVP et le cœur droit
  - VG transforme la Pression Aortique en Pression Capillaire Pulmonaire/POG
  - La traversée du poumon transforme la Pression Capillaire en PAP
  - VD transforme la PAP en Pressions Caves
- A partir du VG, c'est le Débit qui est imposé
  (VES = Qs/FC)
- Ce sont les Pressions d'amont qui varient en fonction des propriétés intrinsèques du cœur et des RVP

The gold standard of **in vivo measured contractility** is end-systolic elastance (Ees), or end-systolic pressure (ESP) divided by end-systolic volume (ESV)

Contractility(Ees)=AoSystolic Pressure/LVESV

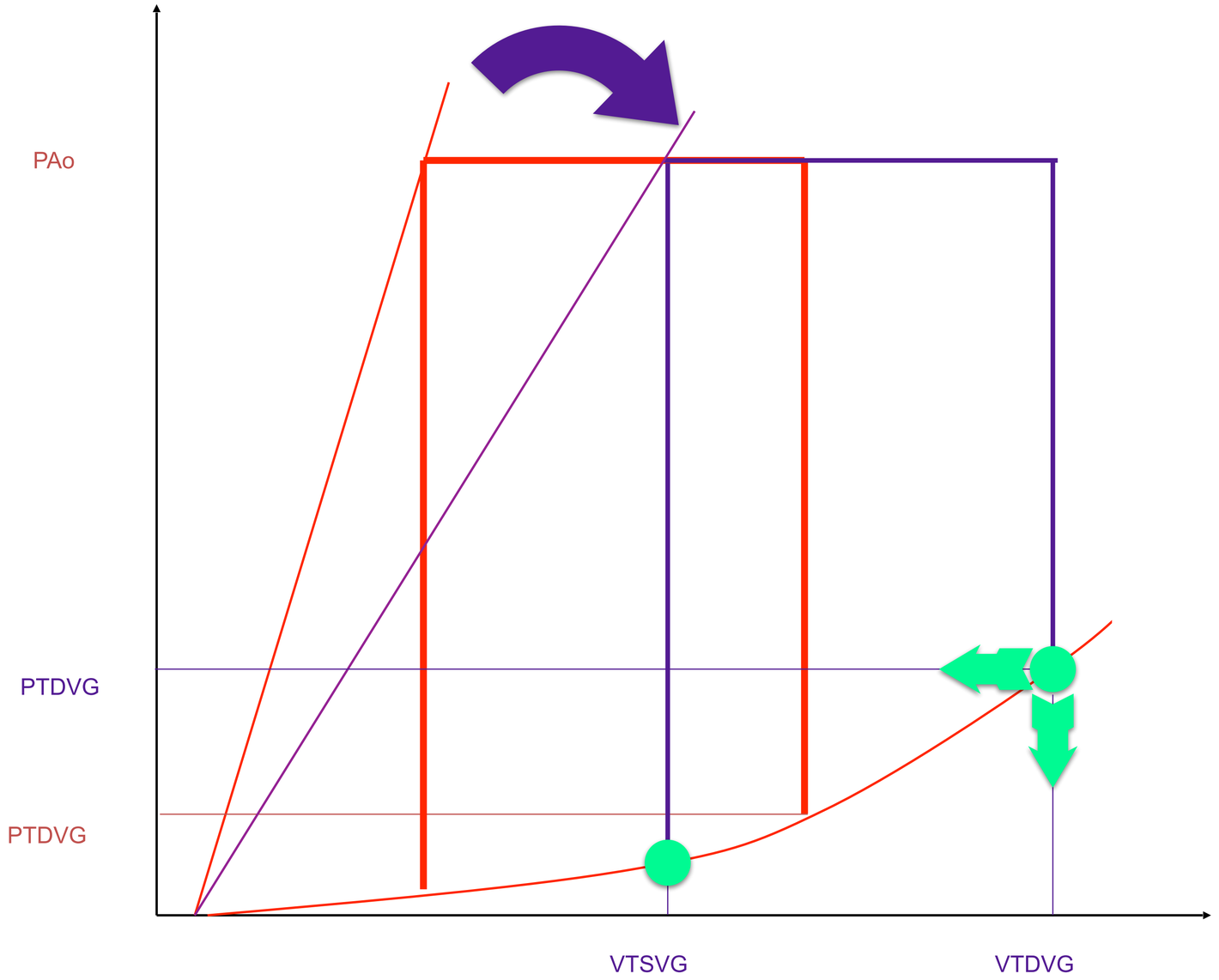
An acceptable measure of **afterload is arterial elastance (Ea),** calculated as ESP divided by stroke volume (SV).

Arterial elastance=AoSystolic Pressure/Stroke volume

The optimal mechanical coupling of RV function to afterload corresponds to an Ees/Ea ratio of 1.

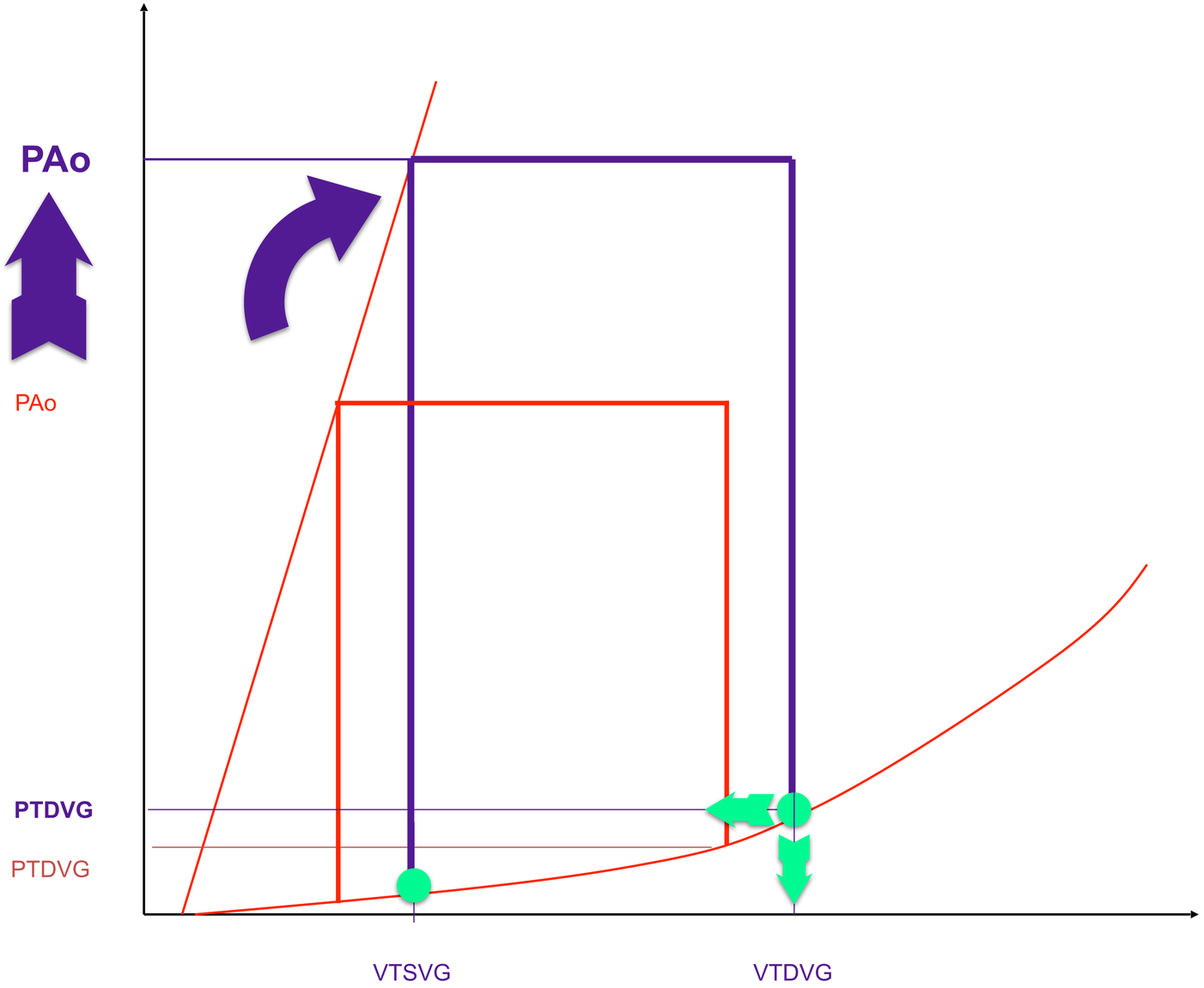
Différents mécanismes de l'insuffisance cardiaque

Altération de la contractilité



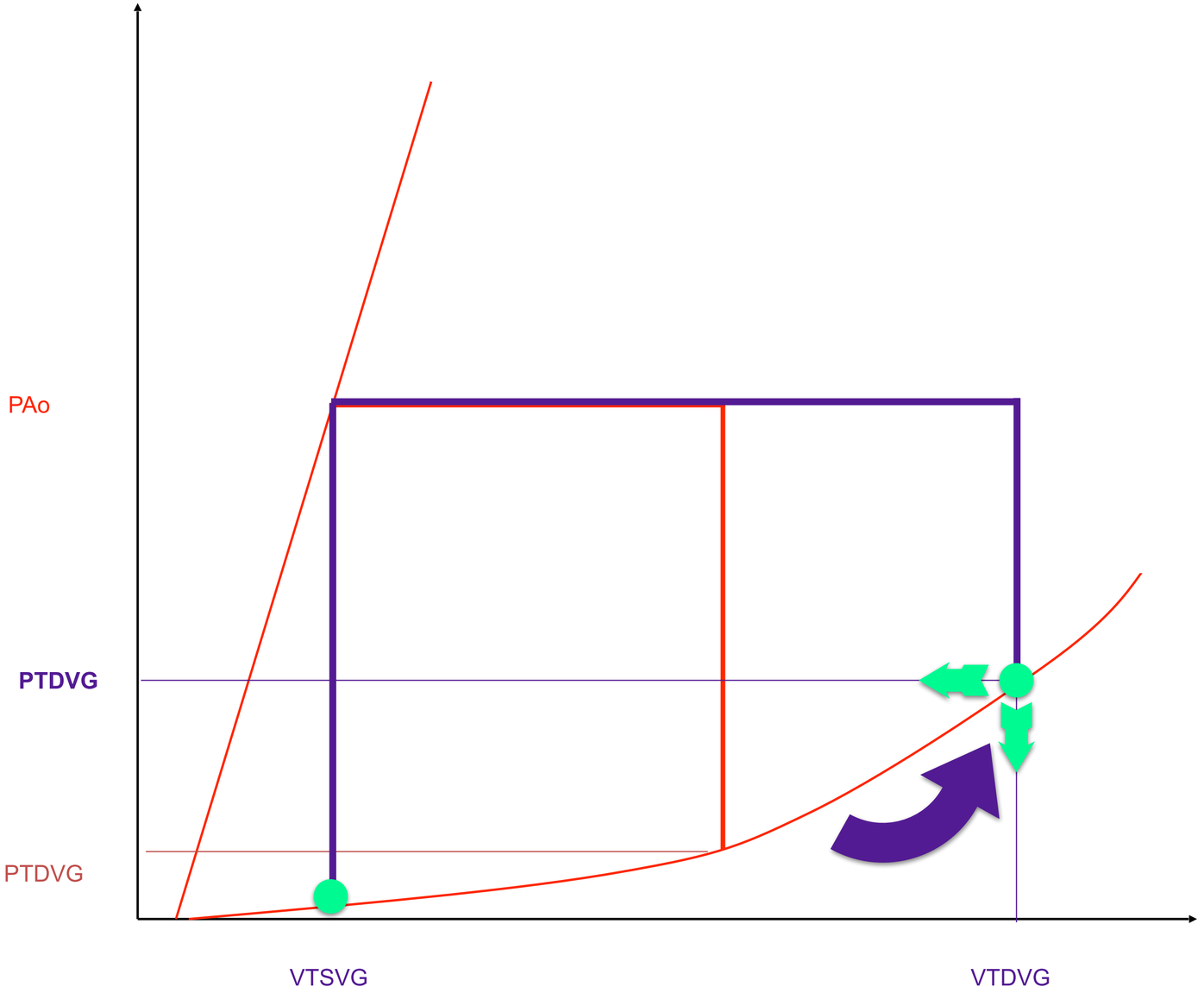
Différents mécanismes de l’insuffisance cardiaque

Augmentation de la post-charge



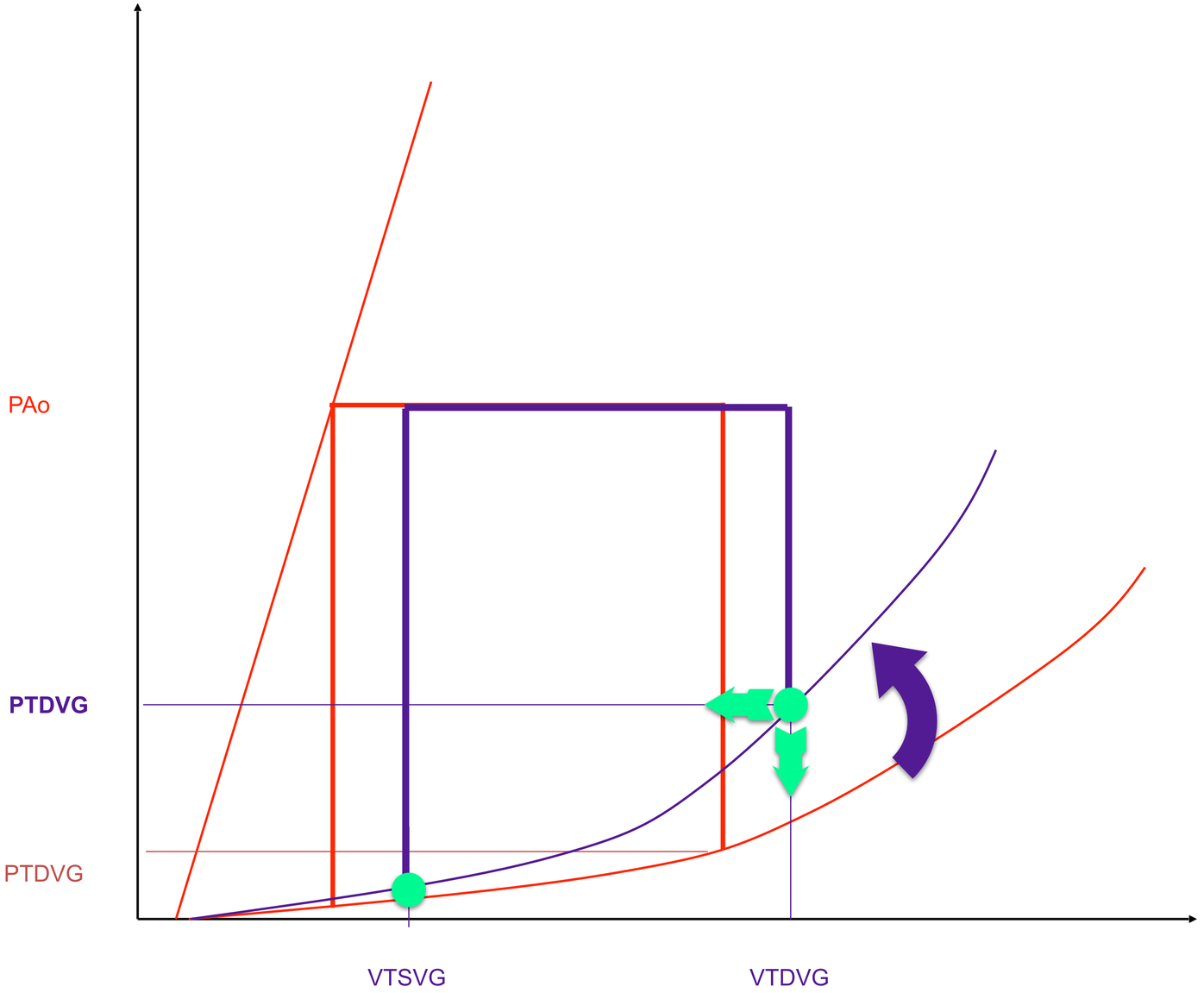
Différents mécanismes de l’insuffisance cardiaque

Augmentation de la pré-charge



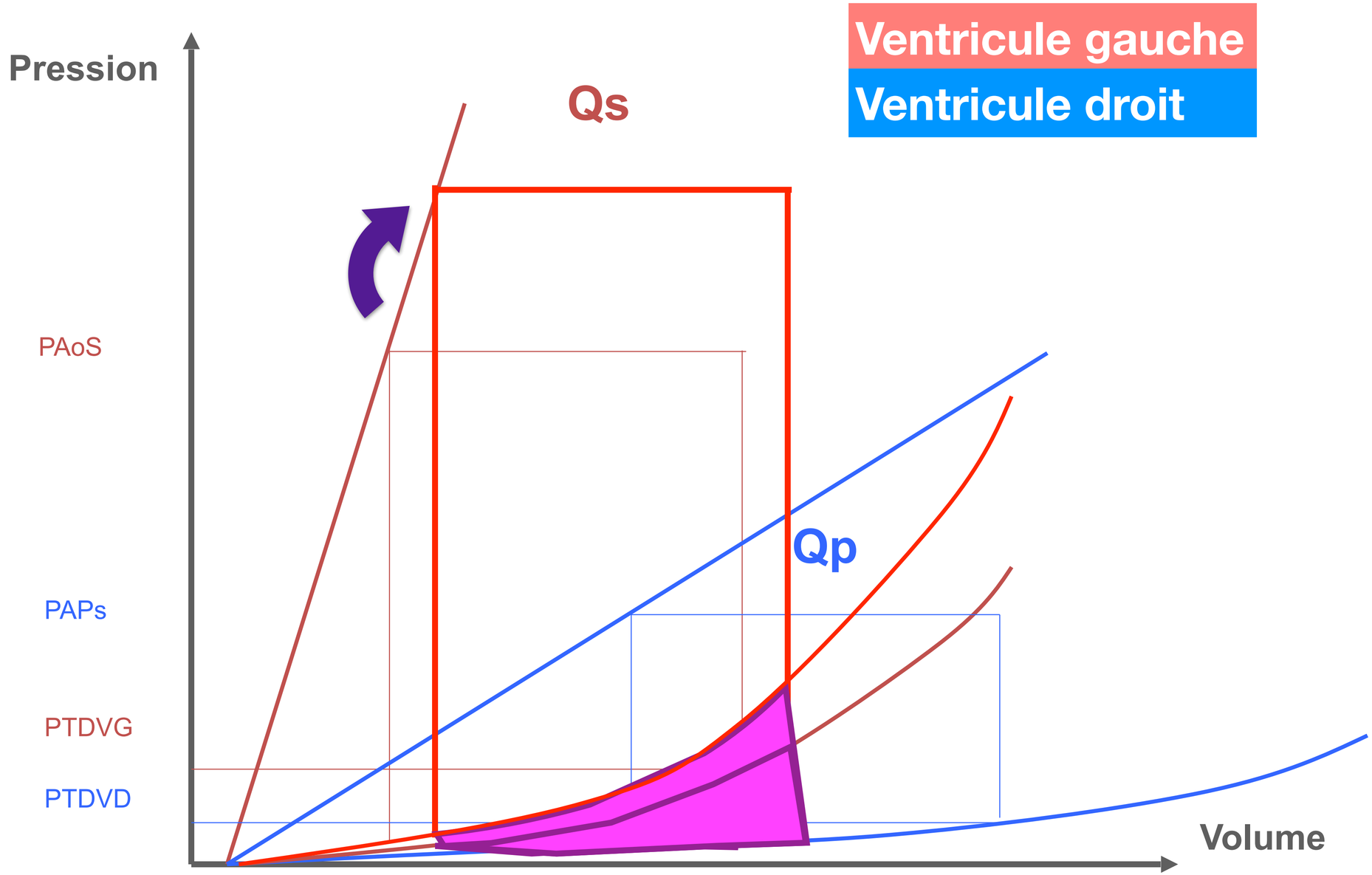
Différents mécanismes de l'insuffisance cardiaque

Diminution de la compliance ventriculaire



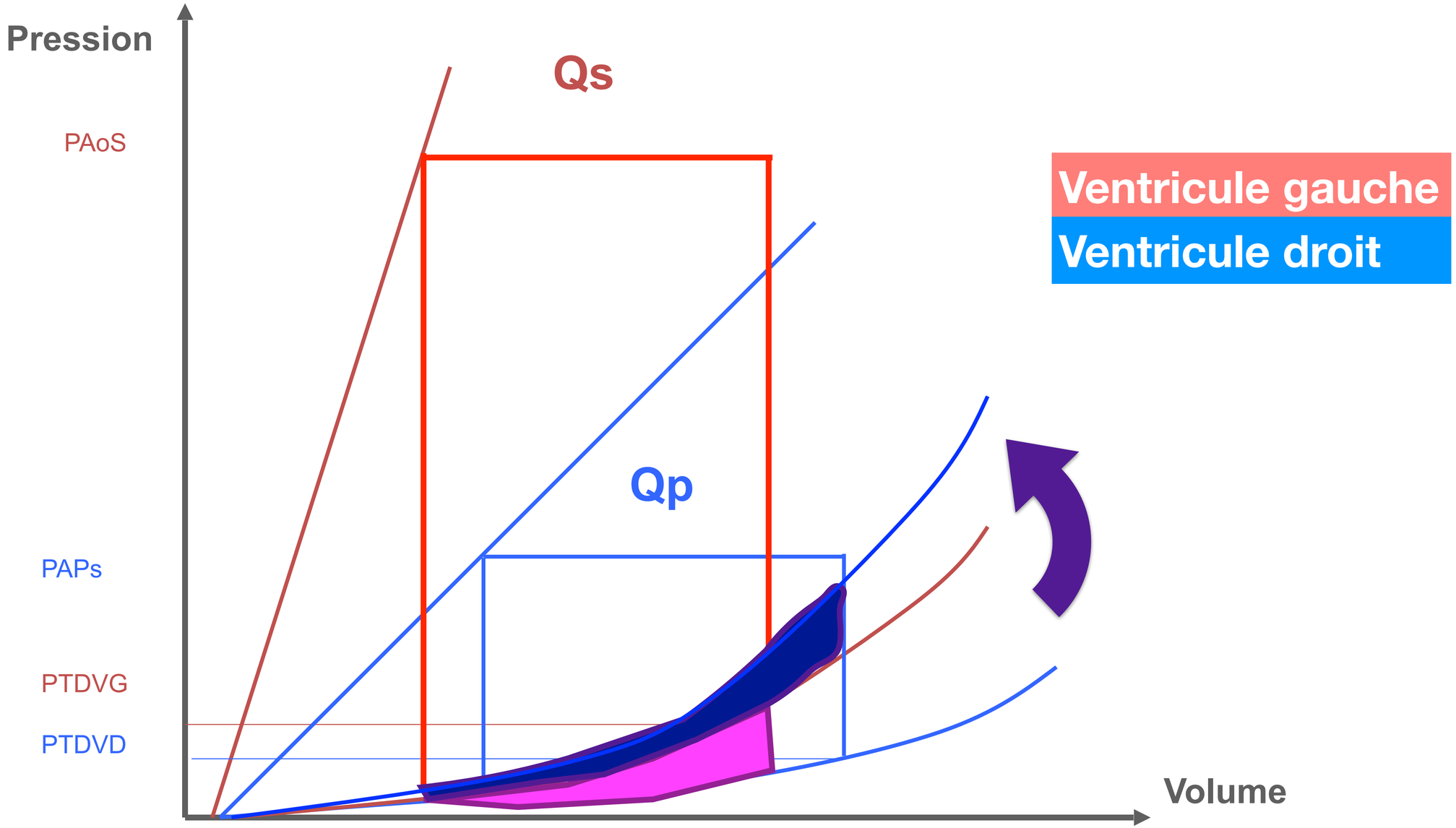
Interactions des mécanismes en cas de cardiopathie congénitale

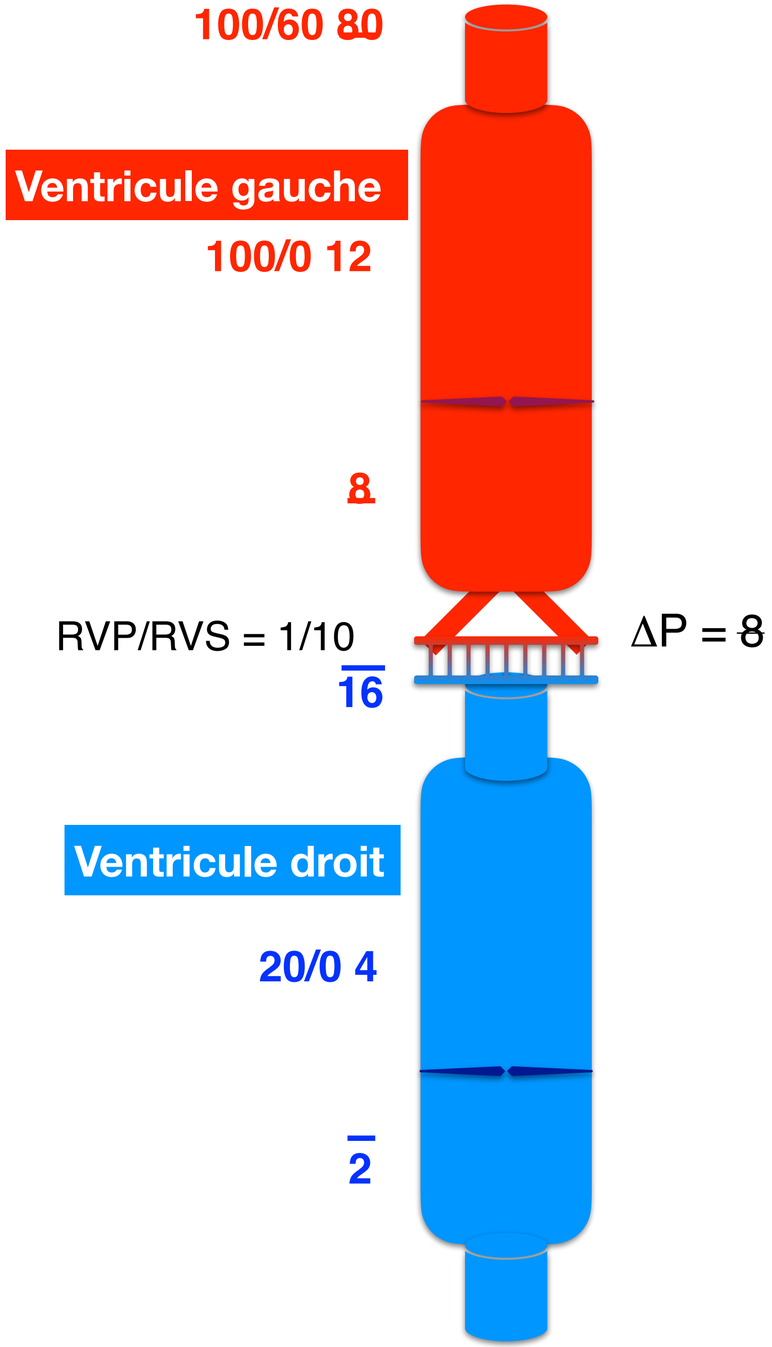
Augmentation de la post-charge du VG et CIA

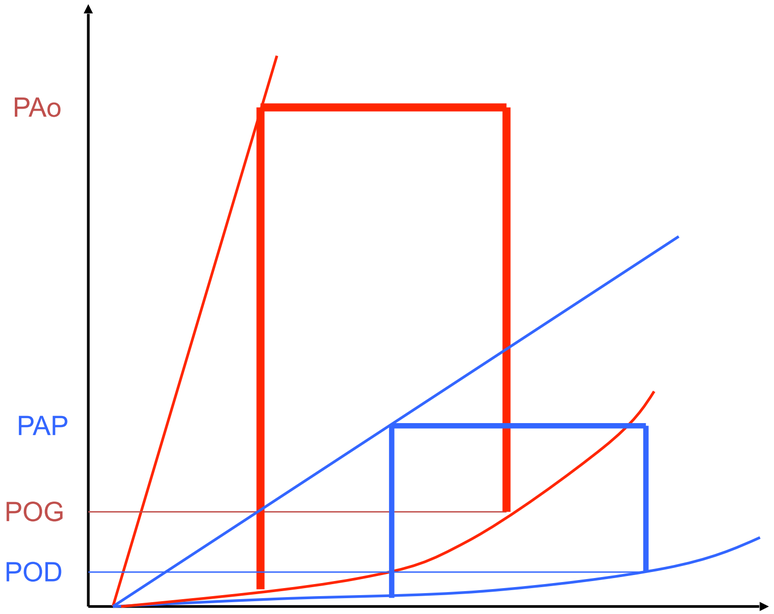


Interactions des mécanismes en cas de cardiopathie congénitale

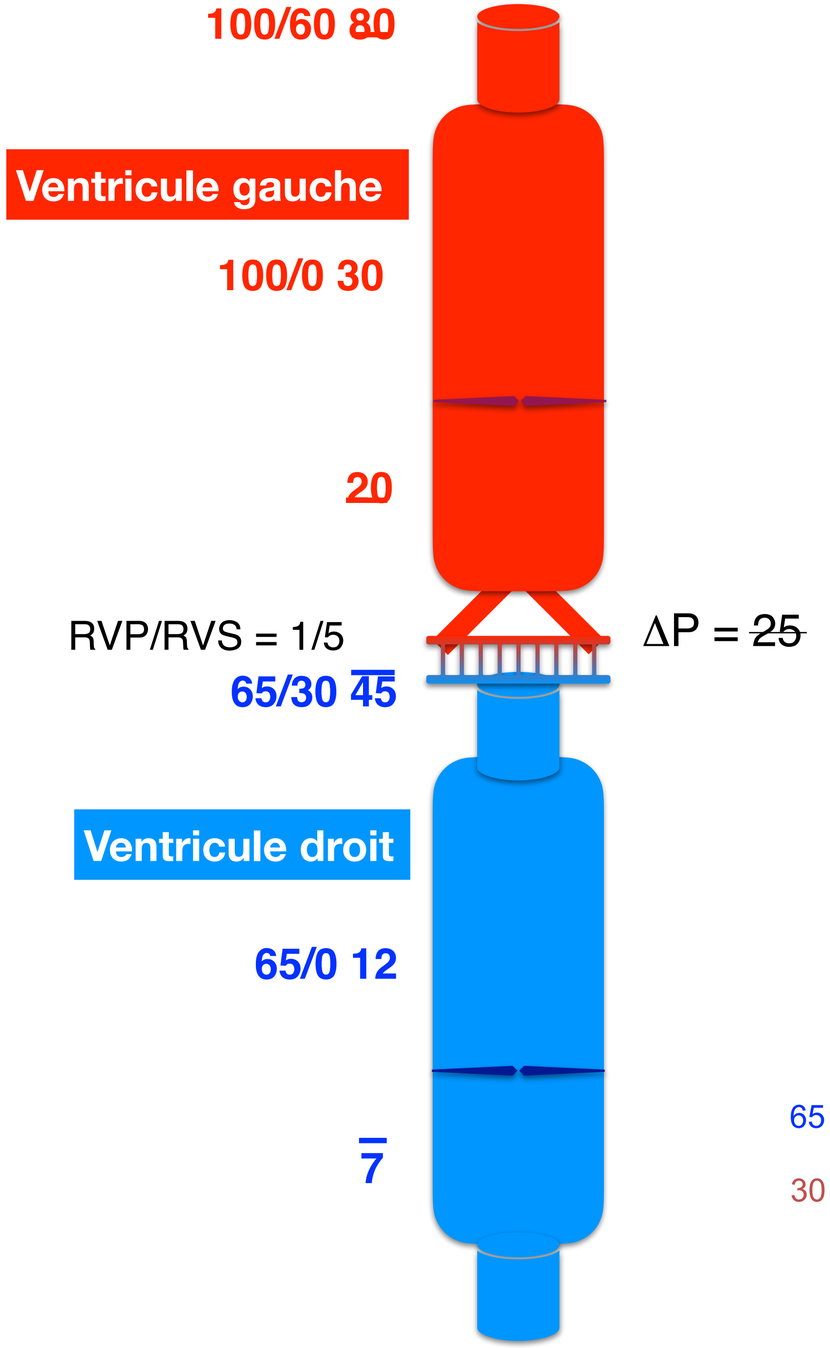
Diminution de la compliance du VD et CIA

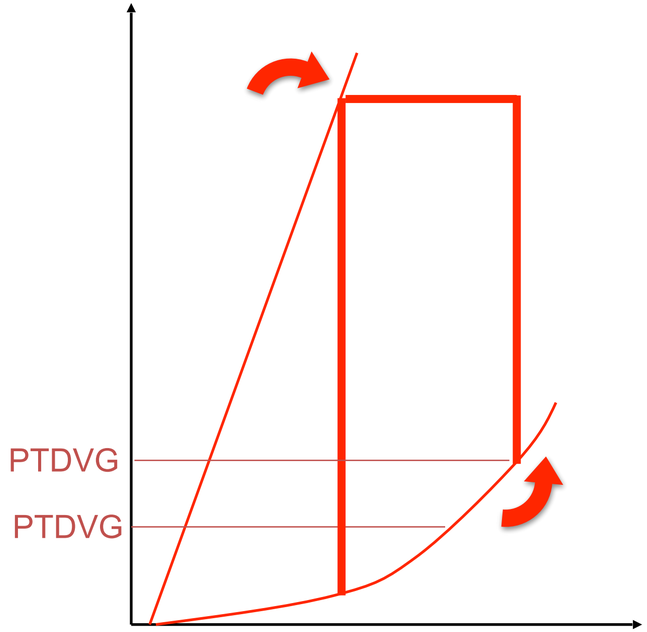


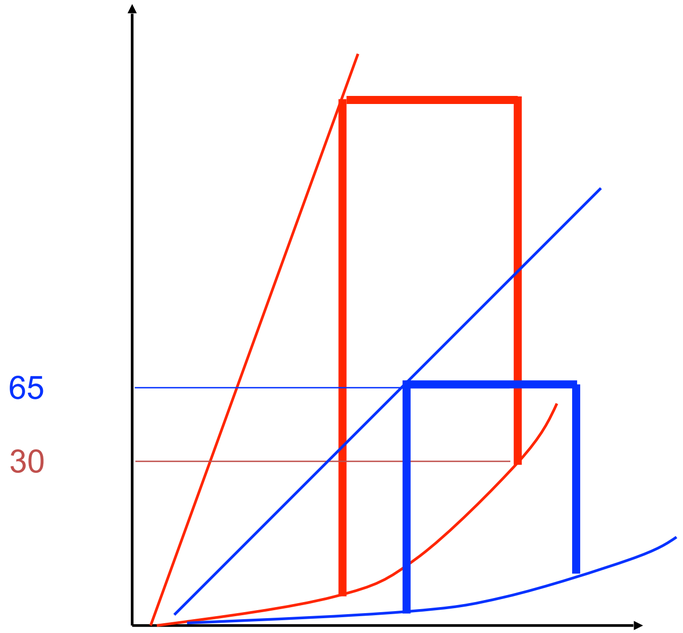

100/60 80
Ventricule gauche
100/0 12
8
RVP/RVS = 1/10
16
ΔP = 8
Ventricule droit
20/0 4
2

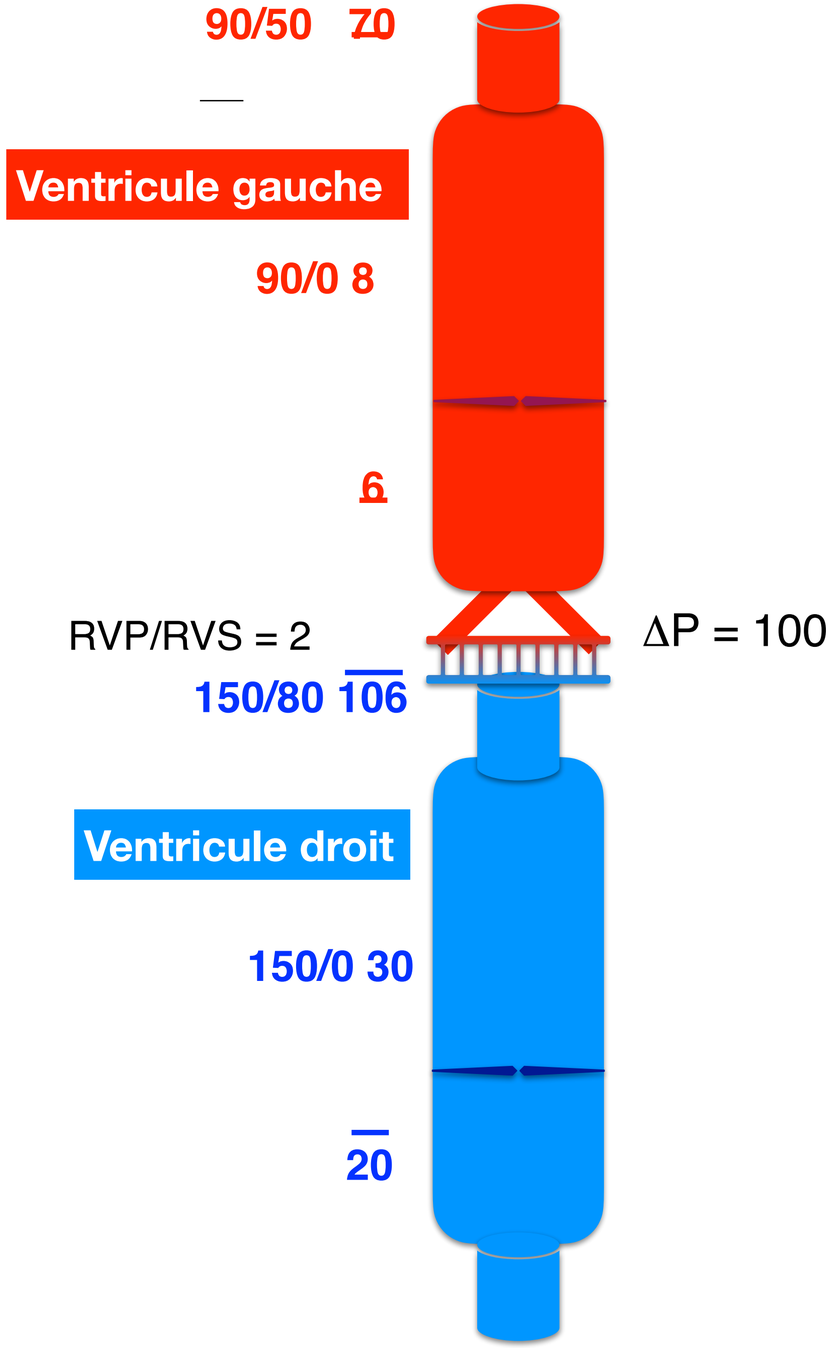

PAo
PAP
POG
POD

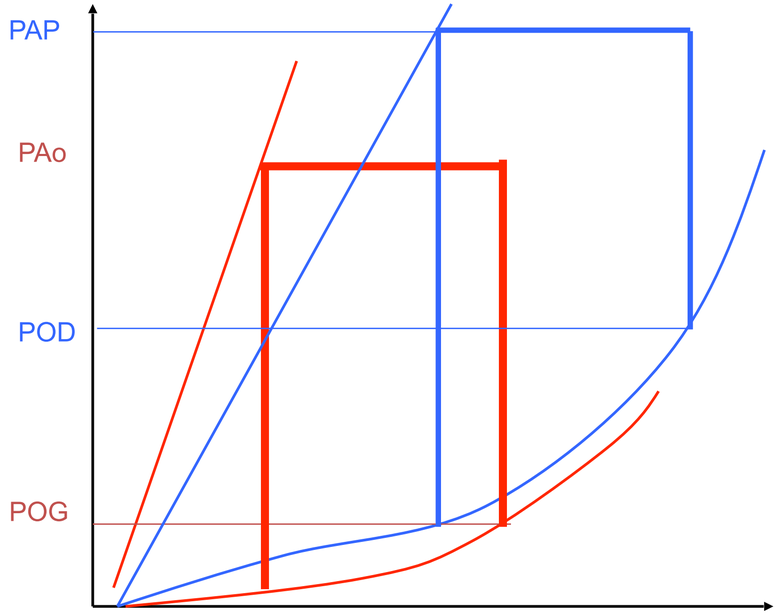
Insuffisance ventriculaire gauche et Post charge du ventricule droit



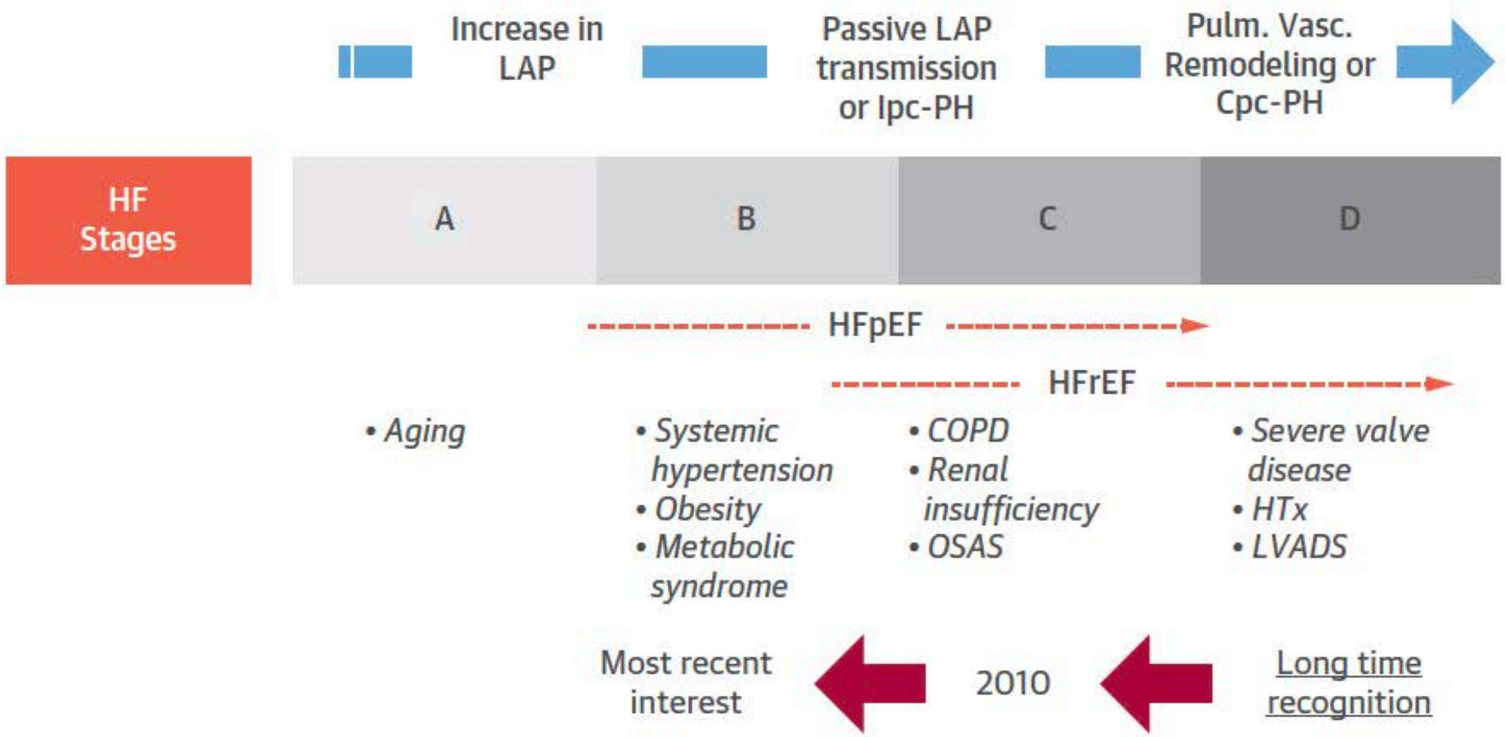





90/50 70
Ventricule gauche
90/0 8
6
RVP/RVS = 2
ΔP = 100
150/80 106
Ventricule droit
150/0 30
20

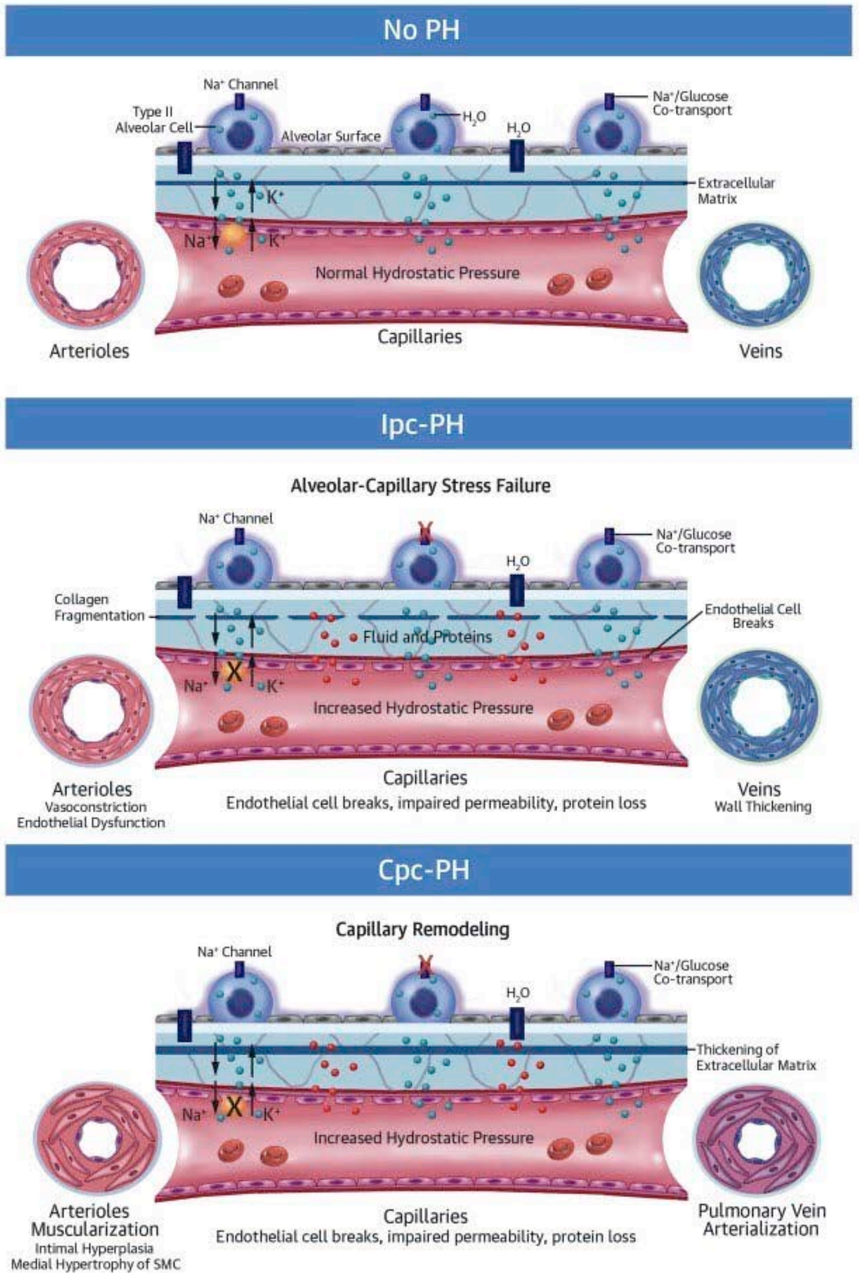

PAP
PAo
POD
POG

PH and its clinical « inducers » according to heart failure stages



For many years, pulmonary hypertension (PH) has been considered to have clinical meaning in advanced heart failure (HF) stages, whereas more recently, interest is focused on earlier stages and corresponding comorbid precipitating factors. COPD = chronic obstructive pulmonary disease; Cpc = combined pre- and post-capillary; HFpEF = heart failure with preserved ejection fraction; HFrEF = heart failure with reduced ejection fraction; HTx = heart transplantation; Ipc = isolated post-capillary; LAP = left atrial pressure; LVADS = left ventricular assist devices; OSAS = obstructive sleep apnea syndrome.

Guazzi and Naeije. JACC 2017



Guazzi, M. et al. J Am Coll Cardiol. 2017;69(13):1718-34.

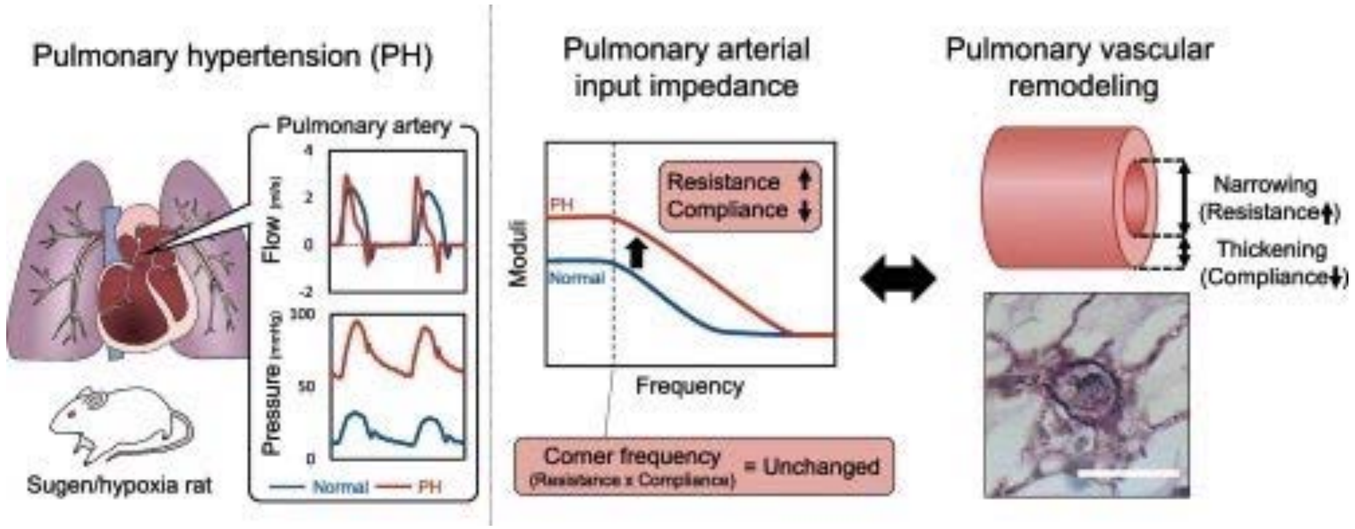
Pulmonary arterial compliance & Pulmonary vascular resistances

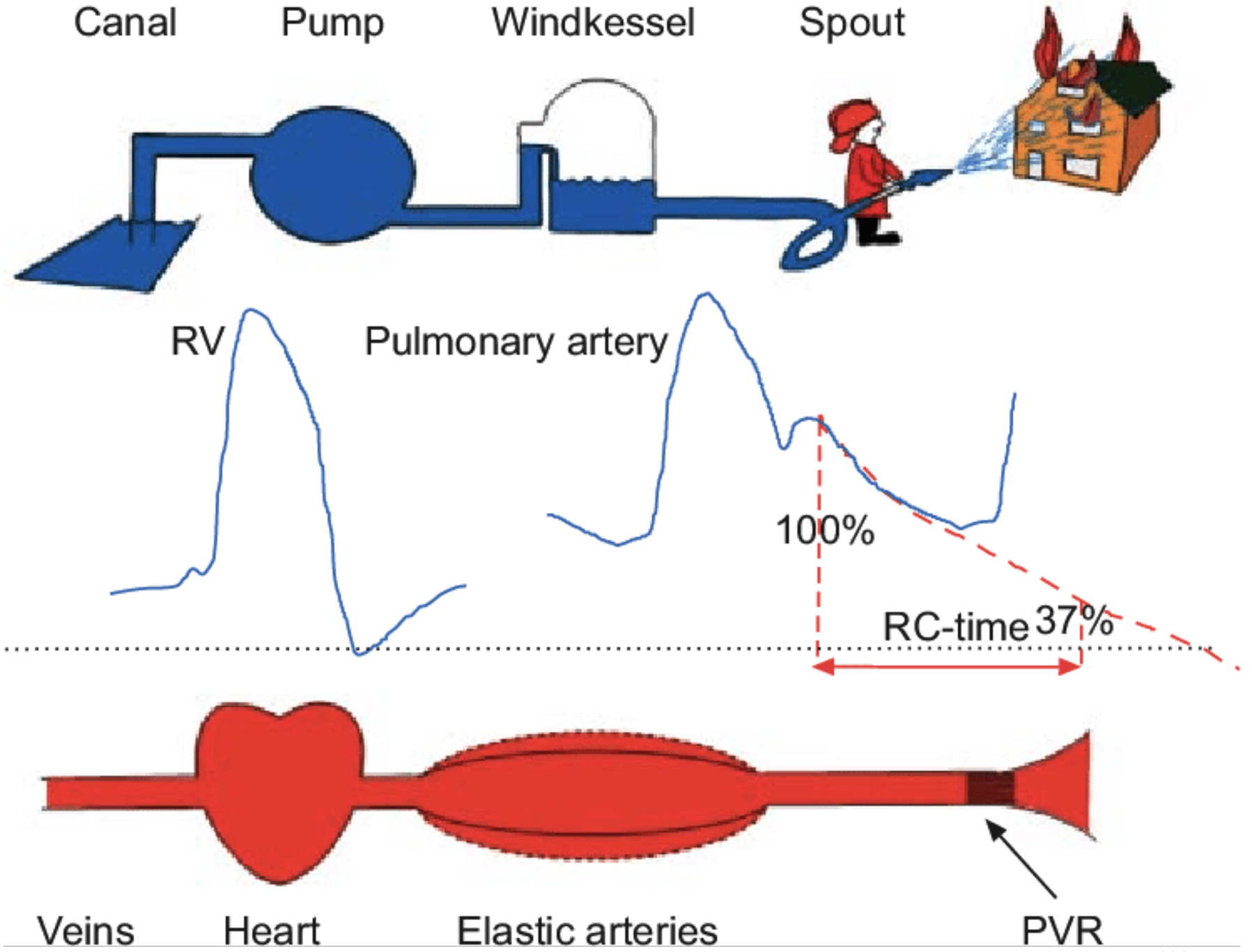
PAC=Qp/Pulse pressure=Qp/PAPs-PAPd

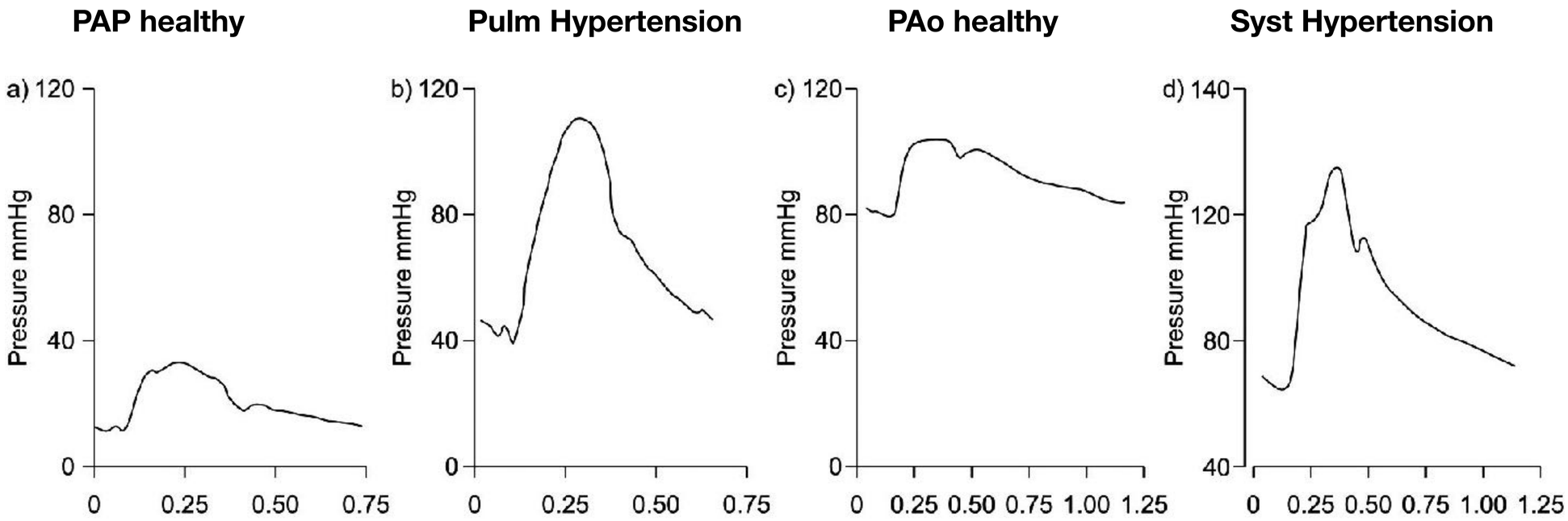
PVR=PAPm-PWP/Qp

La PAC est la composante pulsatile de la post-charge ventriculaire droite.

Les PVR sont la composante résistive de la post-charge ventriculaire droite.

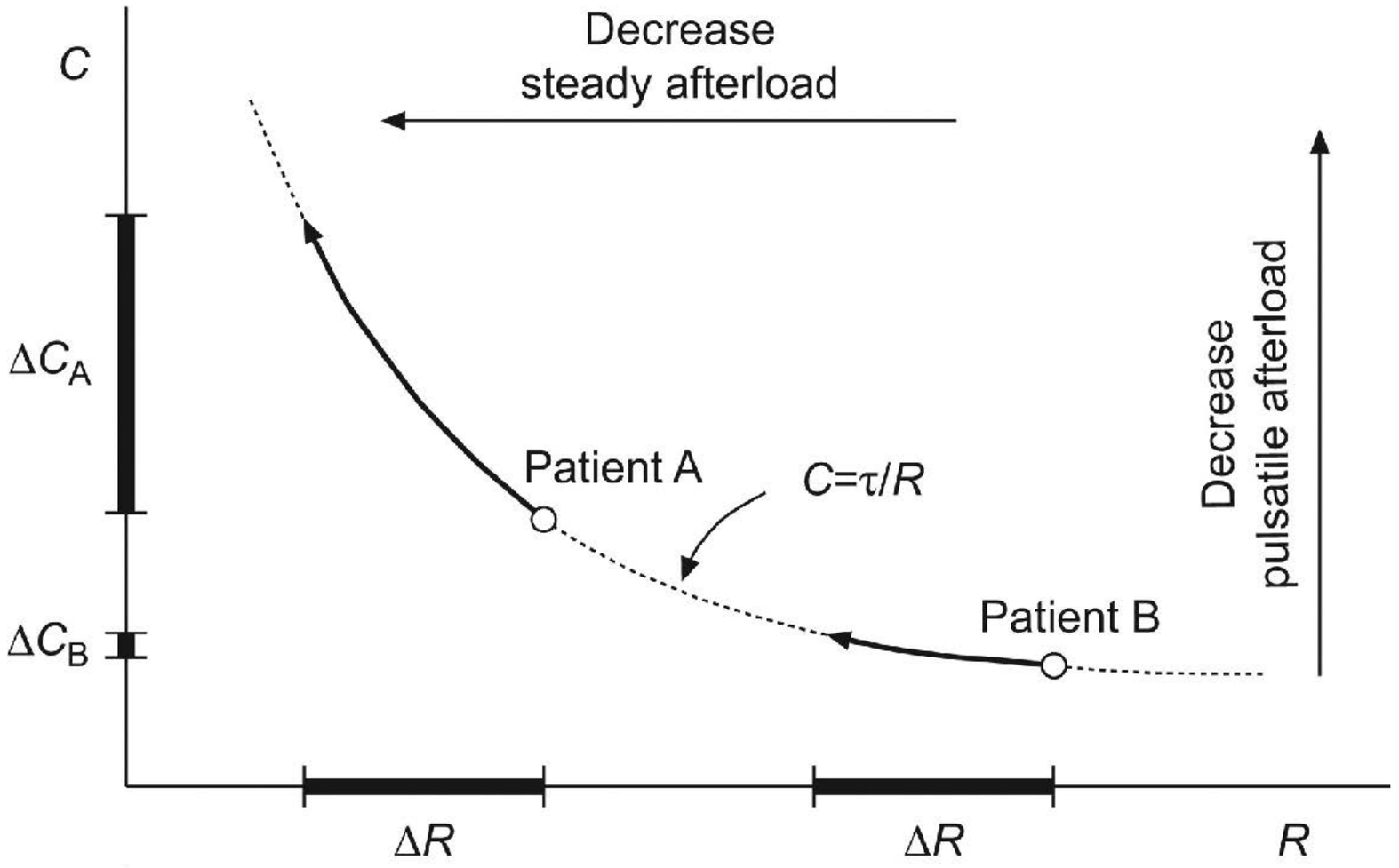



Canal
Pump
Windkessel
Spout
RV
Pulmonary artery
100%
RC-time
37%
Veins
Heart
Elastic arteries
PVR



Pulmonary artery pressure in a) healthy subjects and b) pulmonary hypertension (PH) subjects. Aortic pressure in c) healthy subjects and d) subjects with systemic hypertension subjects. **In PH, systolic, diastolic and pulse pressure increased in proportion with mean pulmonary artery pressure.** In systemic hypertension pulse pressure increases much more than mean aortic pressure and diastolic pressure may even decrease.

Resistance.Compliance-time is a constant / RC-time is constant



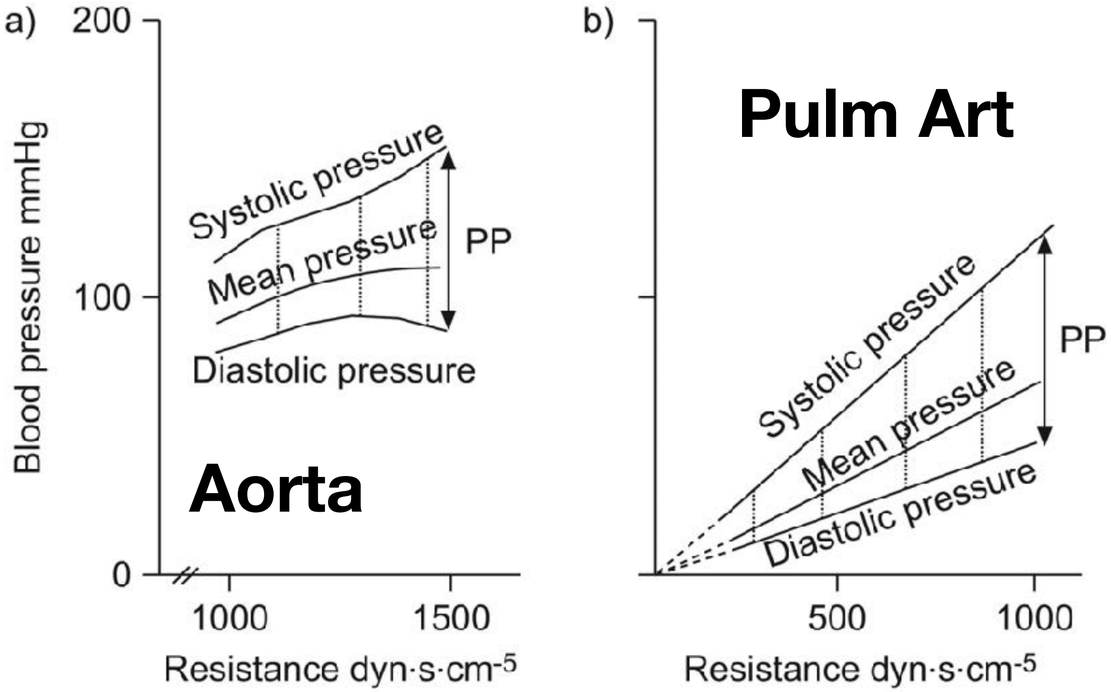
In patient A early stage of PAH, decrease in PVR will result in important decrease in PAC
In patient B, the same decrease in PVR will have limited effect on PAC

The anatomical differences between the pulmonary and systemic arterial system are the main cause of the difference in the distribution of compliance.

Compliance in the pulmonary arterial system is distributed over the entire arterial system and stands at the basis of the constancy of the RC time. This distribution depends on the number of peripheral vessels, which is ~8–10 times more in the pulmonary system than in the systemic tree.

In the systemic arterial tree the compliance is mainly located in the aorta (80% of total compliance in thoracic-abdominal aorta).

The constant RC-time in the pulmonary bed results in proportionality of systolic and diastolic with mean pressure and, in turn, in the constant ratio of oscillatory and mean power.



Physiologie cardiaque appliquée aux cardiopathies congénitales

Partie 5

Adaptation du myocarde à la contrainte en volume et en pression

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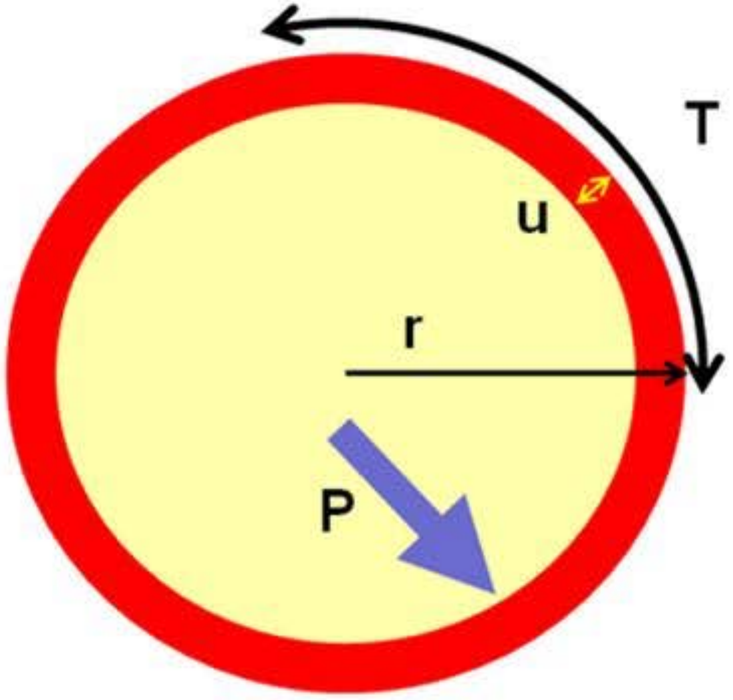
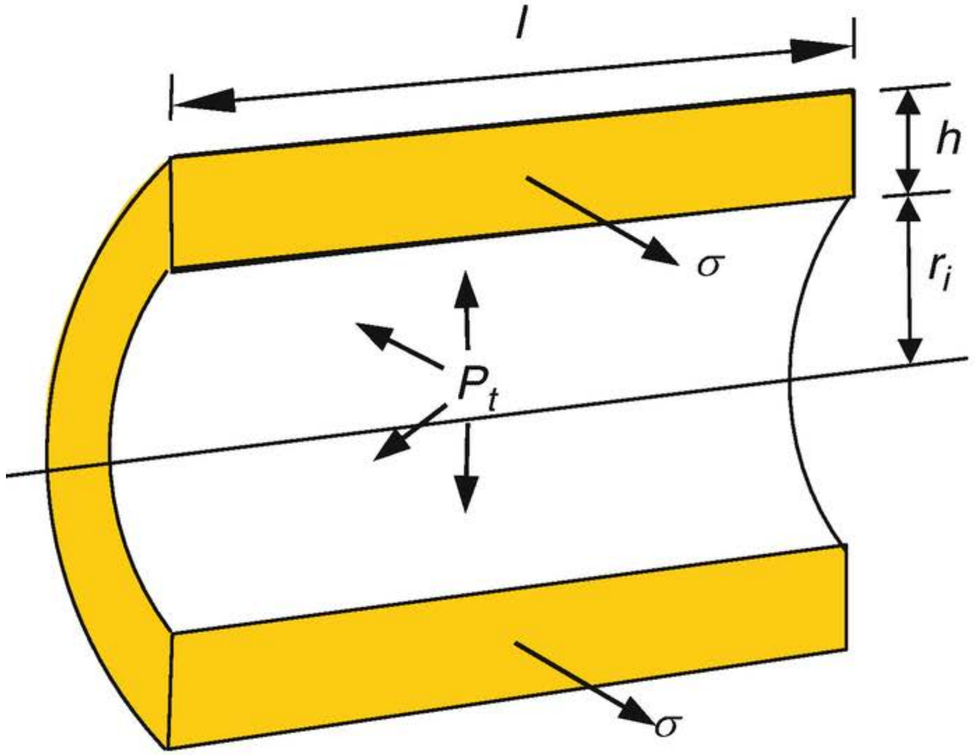
Centre de Référence Maladies Rares
Malformations Cardiaques Congénitales Complexes-M3C
Centre de Référence Maladies Rares
Maladies Cardiaques Héréditaires- CARDIOGEN

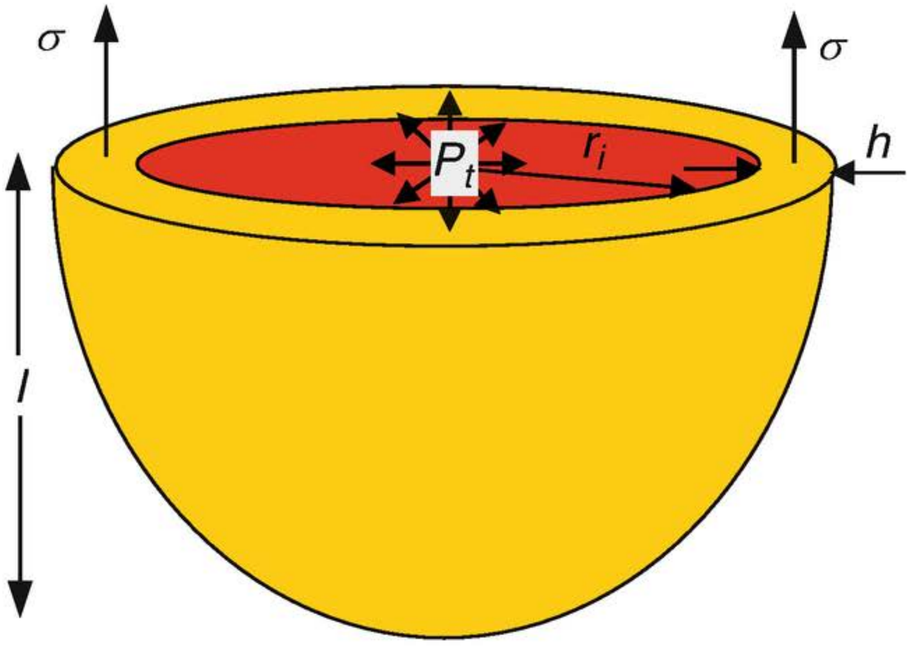
LaPlace's Law



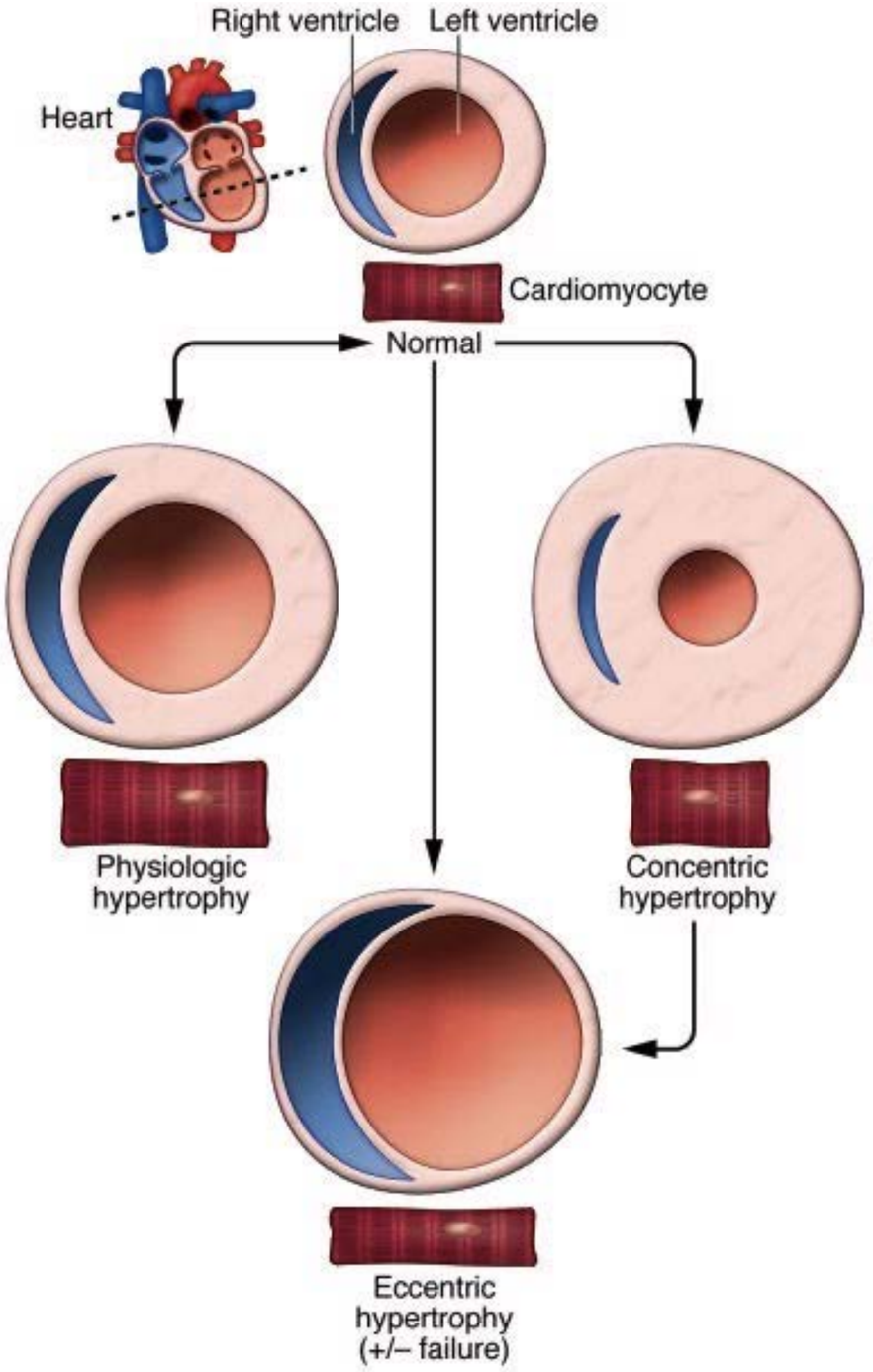
$$\text{Wall Tension (T)} = \frac{\text{Transmural Pressure (P)} \times \text{Radius (r)}}{2 \times \text{Wall Thickness (u)}}$$

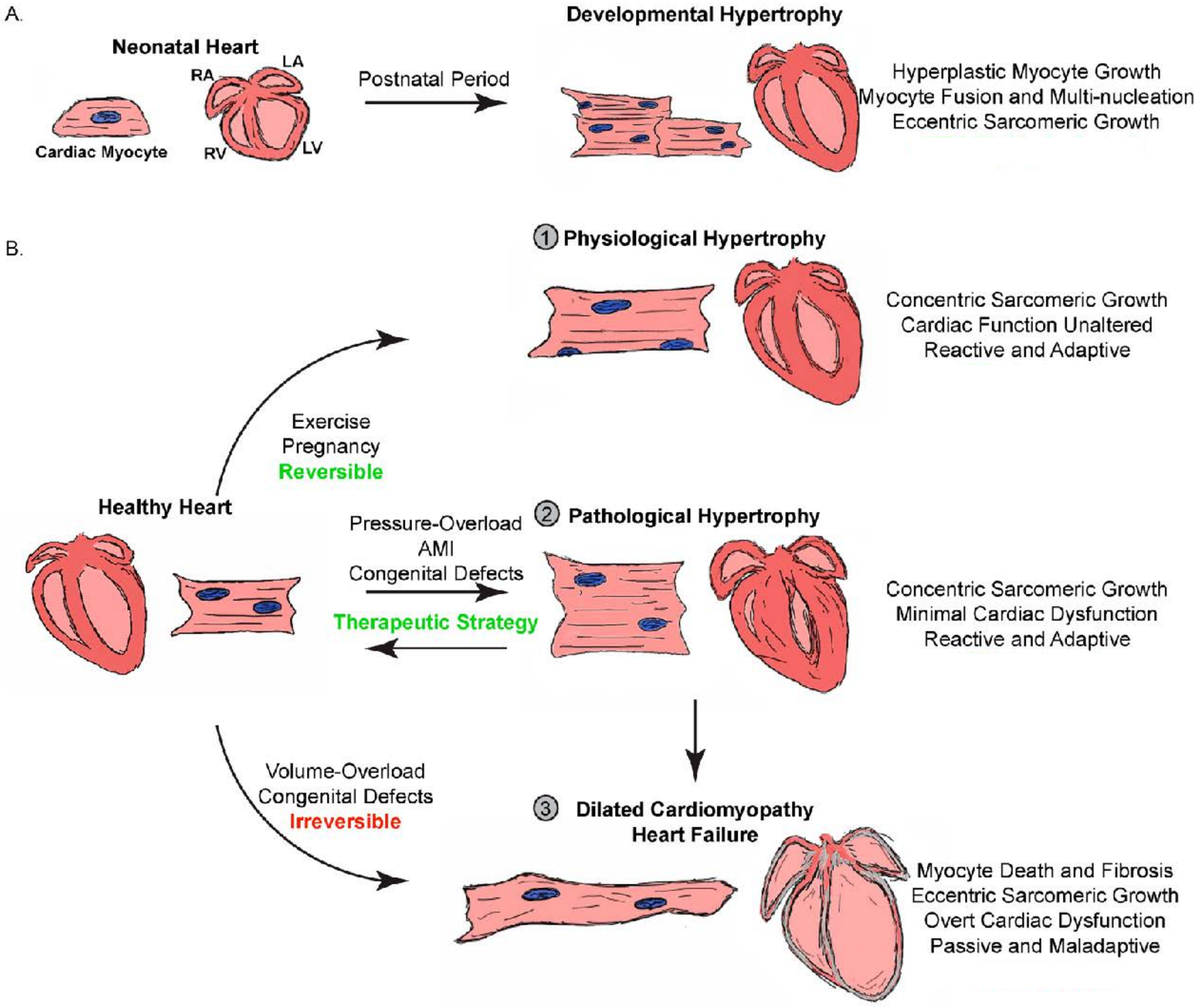


Wall stress $\sigma = P_t r_i / h$



Wall stress $\sigma = P_t r_i / (2h)$


Right ventricle
Left ventricle
Heart
Cardiomyocyte
Normal
Physiologic hypertrophy
Concentric hypertrophy
Eccentric hypertrophy (+/– failure)


A.
Neonatal Heart
RA
LA
RV
LV
Cardiac Myocyte
Postnatal Period
Developmental Hypertrophy
Hyperplastic Myocyte Growth
Myocyte Fusion and Multi-nucleation
Eccentric Sarcomeric Growth
B.
1 Physiological Hypertrophy
Concentric Sarcomeric Growth
Cardiac Function Unaltered
Reactive and Adaptive
Exercise
Pregnancy
Reversible
Healthy Heart
Pressure-Overload
AMI
Congenital Defects
Therapeutic Strategy
2 Pathological Hypertrophy
Concentric Sarcomeric Growth
Minimal Cardiac Dysfunction
Reactive and Adaptive
Volume-Overload
Congenital Defects
Irreversible
3 Dilated Cardiomyopathy
Heart Failure
Myocyte Death and Fibrosis
Eccentric Sarcomeric Growth
Overt Cardiac Dysfunction
Passive and Maladaptive


Thank you
M3C
Collective ignorance is the motivation
Curiosity is the strength
Research is the path
Individual experience is the brake
Indifference is the weakness
Authority argument is the threat