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# Physiologie de la DCPT

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Seminaire 2010

# Plan du topo

- ” Rappel de la Physiologie du Coeur normal à 2 ventricules
- ” Physiologie Cardiaque en cas de communication large entre les Ventricules ou les oreillettes
- ” La Défaillance Cardiaque dans une circulation en série
  - . Défaillance Cardiaque Gauche et Droite
- ” La Défaillance Cardiaque en cas de shunt Auriculaire
- ” La défaillance Cardiaque
  - . En cas de fuite pulmonaire libre
  - . Dans les Dérivations Cavo -Pulmonaires Totales

# Biologie de la Circulation Normale et Pathologique

- ” **La perfusion:**  $\Delta P$  (Ao- VC) = Qs x RVs
  - . Vasoconstriction pour maintenir Pr en cas de   Q
- ” **L'oxygénation:** VO<sub>2</sub> = Qs x (Ao-VC) O<sub>2</sub>
  - . Extraction pour compenser   Q et éviter Acidose
- ” **La congestion:** exsudation plasmatique LEC et lymphatiques débordés: Oedème
  - . ) dème pulmonaire pour Pr Cap > 25mmHg
  - . ) dème Cave et Portal pour Pr veineuse >10 à 15mmHg

# Contrôle de la Circulation

## Hiérarchie de priorité et *Moyens*

- ” Le cœur ne décide pas, il exécute
- ” Le contrôle de la circulation donne **priorité**
  - 1/ la perfusion au dépens de la congestion
  - 2/ la Pr Ao au dépens du Qs
- ” C'est un contrôle **Neuro-humoral** qui, en agissant sur le cœur et sur les conditions de charge assure ces demandes et priorités

# Role de la Circulation

## Hiérarchie de priorité et Moyens

C'est un contrôle **Neuro-humoral** qui agit:

” **Sur les vaisseaux**

- . Répartition du débit par vasoconstriction et vasodilatation
- . mobilisation du retour veineux

” **Sur le Cœur par Stimulation Sympathique**

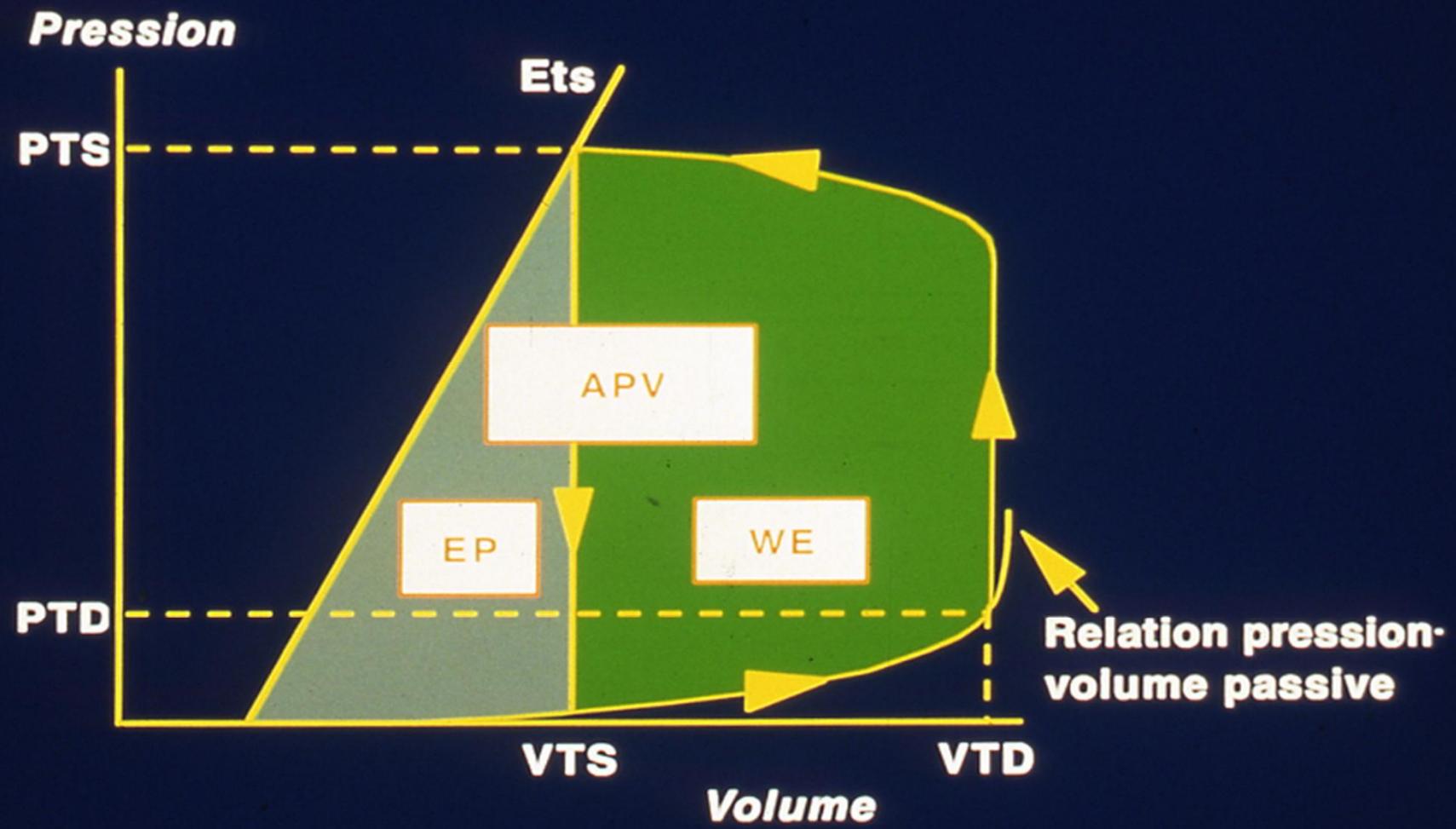
- . F. C. Tachycardie, Contractilité ..

” **Le prix à payer de la Stimulation Sympathique et de la Dilatation du Coeur est élevé**

# Les grands principes de la circulation

- “ **La pompe cardiaque ne décide pas**, elle essaye de répondre aux demandes de la circulation
- “ Cette réponse dépend des **propriétés du cœur** sollicitées par les **conditions de charge** qui sont régulées par le **contrôle neuro humorale**
- “ Le principe de base est basé sur le « **Starling** » c'est à dire « **se remplir pour se vider** »
- “ **Les Courbes Pr/Vol** illustrent bien l'ensemble propriétés myocardiques et conditions de charge

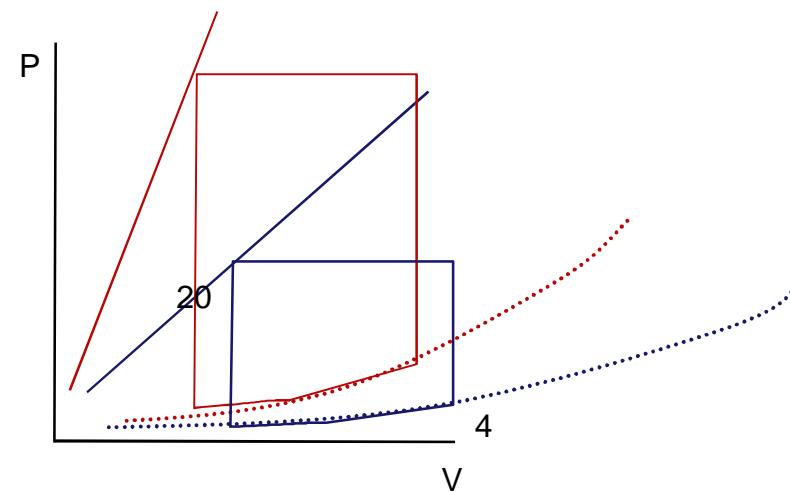
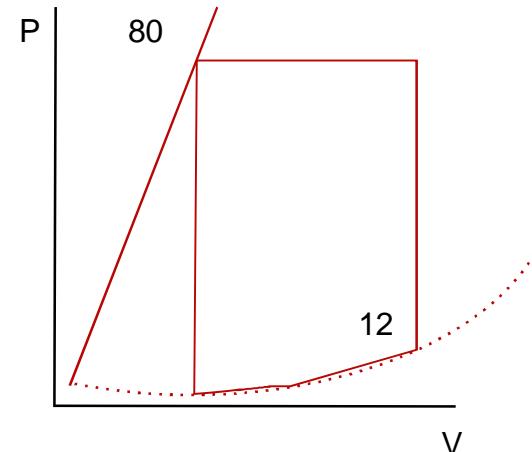
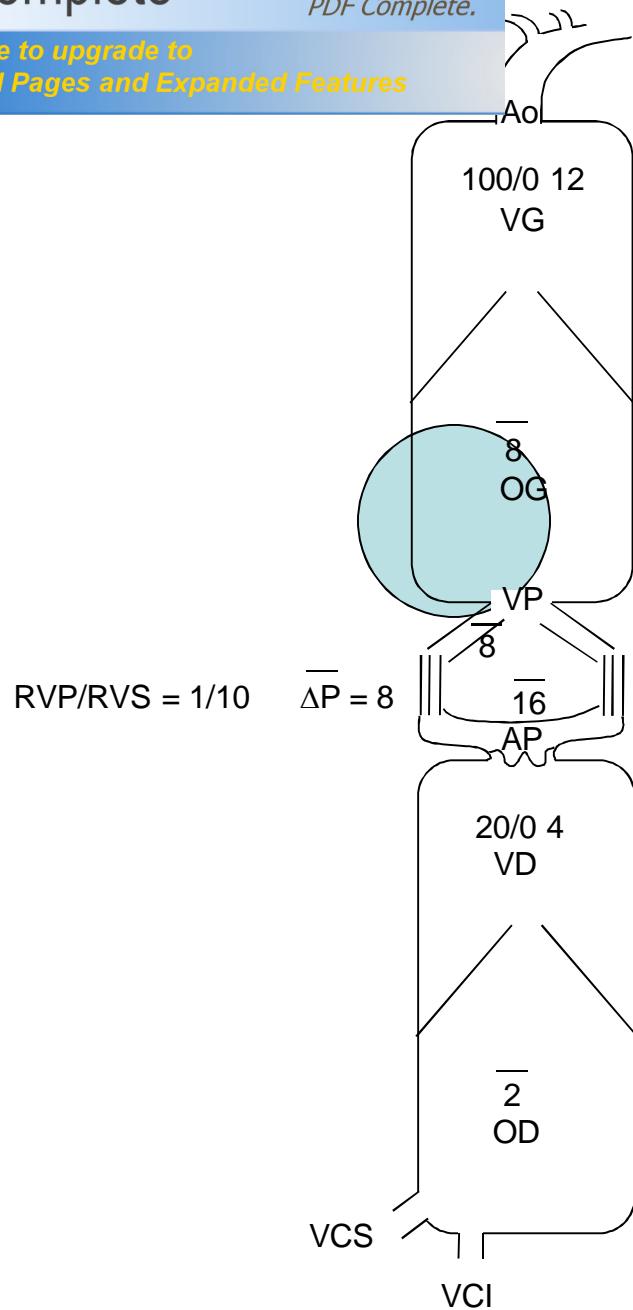
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## C%ur en Série:

Indépendance *relative* des circulations Gauche et Droite

### “ La défaillance Gauche affecte

- . En aval le Qs et la Pr Ao
- . En amont les Pr Cap Pulm (*post charge VD*)

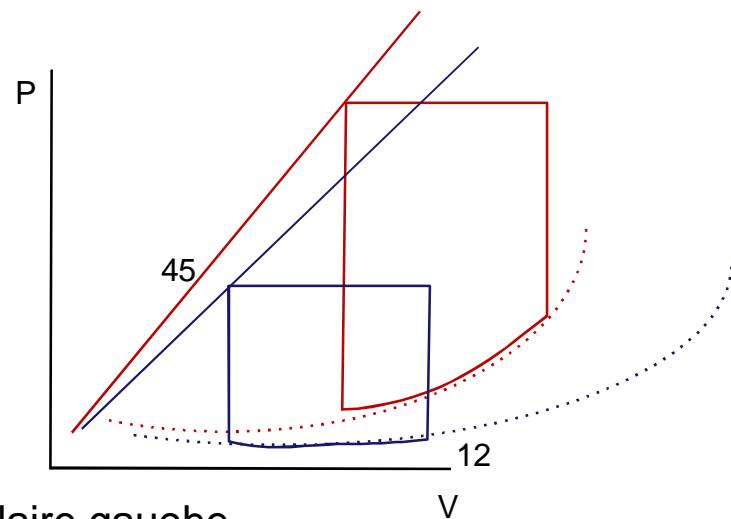
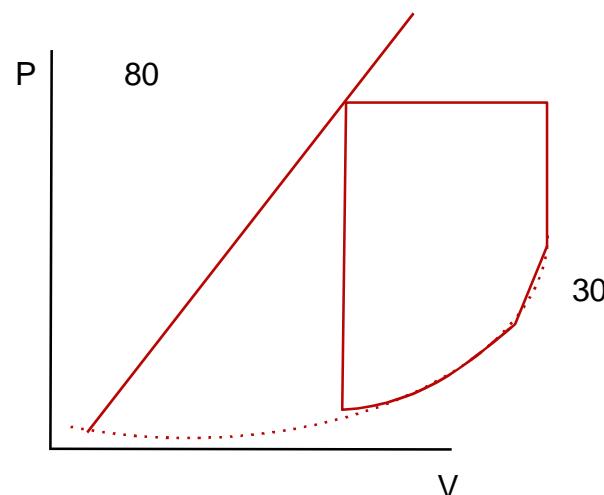
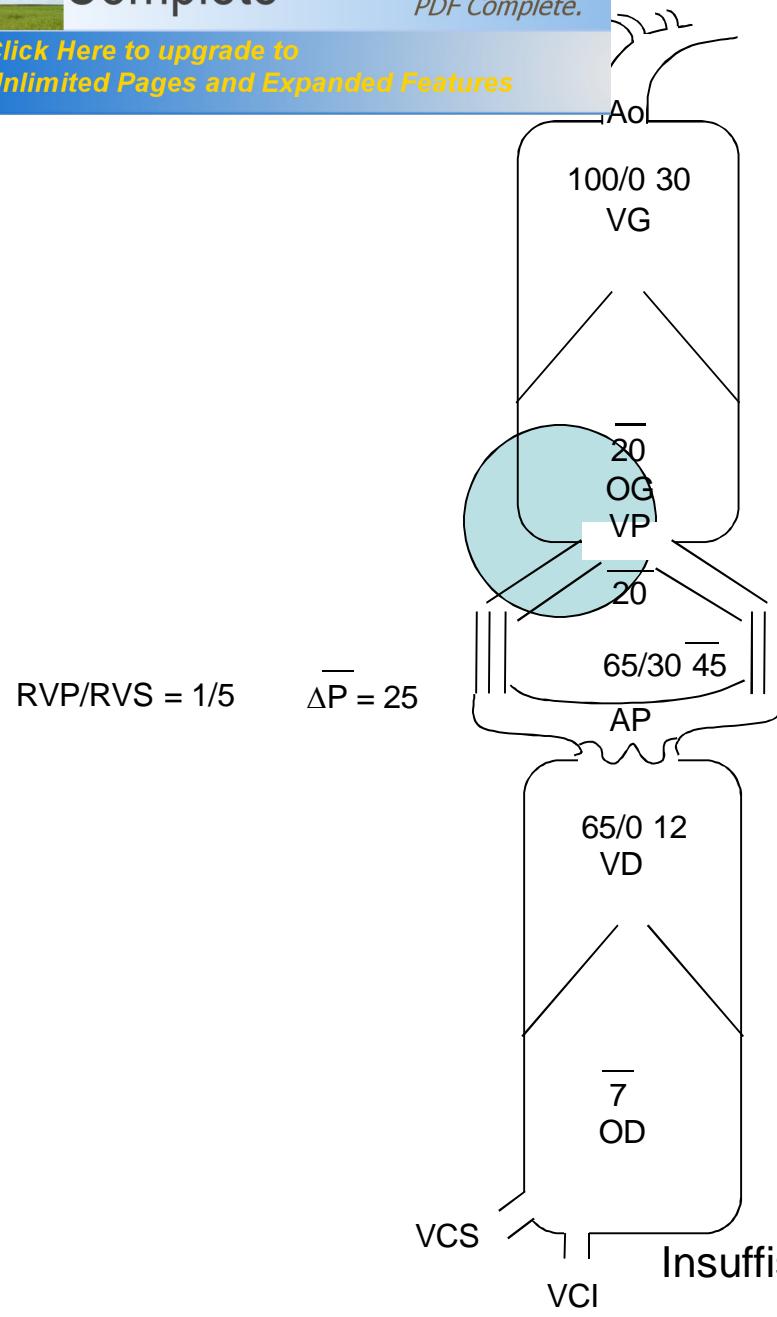
### “ La défaillance Droite affecte

- . En aval le QP(=QS) et la Pr AP (*précharge VG*)
- . En amont les Pr Caves

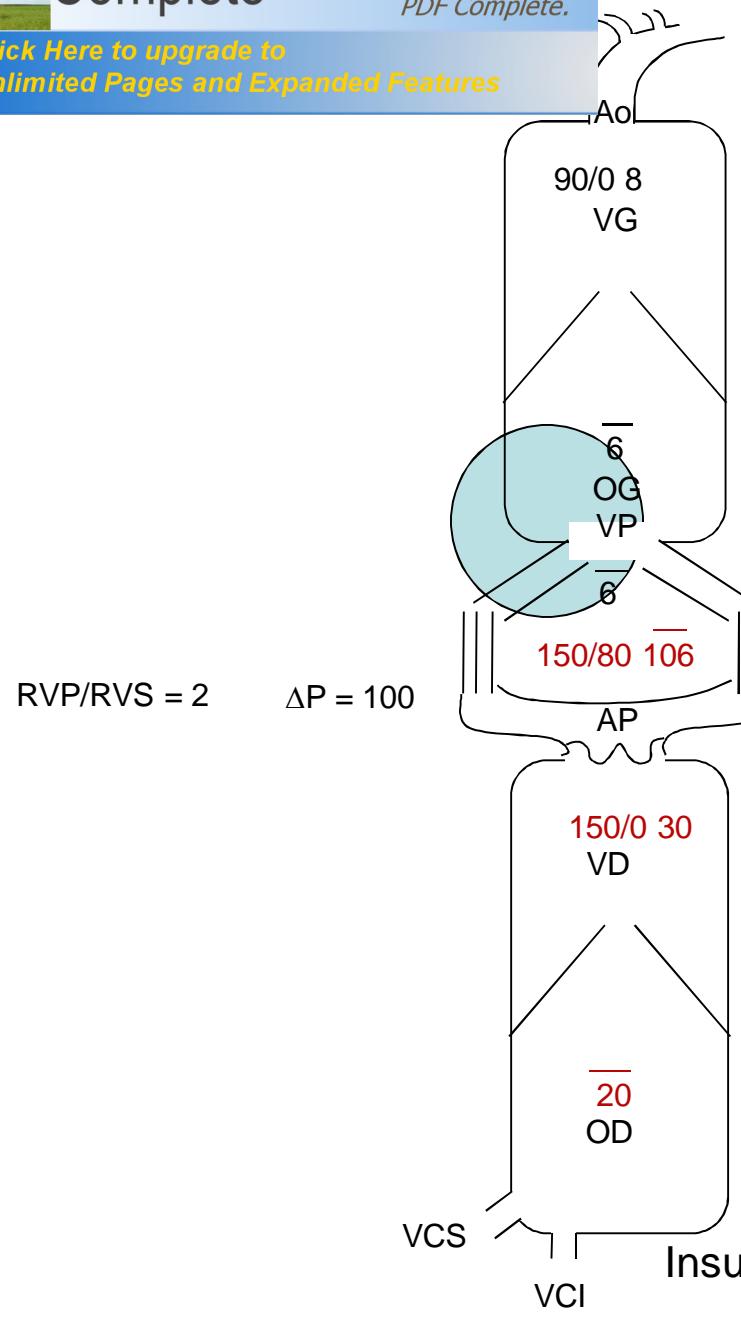


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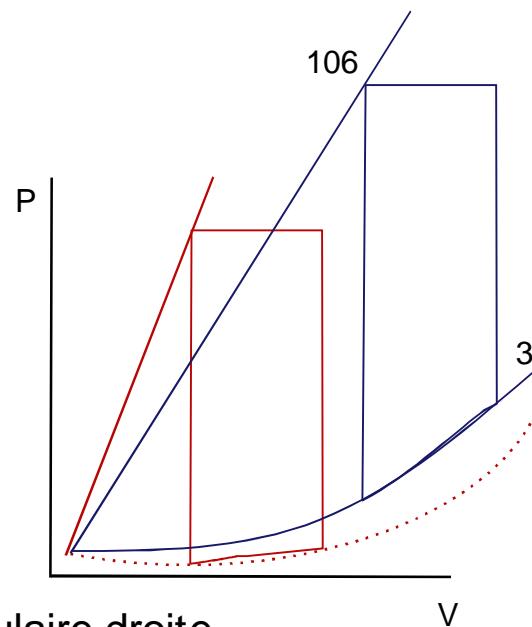
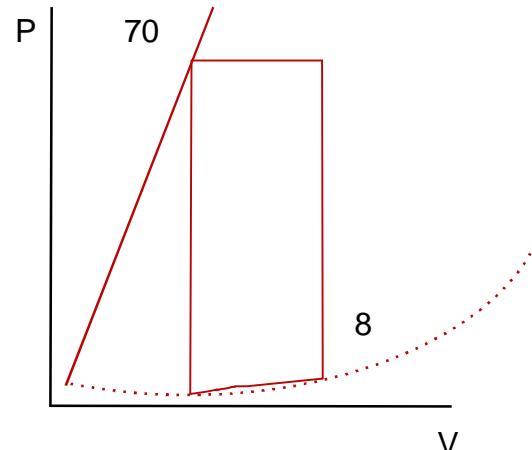
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HTAP primitive

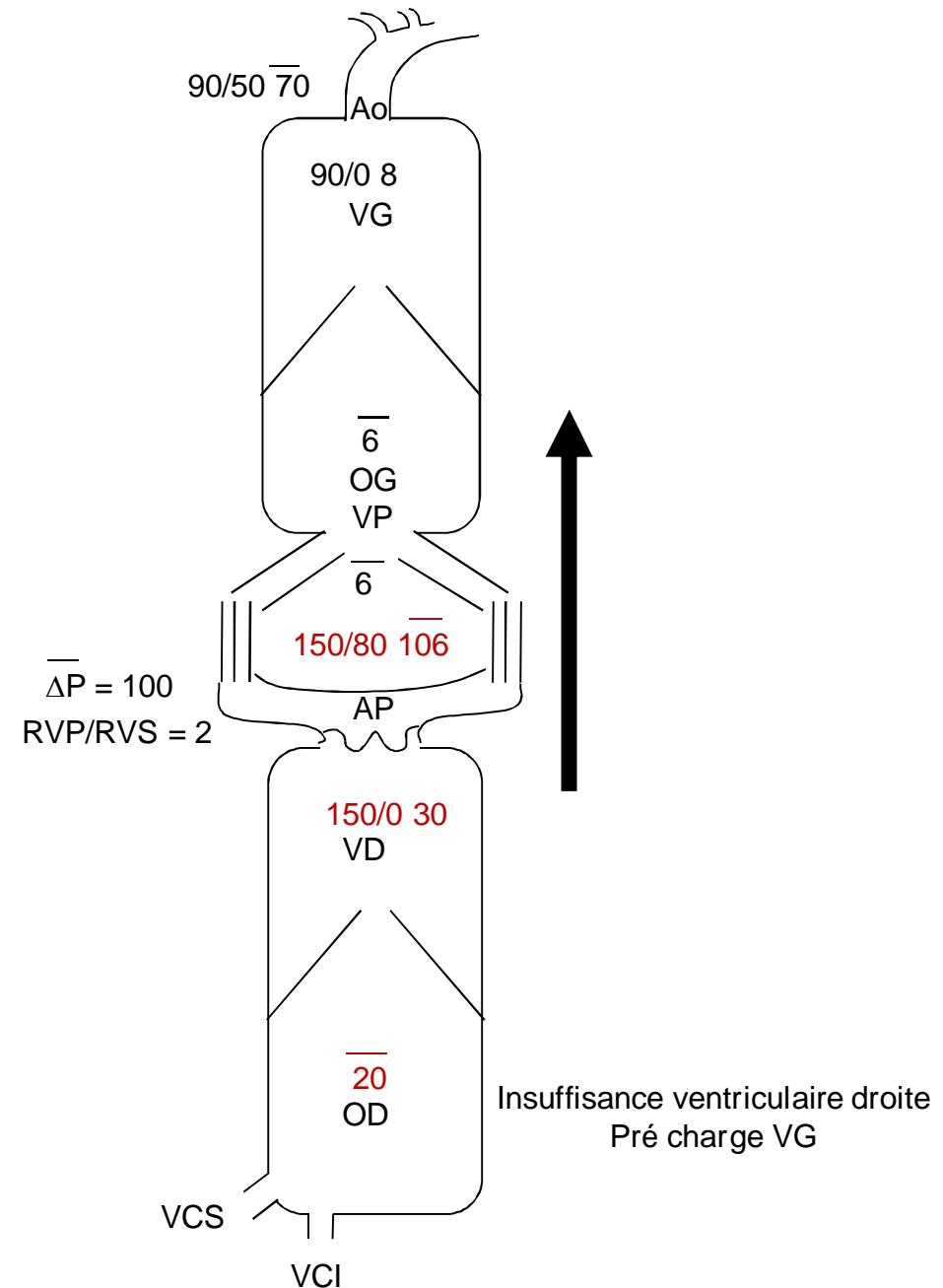
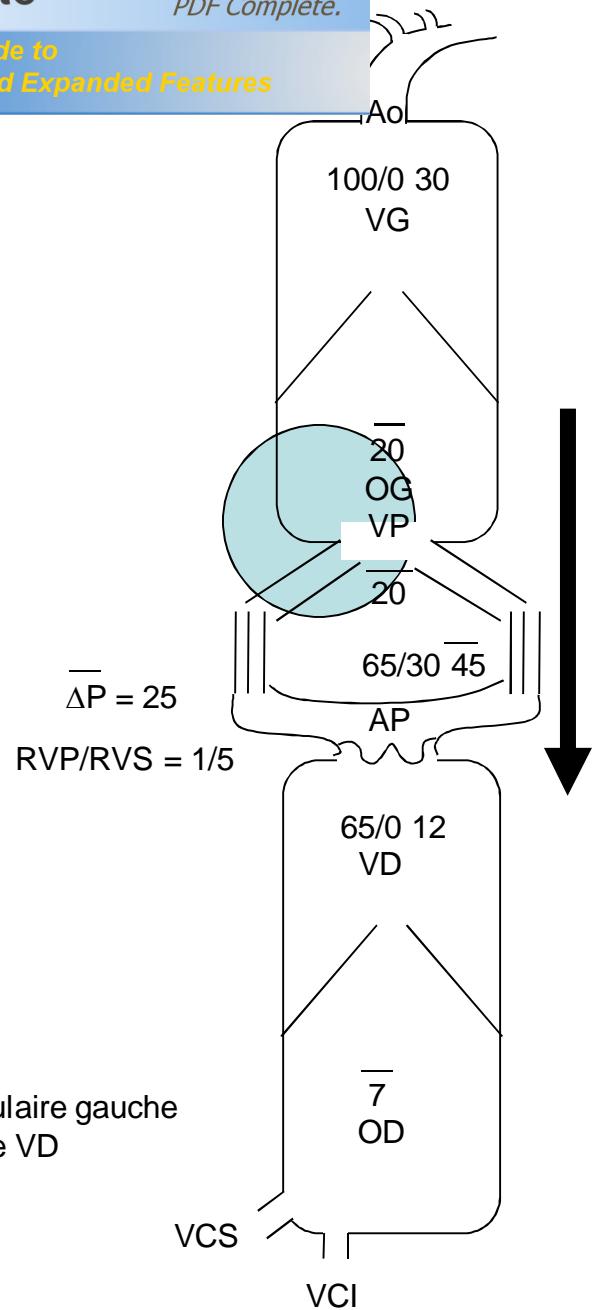


Insuffisance ventriculaire droite  
Pré charge VG



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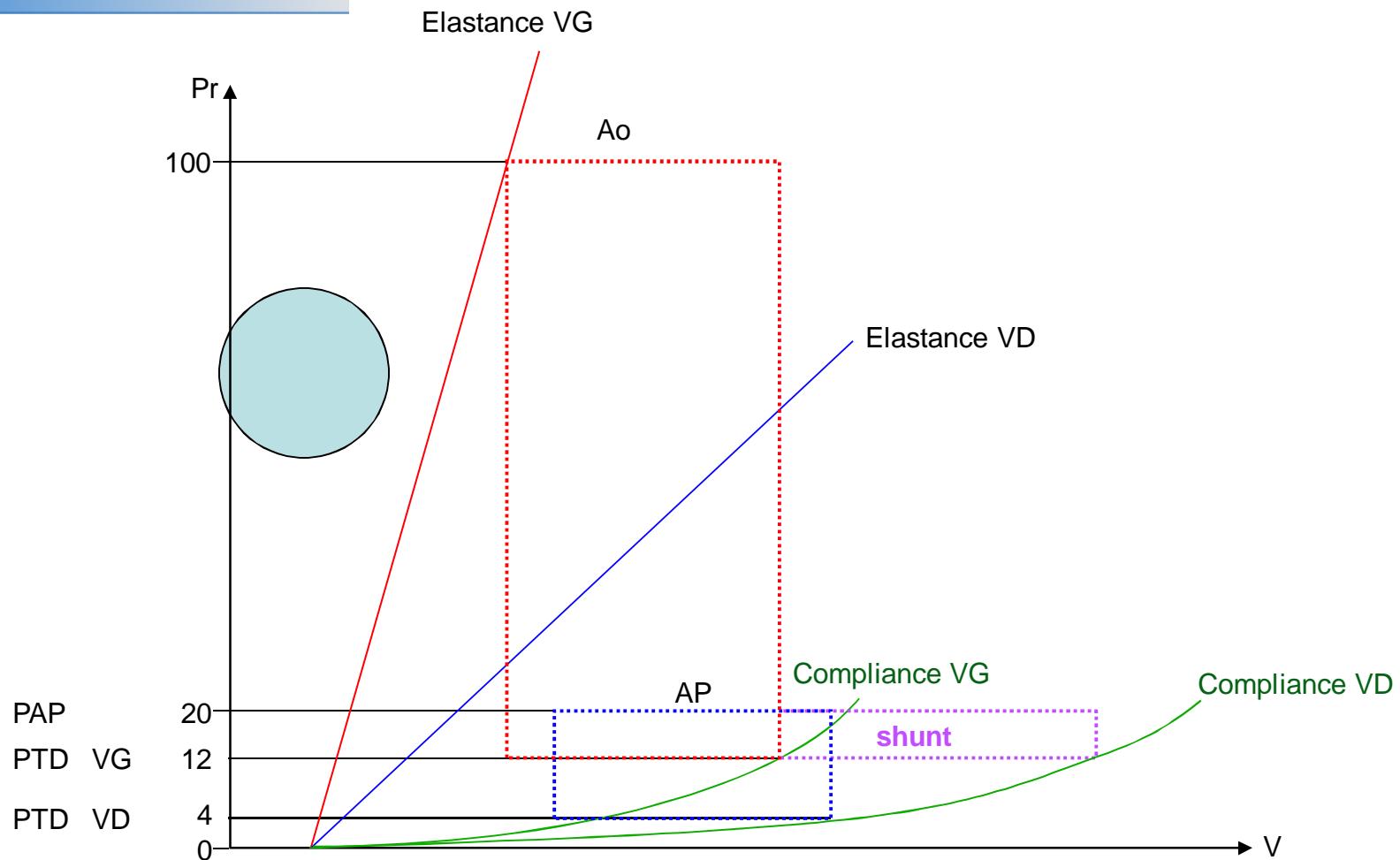
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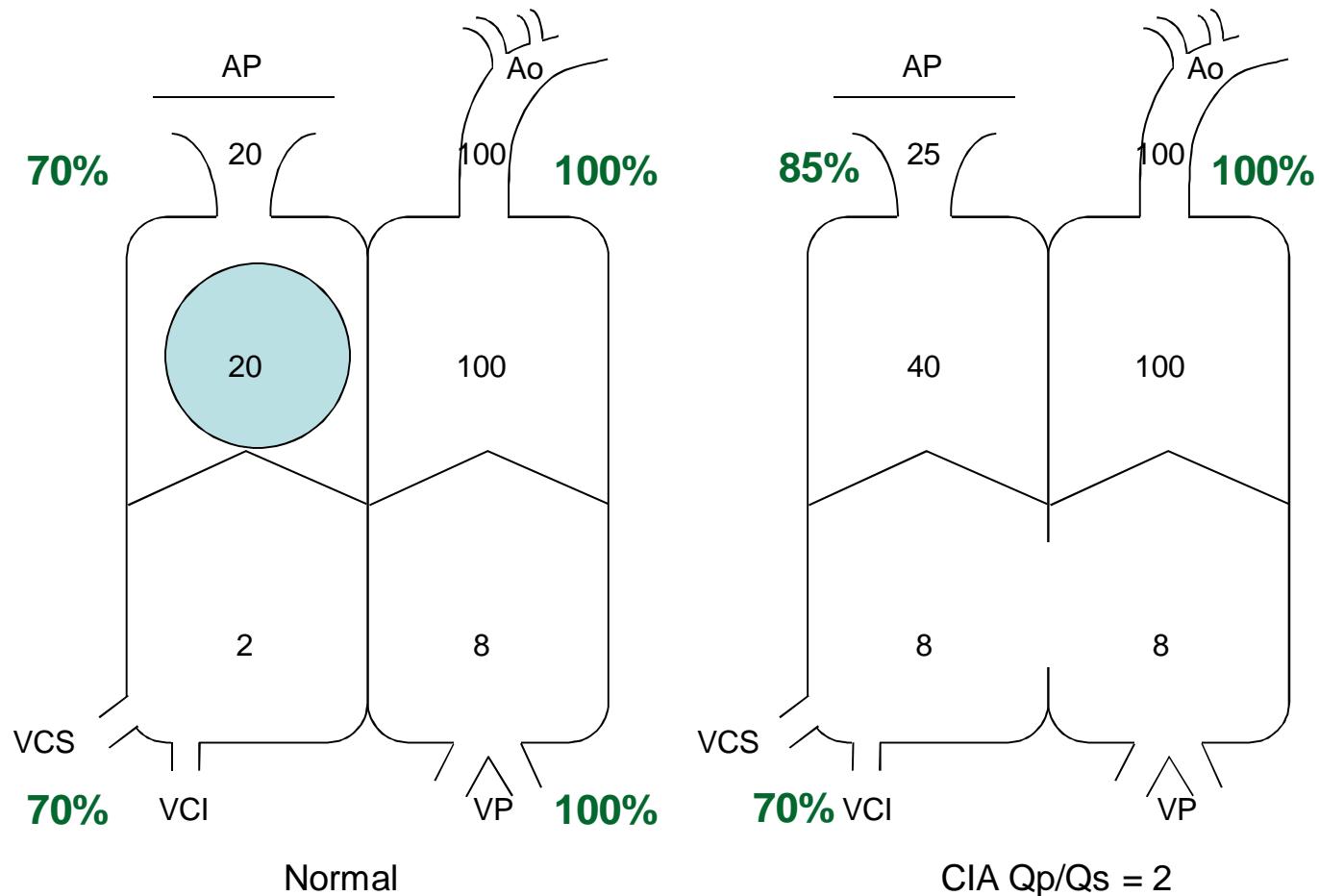
# iologie et pathologie en cas de large CIA

- ” La CIA égalise les Pr dans les oreillet tes, et si les VAV sont normales, les Pr sont les mêmes en diastole dans toute les cavité cardiaques
- ” C'est la Pr Gauche (VG en diastole) qui fixe le niveau des Pr de remplissage et l'hypertension cave reflète la fonction du VG
- ” Le VD se remplit à la Pr du VG (c'est la Pr qui est imposée et le Vol du VD qui varie en fonction de la compliance)

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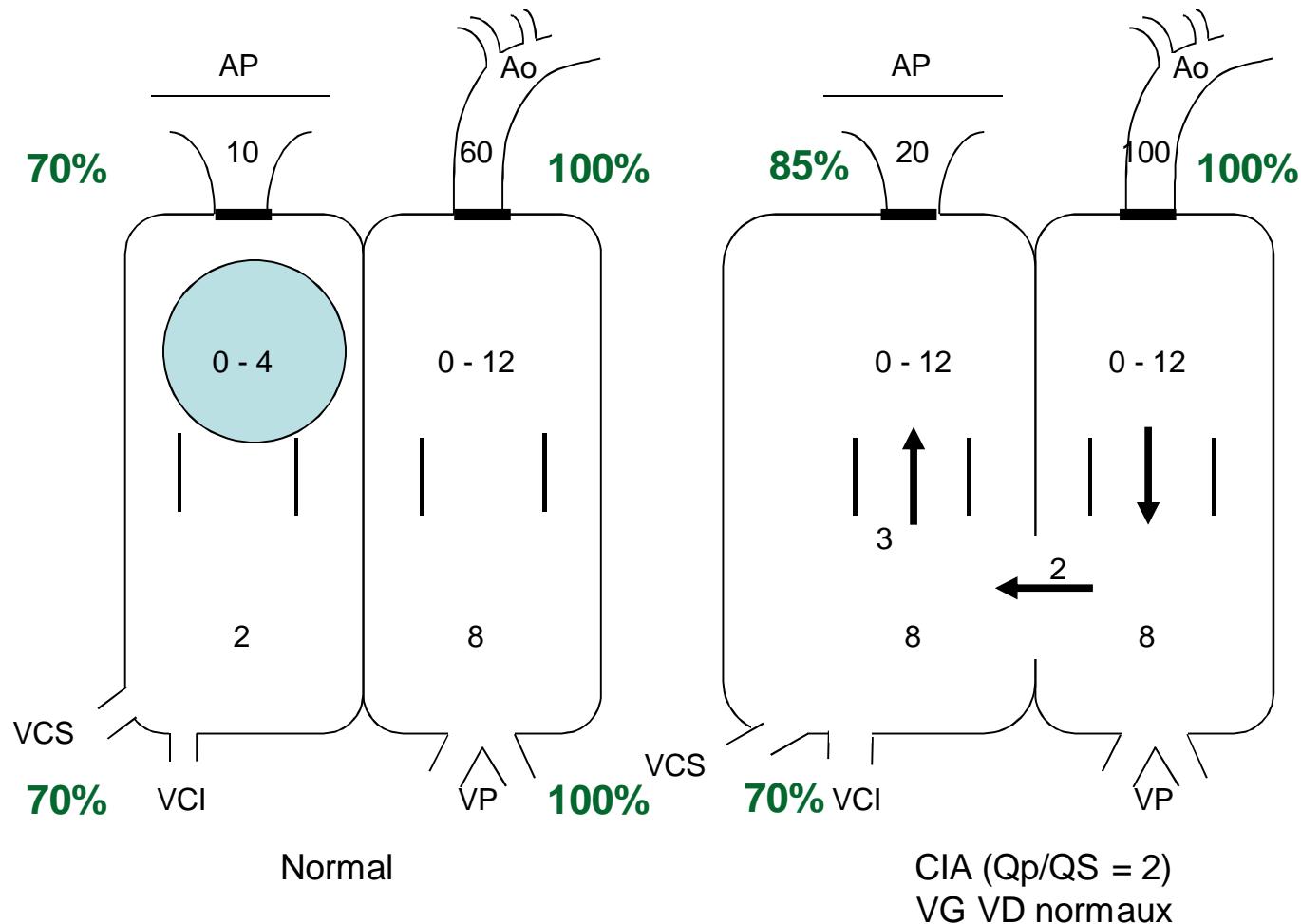


### Systole

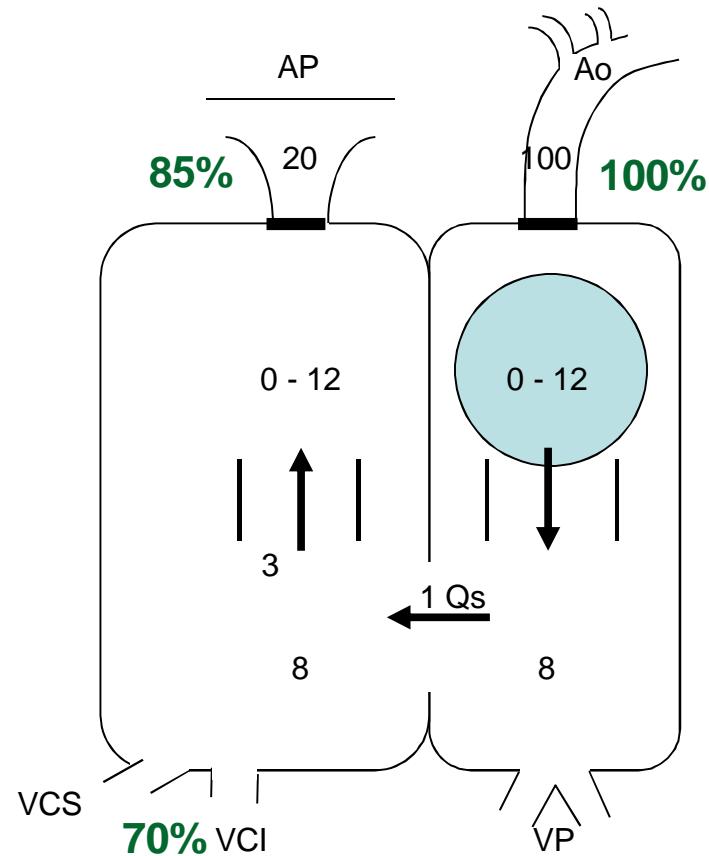


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### Diastole

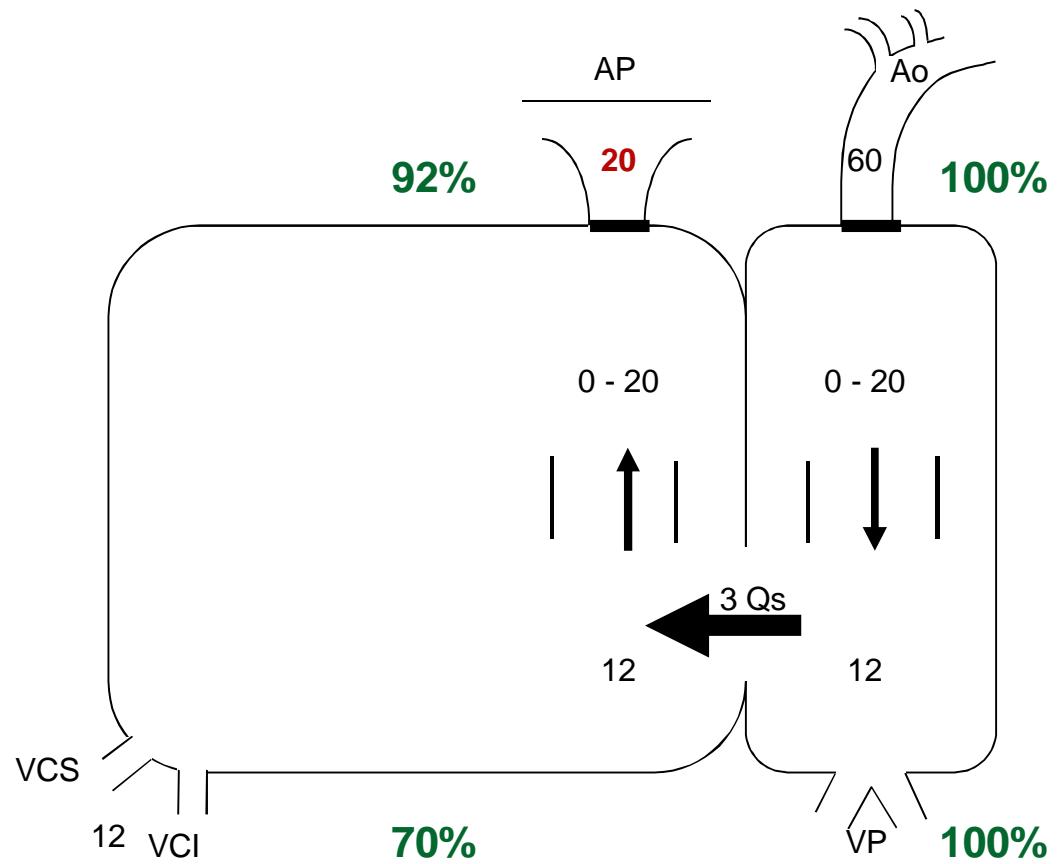


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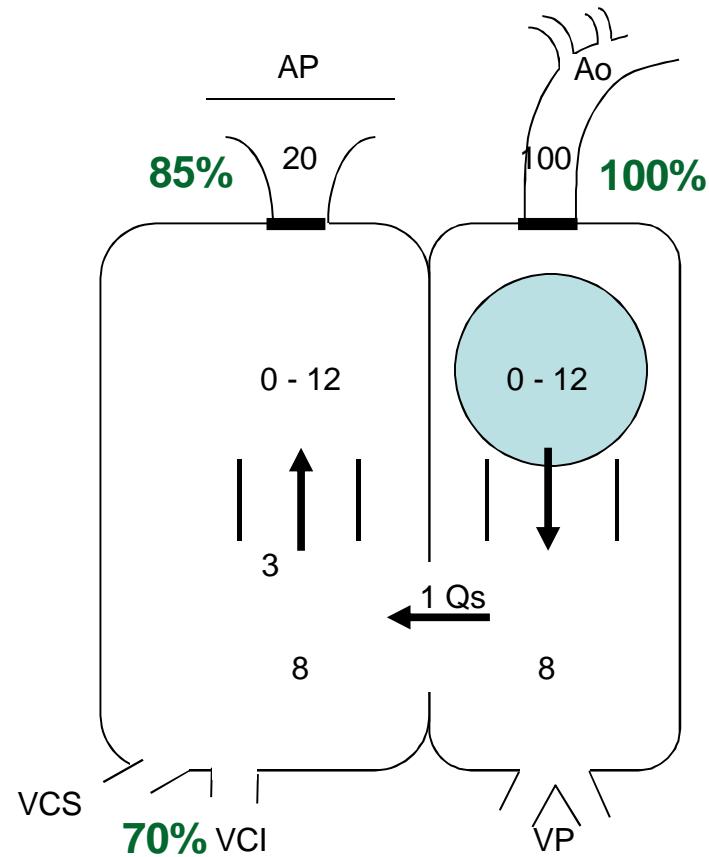
CIA (Qp/Qs = 2)  
VG VD normaux

Diastole

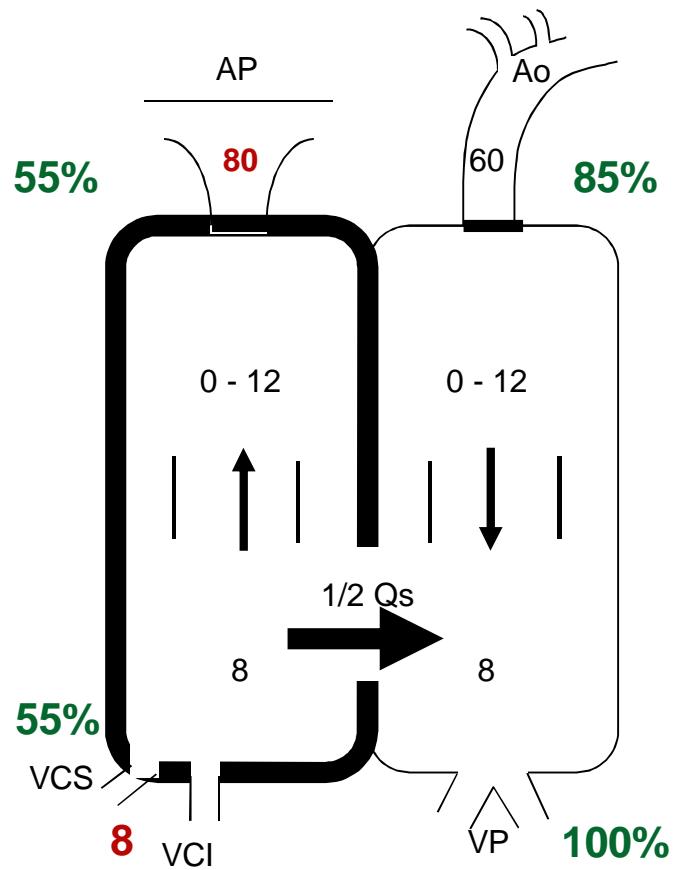


CIA avec défaillance VG

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Diastole

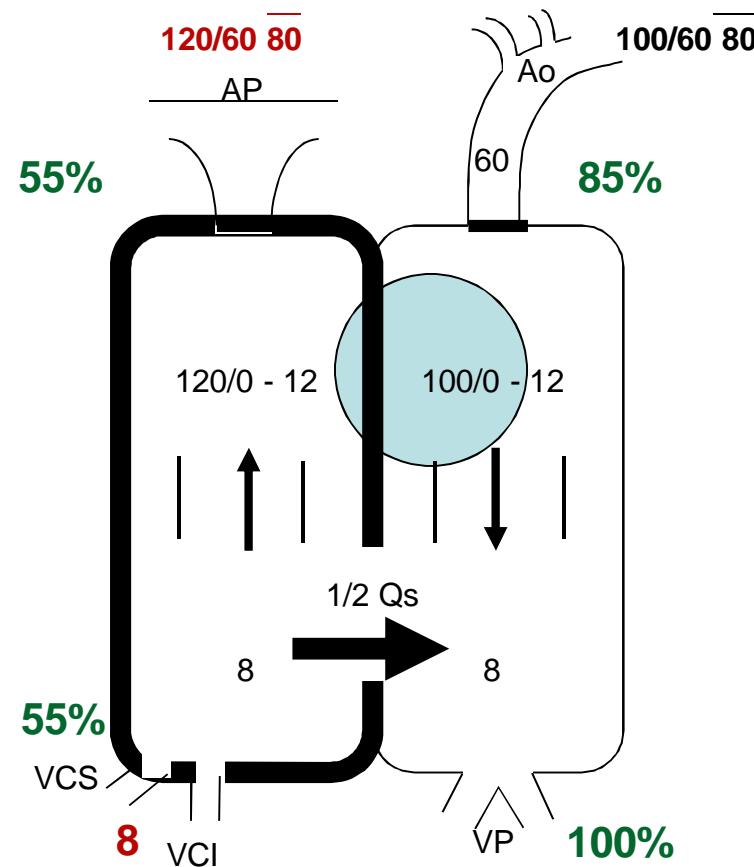


CIA (Qp/QS = 2)  
VG VD normaux

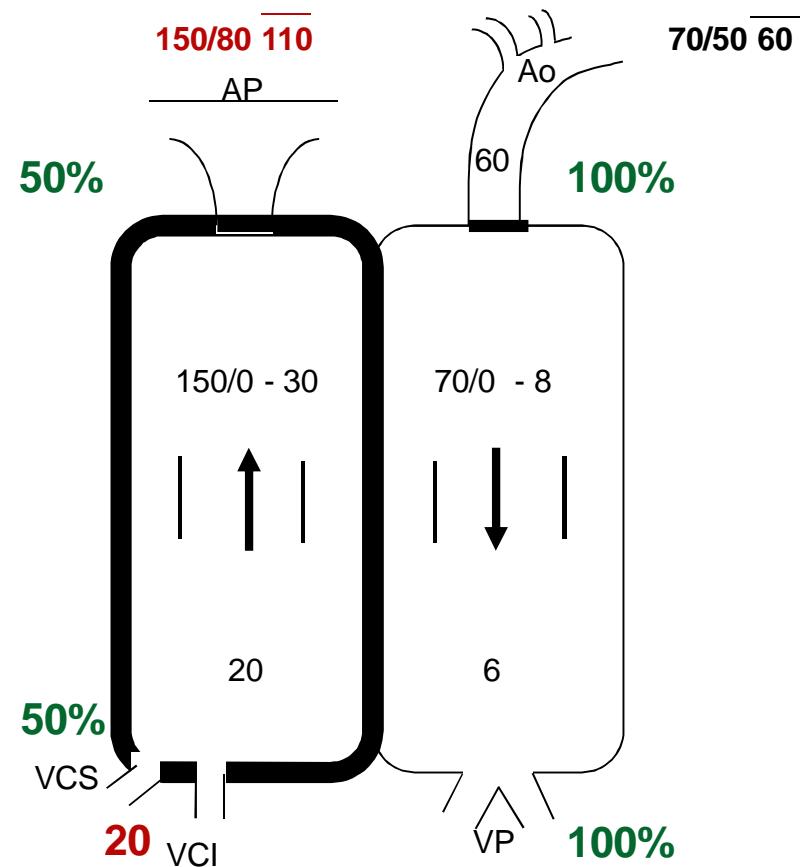
CIA avec défaillance VD  
HTAP primitive

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## Systole et Diastole



Défaillance VD  
HTAP primitive avec CIA



Défaillance VD  
HTAP primitive sans CIA

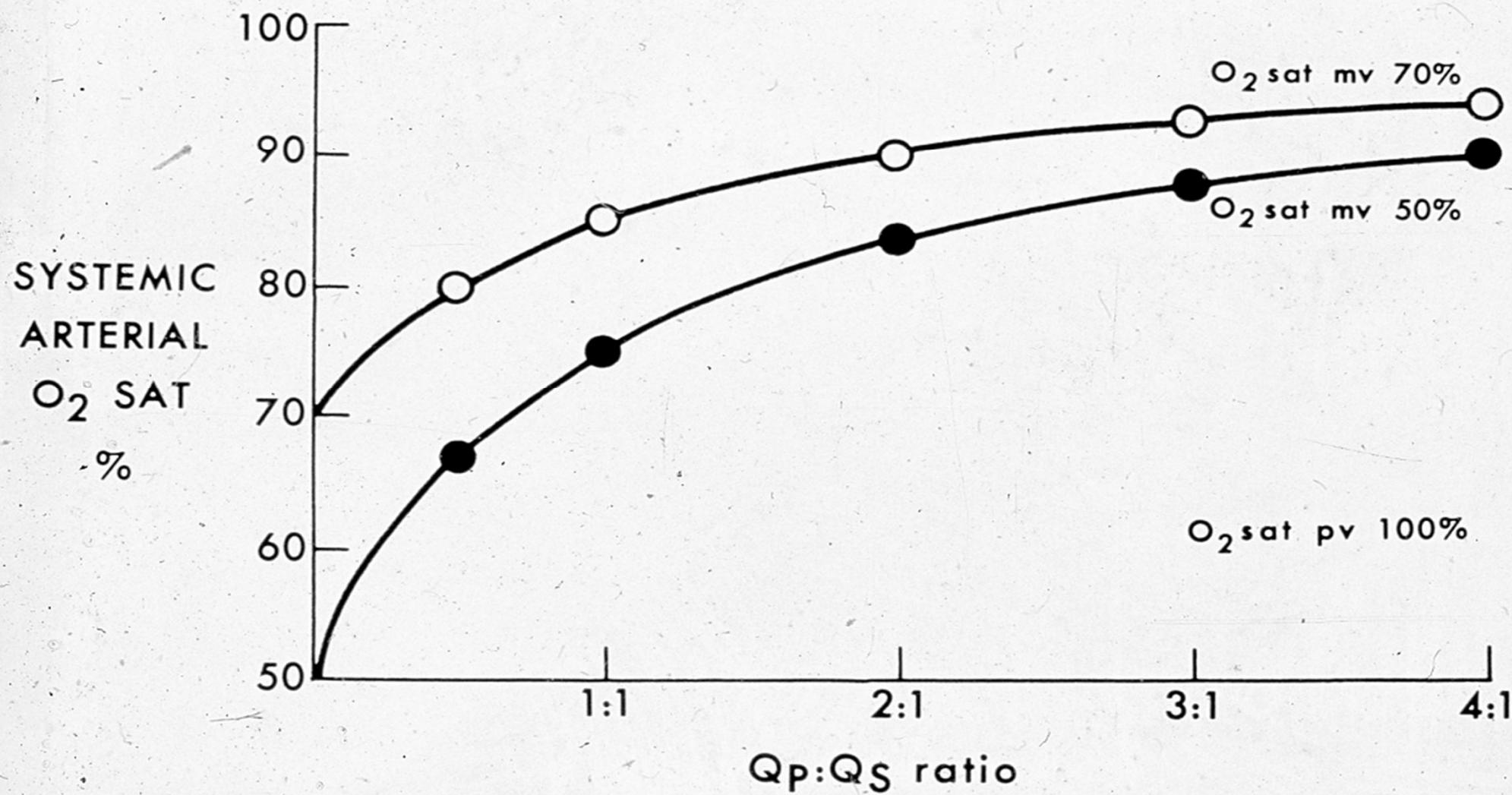
# Pathologie en cas de CIV large

- ” Les pressions se égalisent en systole et diastole au niveau des Pr du Ventricule sous Ao
- ” La Pr Systolique de l'AP est fonction d'un éventuel obstacle Pulmonaire et le Flux (Qp) est fonction de la Pr AP et des RVP (artéries)
- ” Les conditions de charge sont fonction du débit « gaché »
  - . Le sang rouge qui retourne à l'AP
  - . Les fuites des Valves AV ou Sigmoïde
- ” L'hypoxémie n'est présente que si CIV + anomalie

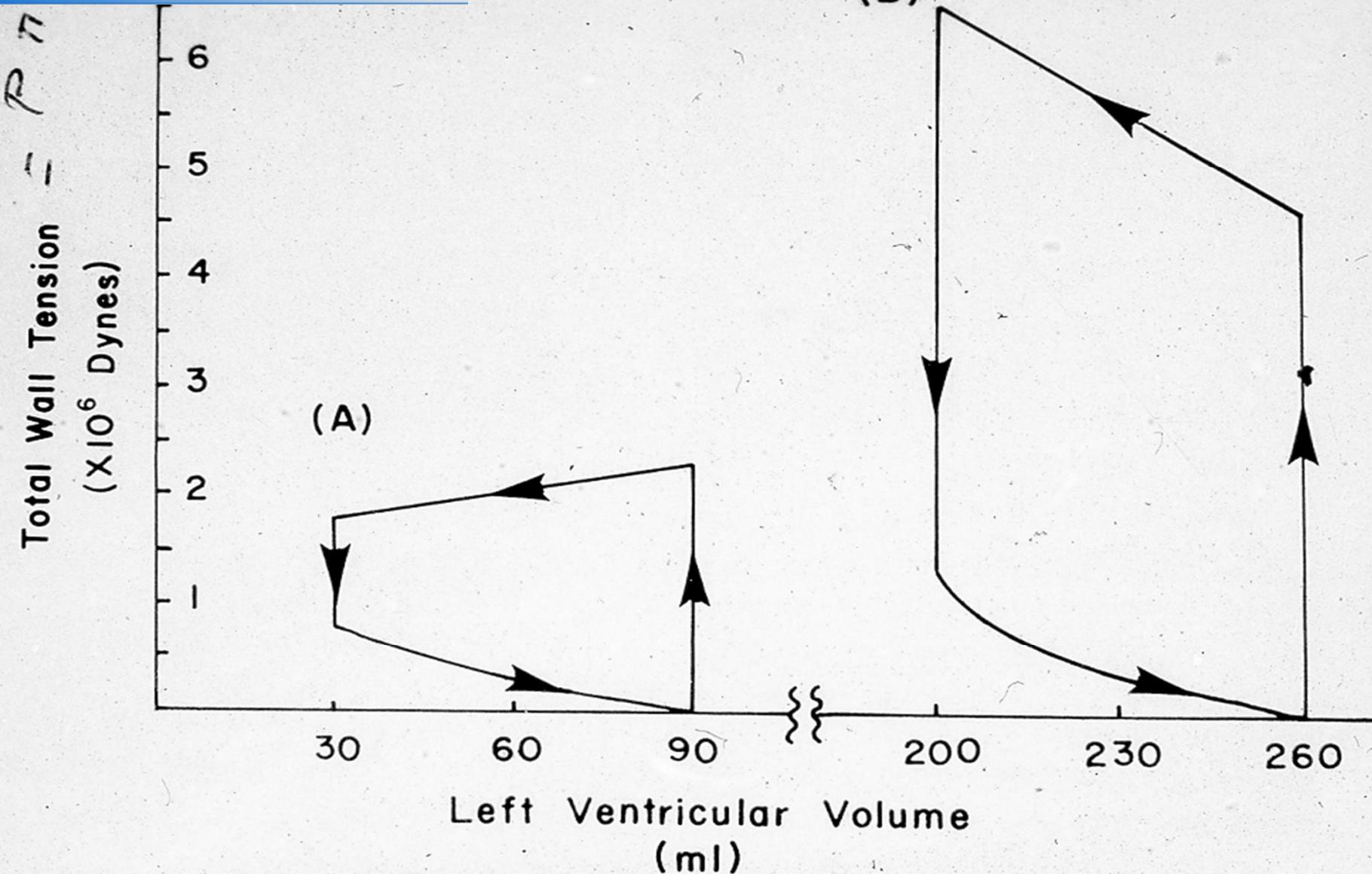
# Physiopathologie du VU sans DCP

- ” Le sang se mélange plus (atrésie d'une valve) ou moins (streaming pattern)
- ” La Cyanose et la surcharge Volumétrique sont inversement reliée (sauf fuite Valvulaire)
- ” Une Saturation acceptable est autour de 90% au repos
  - . Pas de polyglobulie menaçante
  - . Surcharge volumétrique acceptable

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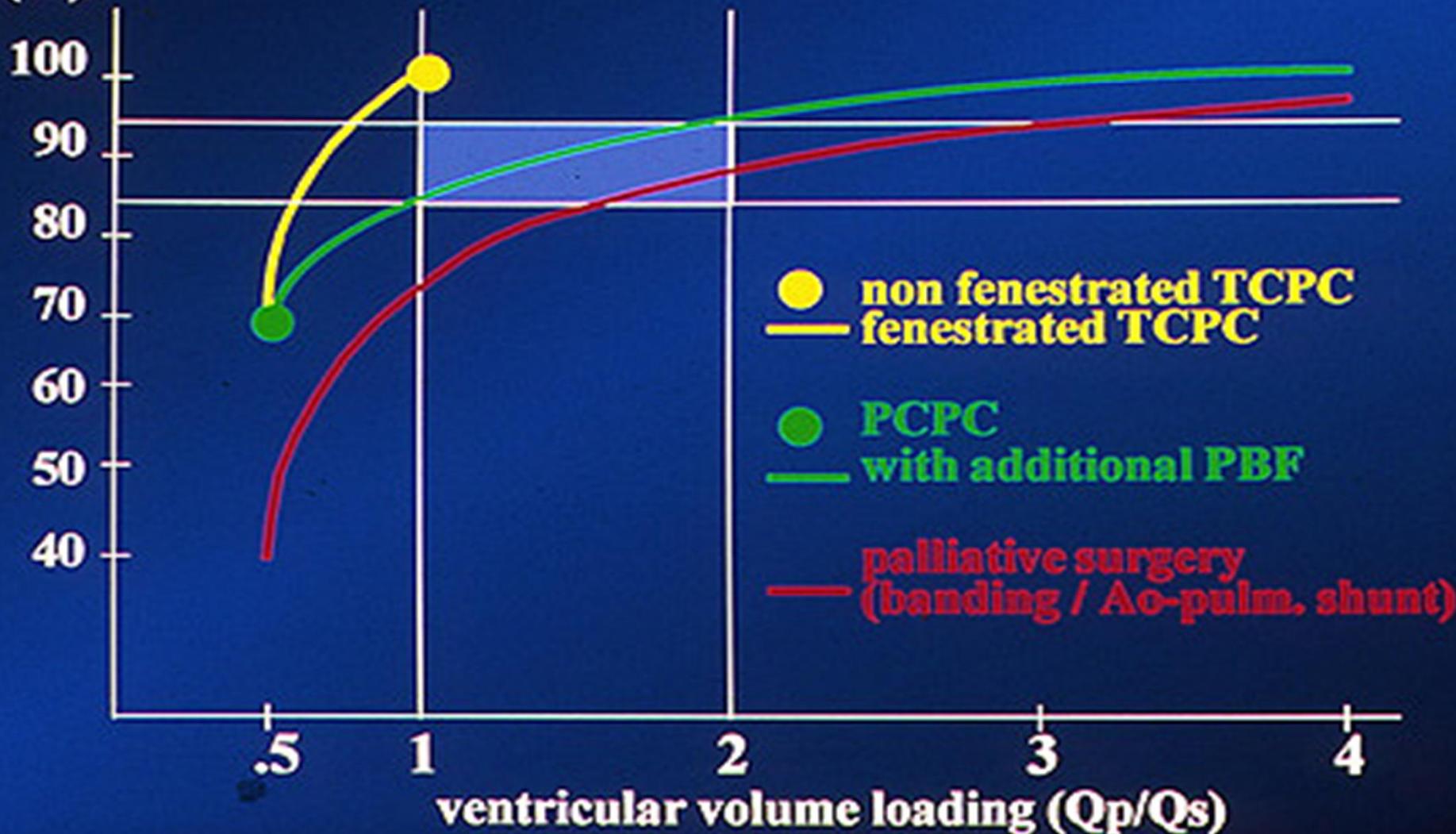


# Physiopathologie du VU avec DCPP

- ” On économise sur la surcharge Volumétrique pour obtenir une saturation satisfaisante grâce au Double salaire
  - . 1/3 du Qs de repos (avec Sat basse est oxygéné sans passer par les ventricules)
  - . Le reste est du sang mélangé
- ” Oui mais, surtout intéressante au repos
- ” Seulement possible pour Pr de remplissage acceptable

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O<sub>2</sub> sat (%)



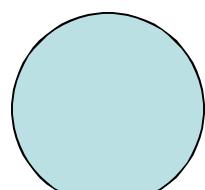
# Série avec IP libre (Fallot)

## Ce qui est imposé

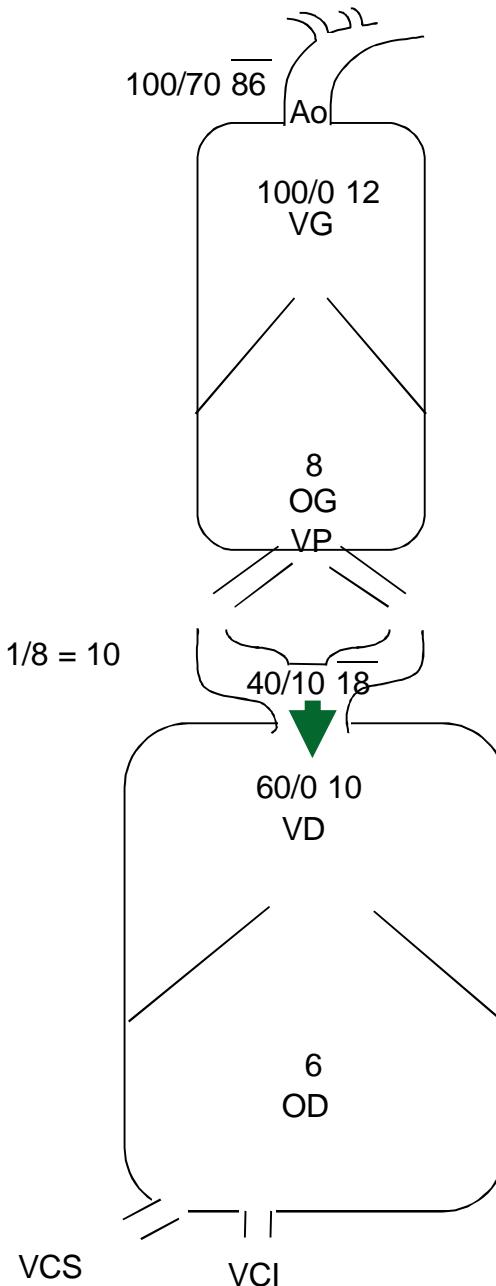
- “ La Pr Ao et le Qs
- “ Les mêmes Pressions Télé (ou méso et télé)  
Diastolique de l'AP aux V Caves (via le Cœur Droit)
- “ Un remplissage du VD par les V Caves qui assure le Qp effectif (Qs) qui remplit l'AP via le VD ou directement en télé diastole à partir de l'oreillette (onde A)

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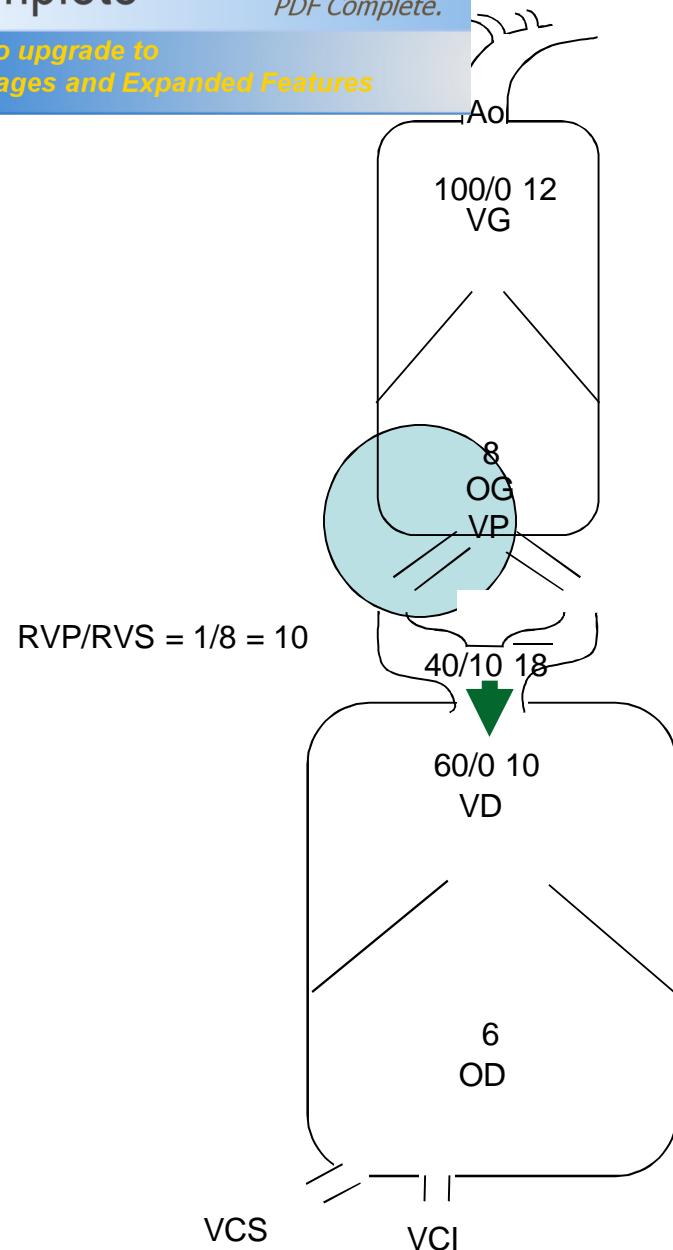
## Fallot fuite pulmonaire



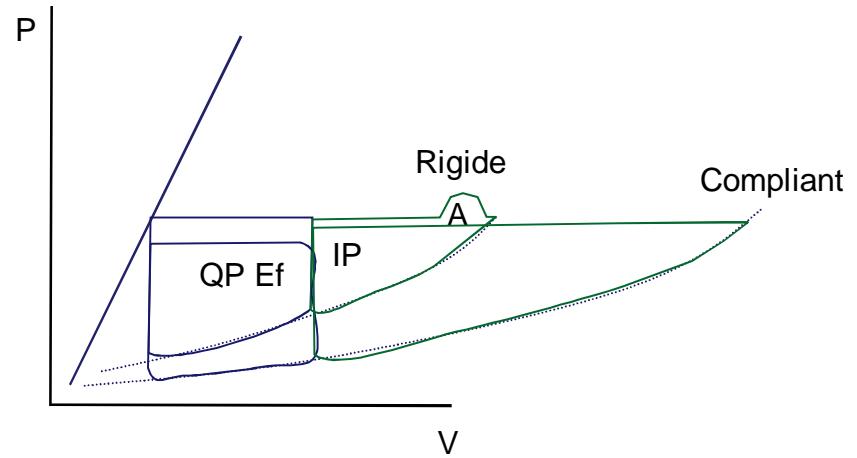
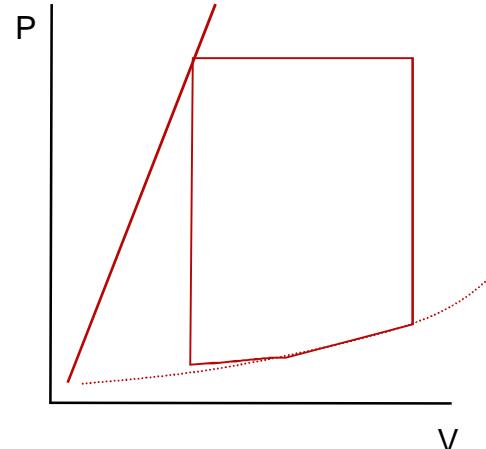
$$\text{RVP/RVS} = 1/8 = 10$$



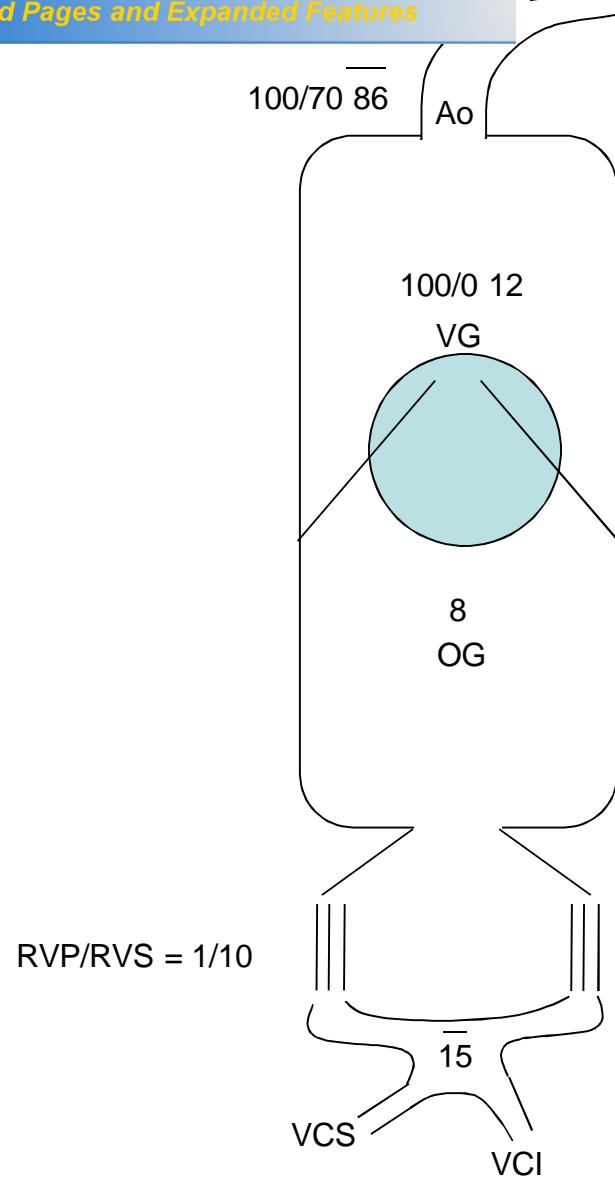
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C'est la pression diastolique qui est transmise

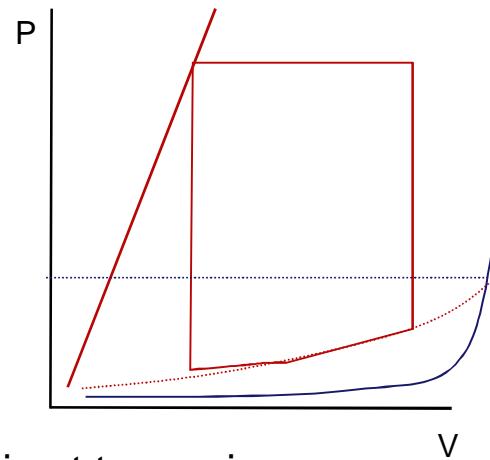
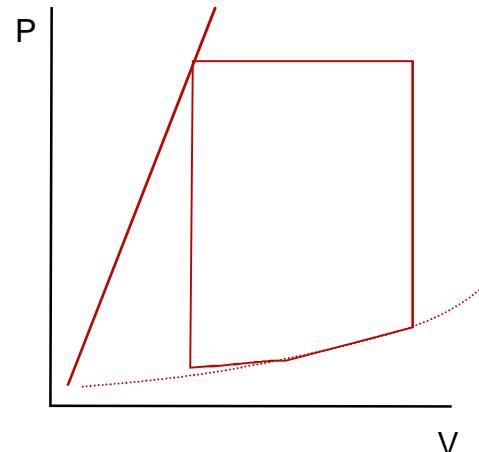


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C'est la pression moyenne qui est transmise

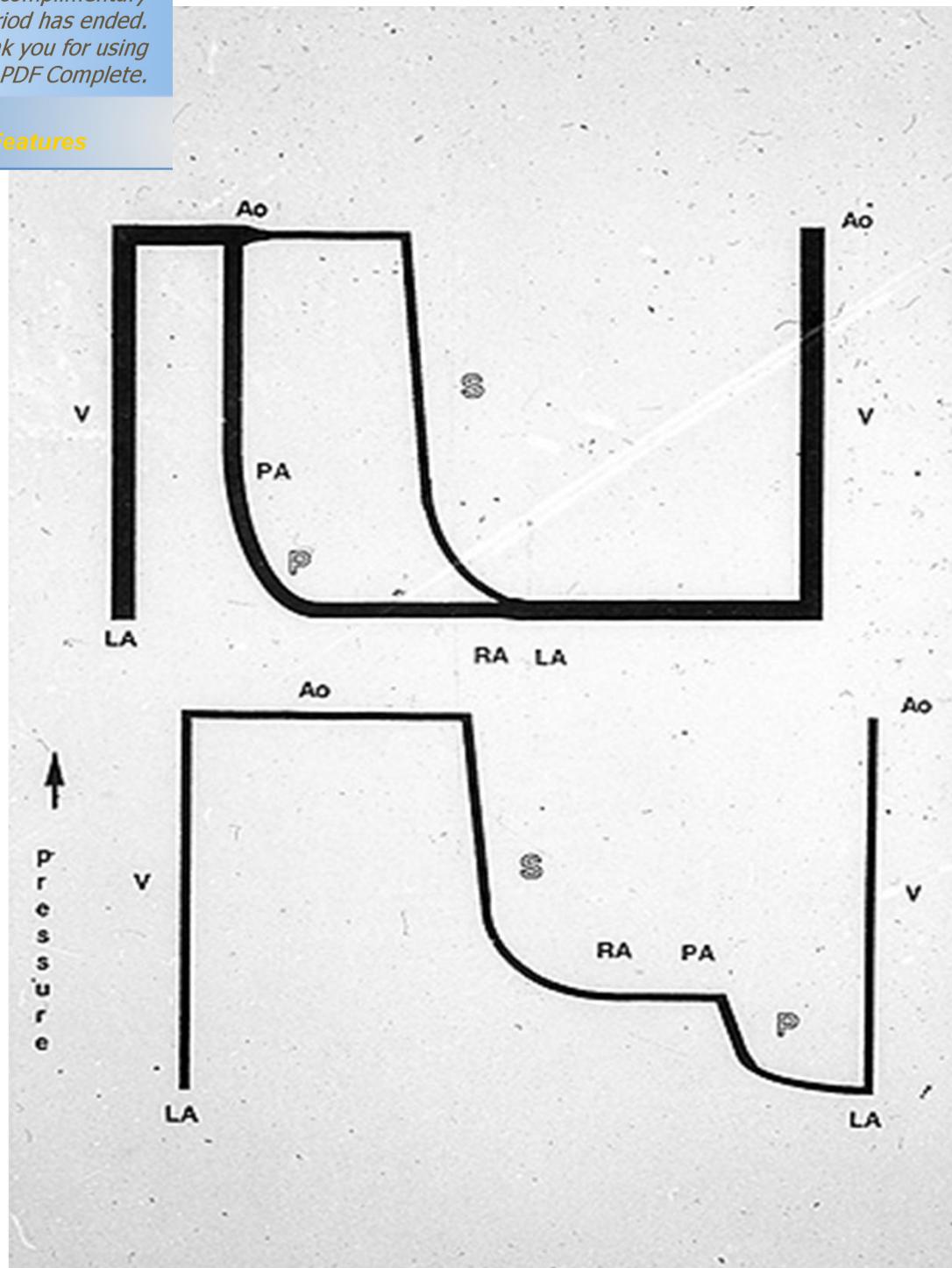
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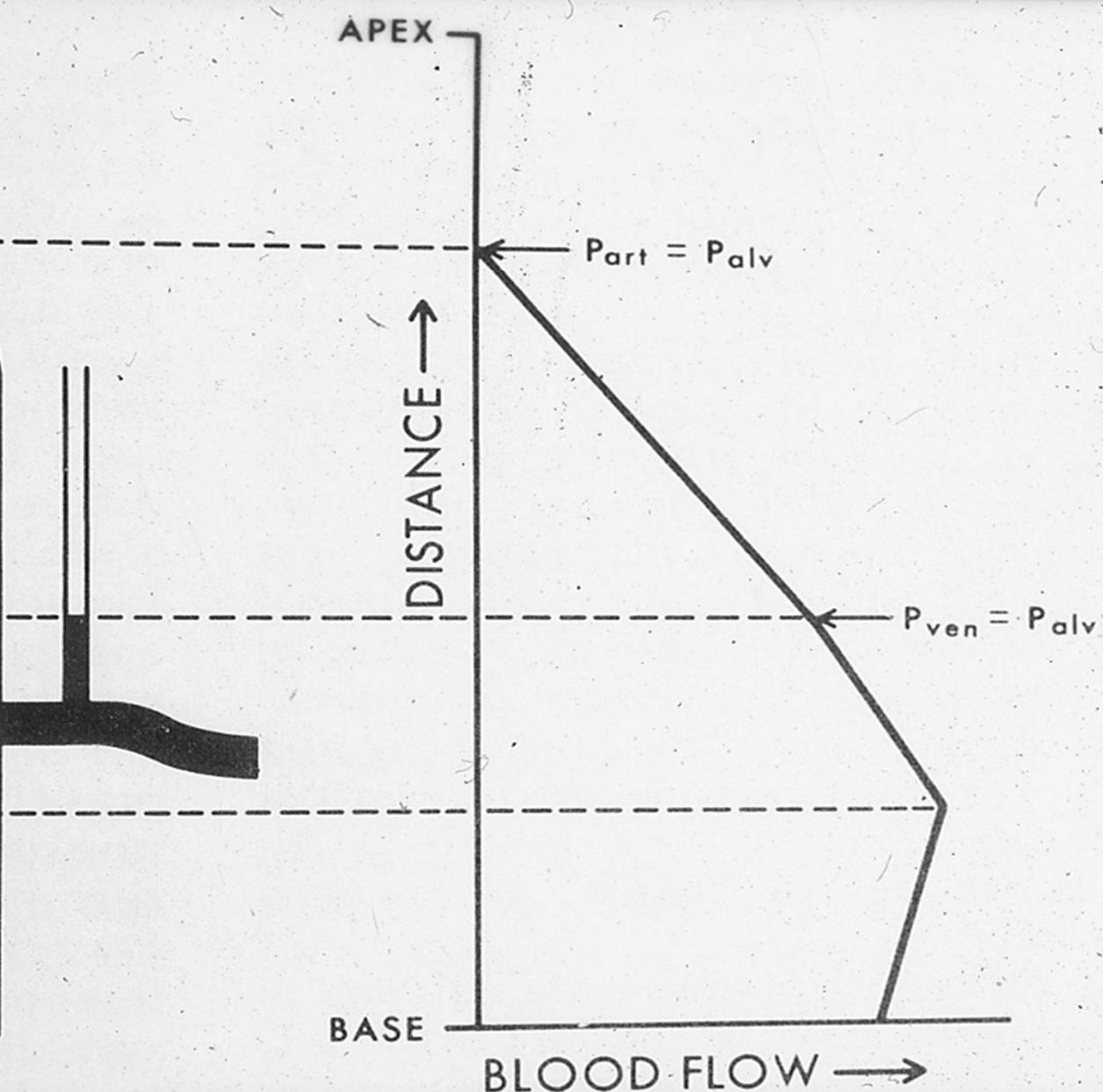
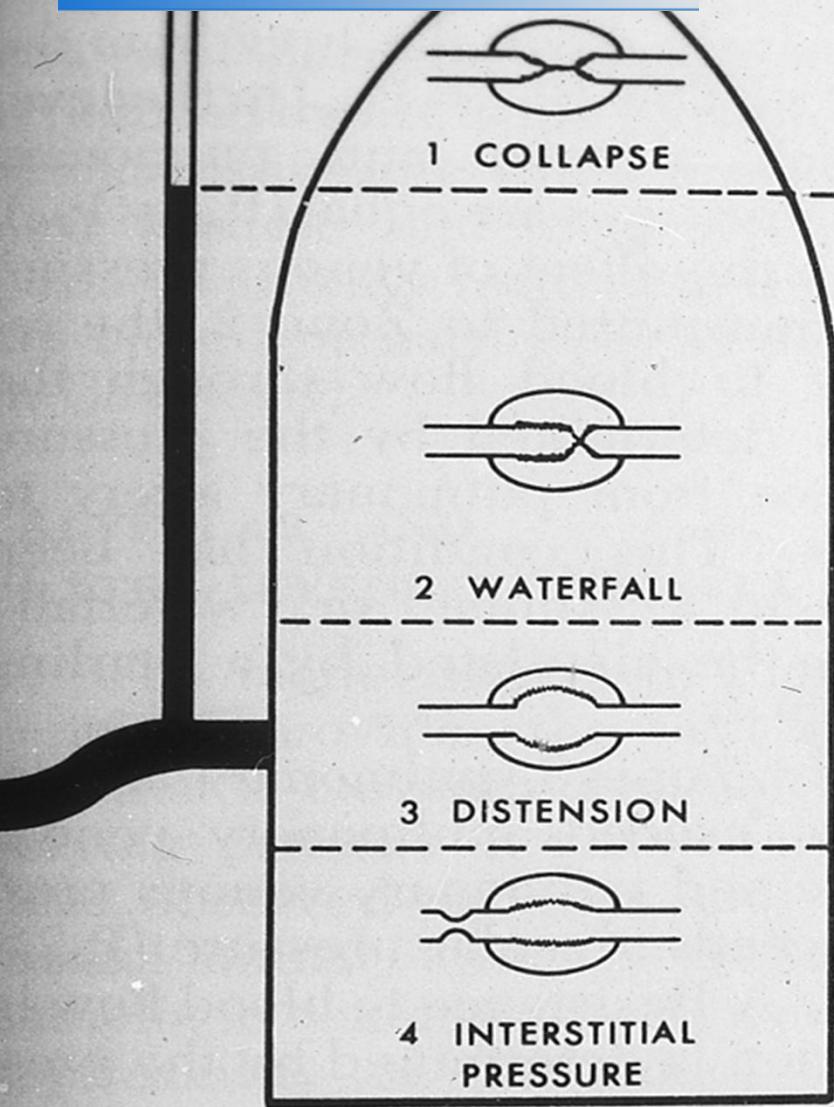
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# Le paradoxe du Fontan

- ” Pour bien perfuser les poumons et précharger le VG on a besoin d'une Pr AP pulsé > 15 mmHg
- ” Pour une bonne perfusion tissulaire sans fuite capillaire la Pr cave doit être < 10 mmHg
- ” Or dans la DCPT la Pr Cave = ou > Pr AP



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# Pathopathologie de la DCPT

- ” La pression Cave est au mieux égale à la Pr AP c'est à dire:
- ” Pr Cap (VG et VAV) + Gdt Pr trans-pulm (RVP)
- ” Pas de réserve pour augmenter la précharge VG
- ” Congestion cave obligatoire + HT Portale
  - . Hyperproduction Lymphatique (Foie et tube dig)
  - . Drainage lymphatique difficile (VCS)

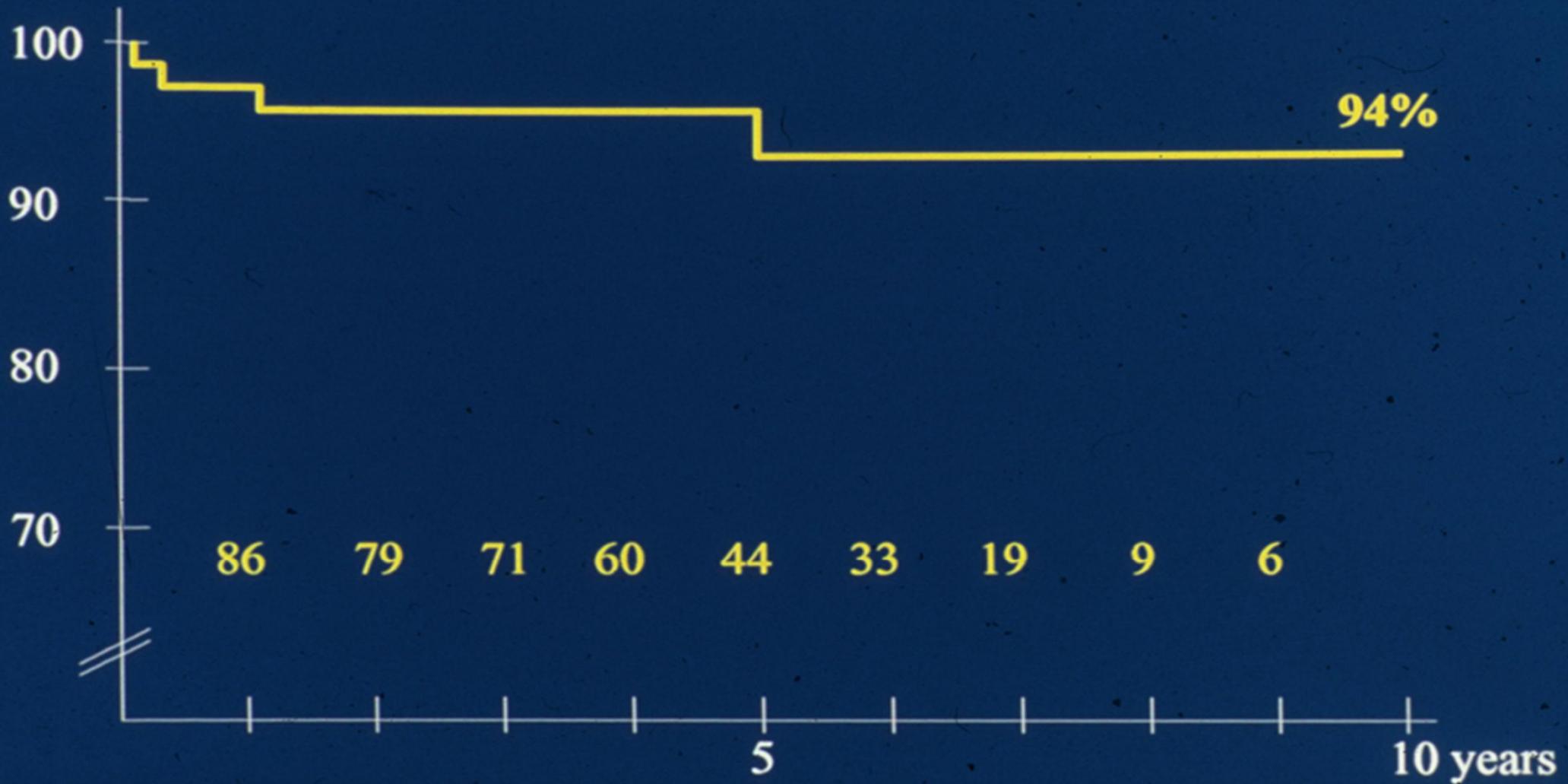


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## Fontan candidates

### Actuarial survival





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# Surgical treatment of univentricular heart

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## Fontan or not Fontan



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# What are the options?

- ” Interruption of Pregnancy
- ” Leave the patient with complete mixing of the Blood with natural or closed heart calibration:
  - . PA banding +/- Ao repair
  - . BT shunt
- ” PCPC followed by TCPC
- ” PCPC + additional Blood Flow

And then eventual Heart Transplantation



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# Goals

” Find the best solution

”Leave the worse  
solution



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## Goals of surgical treatment

- **to provide survival**
  - low operative risk
  - low late mortality
  - preservation of ventricular function
- **to provide adequate quality of life**
  - adequate arterial saturation (>85%)
  - adequate exercise tolerance
  - low late morbidity



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## Theoretical Considerations : TCPC

“The price to pay for the absence of cyanosis

“Probably too high in the long term



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# CHF is a very abnormal circulation

- . Too low PA pressure
- . Too high Venous pressure in the caval and portal circulation
- . Abnormal lymphatic circulation:
  - " increase production
  - " impaired drainage
- . Abnormal Heart's loading conditions



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# Lung Circulation in TCPC

- " Low non pulsatile PA pressure with no perfusion in the upper part of the lungs and inadequate lymphatic drainage
  - . Increases dead space
  - . Promotes Collateral circulation
  - . Leads to Arterio-Venous fistulae



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# Liver and Gut in TCPC

- " High venous pressure and bad lymphatic drainage leading to:
  - . Liver damage
  - . Portal hypertension
  - . Protein loosing enteropathy



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# Heart in TCPC

- “ **Fragile preload** (depending on the passive lung perfusion) on a stiff single ventricle. IVC compression during pregnancy
- “ **Increase afterload** due to the addition of the lung after the systemic circulation
- “ **No access for percutaneous intervention** (PM and ablation)

# Fontan Paradoxe

” Impossible to conciliate:

- . Good Lung perfusion needs pulsatile pressure high enough to counterbalance hydrostatic pressure  $>15$  mmHg (20/10)
- . Low caval pressure to avoid congestion and excessive lymph production  $< 10$  mmHg

**In TCPC, Caval = PA pressure**

**and       $<10$  cannot be  $> 15$**

# Critical considerations

## In SV with complete blood mixing

- ” **Obligatory cyanosis** with CNS risk and polycythemia and dyspnea
  - . < 85 %
  - . > 90 %
- ” **Increased volume load** on the single ventricle (QP/QS + A-V valve Reg + ESV)
  - . QP/QS < 2/1
  - . QP/QS > 3/1



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## doxe in complete mixing circulation

“Need unacceptable overload (QP/QS 3/1) for a reasonable level of hypoxemia (90% O<sub>2</sub> sat at rest)



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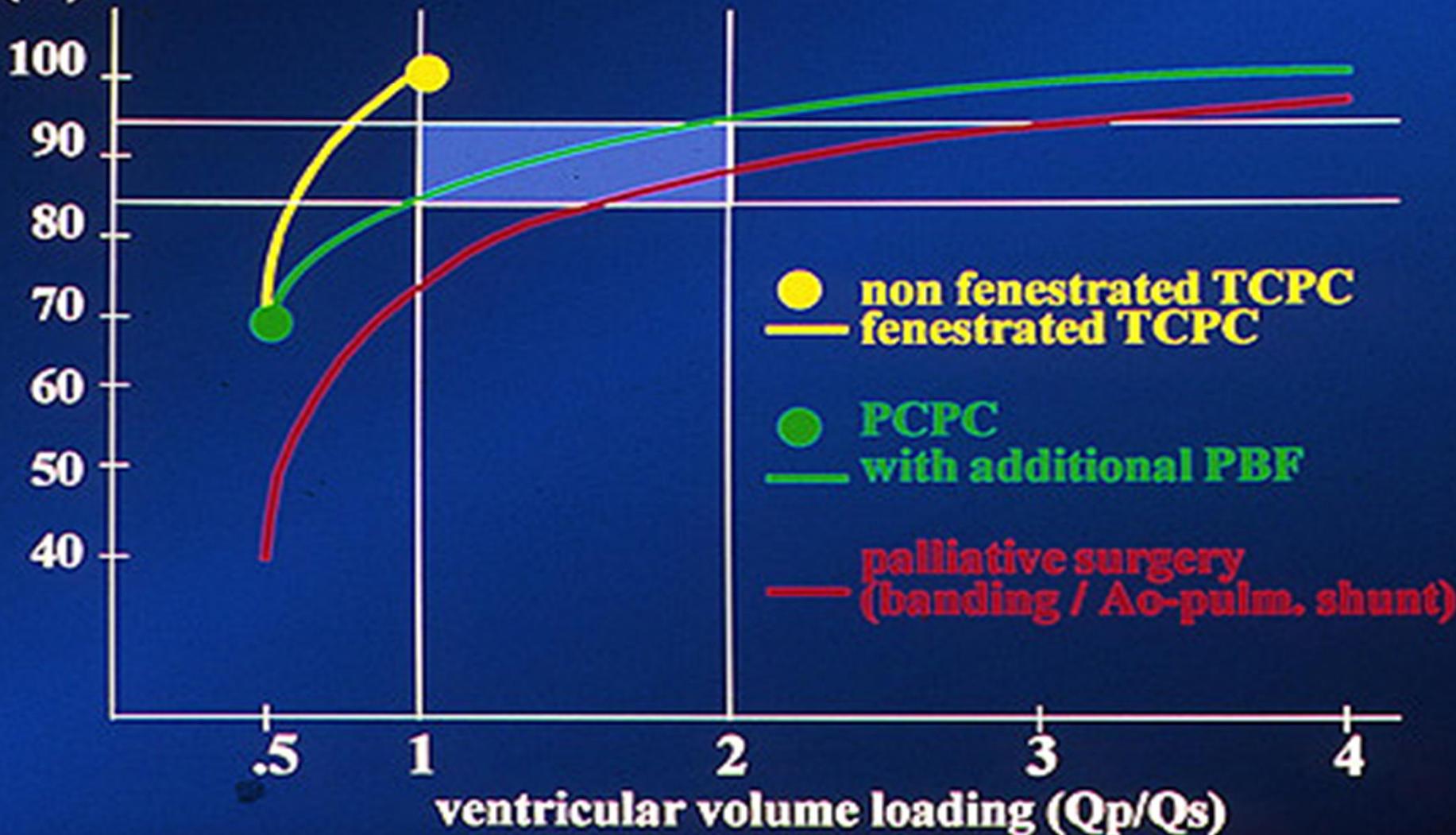
+ additional PA Flow

a good compromise ?

- " Adequate O<sub>2</sub> sat at rest for limited volume overload
- " Better lung perfusion
- " Low IVC pressure and limited increase in lymph production

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O<sub>2</sub> sat (%)





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# Evolution from Theories to Practical considerations

Study on patients suitable for CP circulation that had either:

- “ TCPC
  - . Atrio-pulmonary (old Fontan for Tric atr )in late 70 and early 80
  - . TCPC for any SingleVentricle in late 80 and 90
- “ PCPC + additional PA Blood Flow



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# Long term of Fontan circulation: The Paris (NEM) experience

Daniel Sidi

2005 for World Congress

Buenos Aires



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# Population

- " **Group 1: 1975-1985: 76 old true Fontan (Atrio-PA Connection) for Tricuspid Atresia**
- " **Group 2: TCPC for all kinds of SingleVentricles with good PA pr and PA branches. (198 pts)**
  - . 1988-1999: Intra Cardiac Lateral Tunel(s) (86 pts)
  - . 1998- 2004: Extra Cardiac Tube in a 2 steps TCPC with fenestration (112 pts)

# Old Fontan(s) conclusions

- ” A disaster
- ” It is indeed a **terrible palliative surgery**
- ” Less than 50% survived 20 years and less than 20% have really good results (including half after conversion)
- ” Transplantation
- ” Dysrhythmia is the rule and not the exception
- ” Not very good candidate for Heart



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# TCPC results

- “ Early Mortality is low: 4/198: 2%,
- “ Cerebro-vascular problems in 5/198 with sequella in 3
- “ Lost for correct follow-up is high : 27/195: 14%
- “ Late mortality or HT:21/168: 12%



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# Complications of the TCPC for non converted TCPC: 131 pts

- “ Dysrhythmia: 51 + 3 : 39% + 2%
- “ Venous cong. (diuretics) 65 : 50%
- “ Pers. Cyanosis (f): 48 Ë 6 : 37% - 4%
- “ + severe complications
  - . Brain damage: 3
  - . Protein losing enteropathy: 1 + 2



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104 patients

14  
Fontan non-candidates

90  
potential Fontan candidates

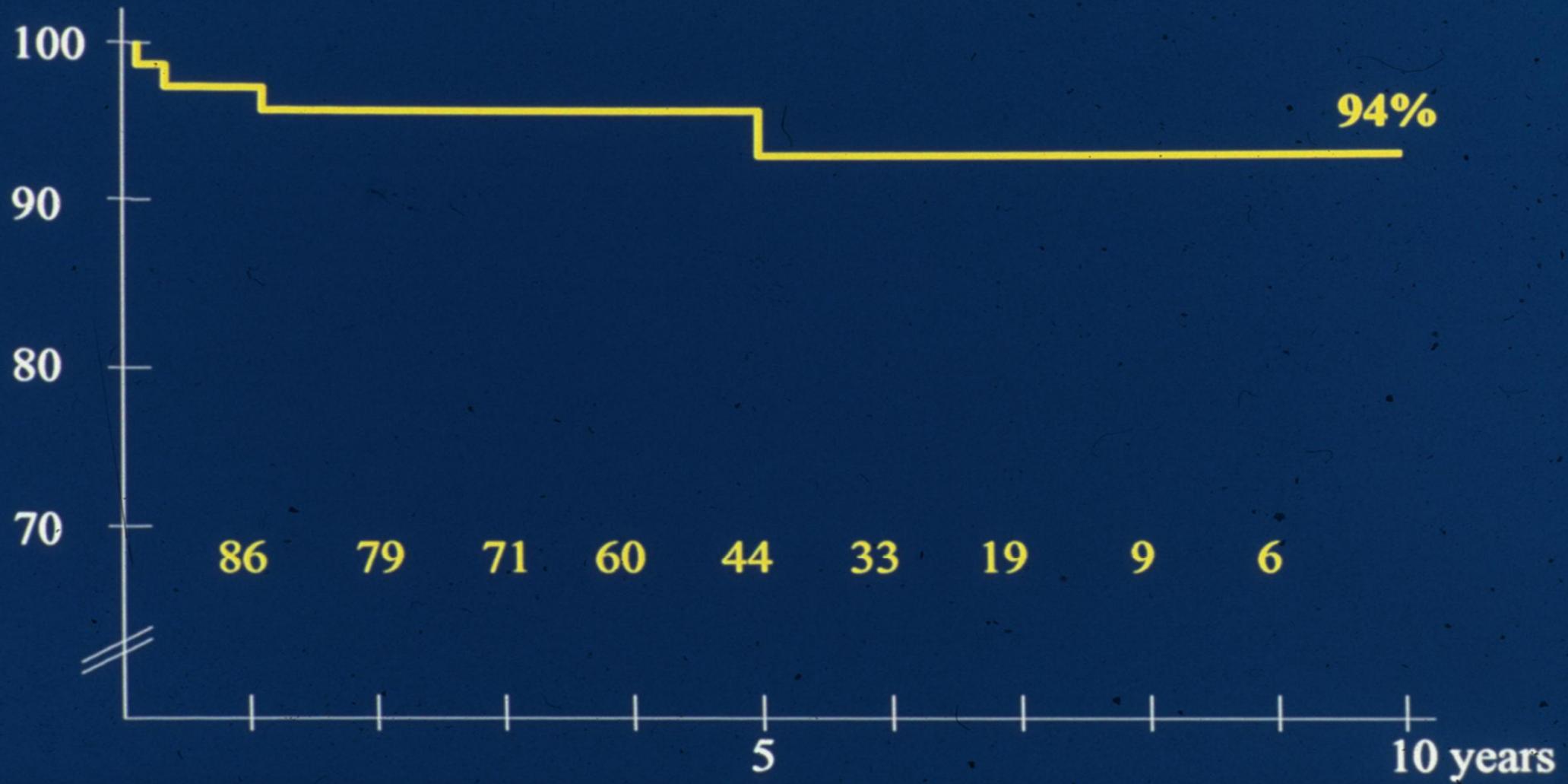


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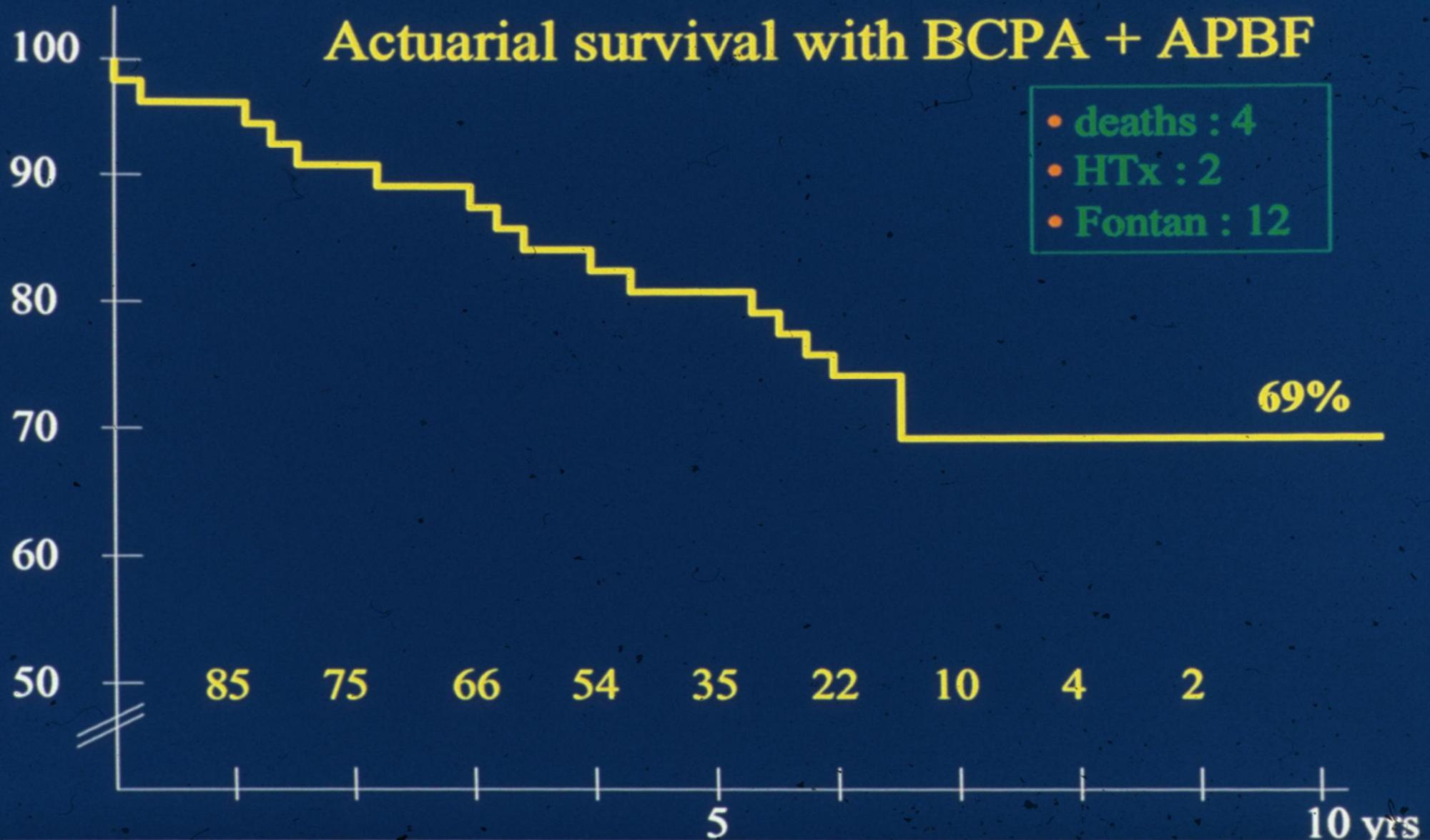
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## *Fontan candidates*

### Actuarial survival



## *Fontan candidates*





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## BCPA with APBF vs TCPC

### Late clinical results

	<b>BCPA with APBF</b>	<b>TCPC</b>
<b>Late mortality</b>	—	<b>1 (4.3%)</b>
<b>Normal EKG</b>	<b>16 (94%)</b>	<b>16 (70%)</b>
<b>Significant arrhythmia</b>	—	<b>3 (13%)</b>
<b>Chronic medication</b>	<b>2 (12%)</b>	<b>11 (48%)</b>



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## *BCPA with APBF*

### **BCPA with APBF vs TCPC**

	<b>BCPA with APBF</b>	<b>TCPC</b>
<b>survival</b>	+	-
<b>cardiac rhythm</b>	+	-
<b>chronic medication</b>	+	-
<b>exercise capacity</b>	=	=
<b>O2 saturation</b>	-	+



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## BCPA with APBF vs TCPC

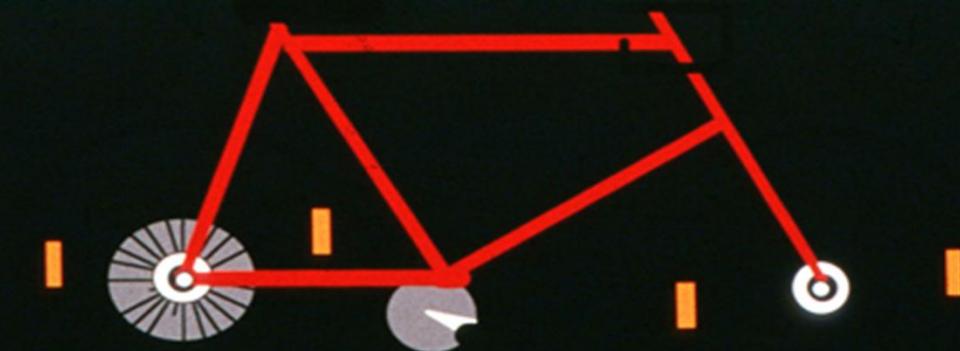
### Exercise stress test

	BCPA with APBF	TCPC	
peak exercise (watts)	91 ± 17	103 ± 15	NS
O2 sat (%) at rest	84 ± 4	93 ± 4	p<0.001
O2 sat (%) at peak exercise	64 ± 8	85 ± 6	p<0.001



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# Difficult calibration of the additional PA flow

” → iterative surgeries

” Natural PA stenosis -

” PA banding +/-

” Systemic shunts +++



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# Best politic?

**"Wait for the TCPC  
or**

**"Go directly to Heart  
Transplantation**



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# What for TCPC?

“ Good option, as long as it does not impair too much the TCPC results

- . Does not affect Ventricular function
- . Does not affect Pulmonary Vascular bed



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# TCPC in « old » children

- ” 65 pts had TCPC beyond the age of 15years
- ” 27 beyond the age of 18 years
- ” 15 beyond the age of 21 years (up to 32)
  
- ” **There was no differences in mortality and morbidity according to age**, but we were very strict on the indications



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# When should we tick the clock?

- “ Only in « ideal » patients otherwise go straight to Heart Transplantation (TCPC is not a good bridge to HT)
- “ In « ideal patients » as late as possible but not too late (before 30 years old?)
  - . When the patient needs a new operation for adequate additional PA flow.
  - . When the cardio-vascular **follow-up** indicates a deterioration of ventricular function
  - . After pregnancy ?



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## Old Fontan

“Only one patient in  
our group had a baby



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# Old Fontan

“But he was a Male

“3 pregnancies

- . 1 early termination
- . 2 miscarriages



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# about interruption of pregnancy?

” Are the results achieved with either policy of TCPC or PCPC + additional PA flow good enough to contre-indicate interruption of pregnancy in single ventricle ?



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# Complication of Pregnancy in Single Ventricle

“Our recommendations in  
Paris in NEM with

“Laurent Fermon and  
Jerome Le Bidois (IPP)

# Consequences of Pregnancy in Single Ventricle

## “ Justified for the Bad Single Ventricle

- . Hypoplastic Left Heart
- . Heterotaxia and AV Canal Single Ventricle

## “ Open discussion for the so called « good » Single Ventricle

- . Tricuspid atresia
- . DILV without Ao obstruction



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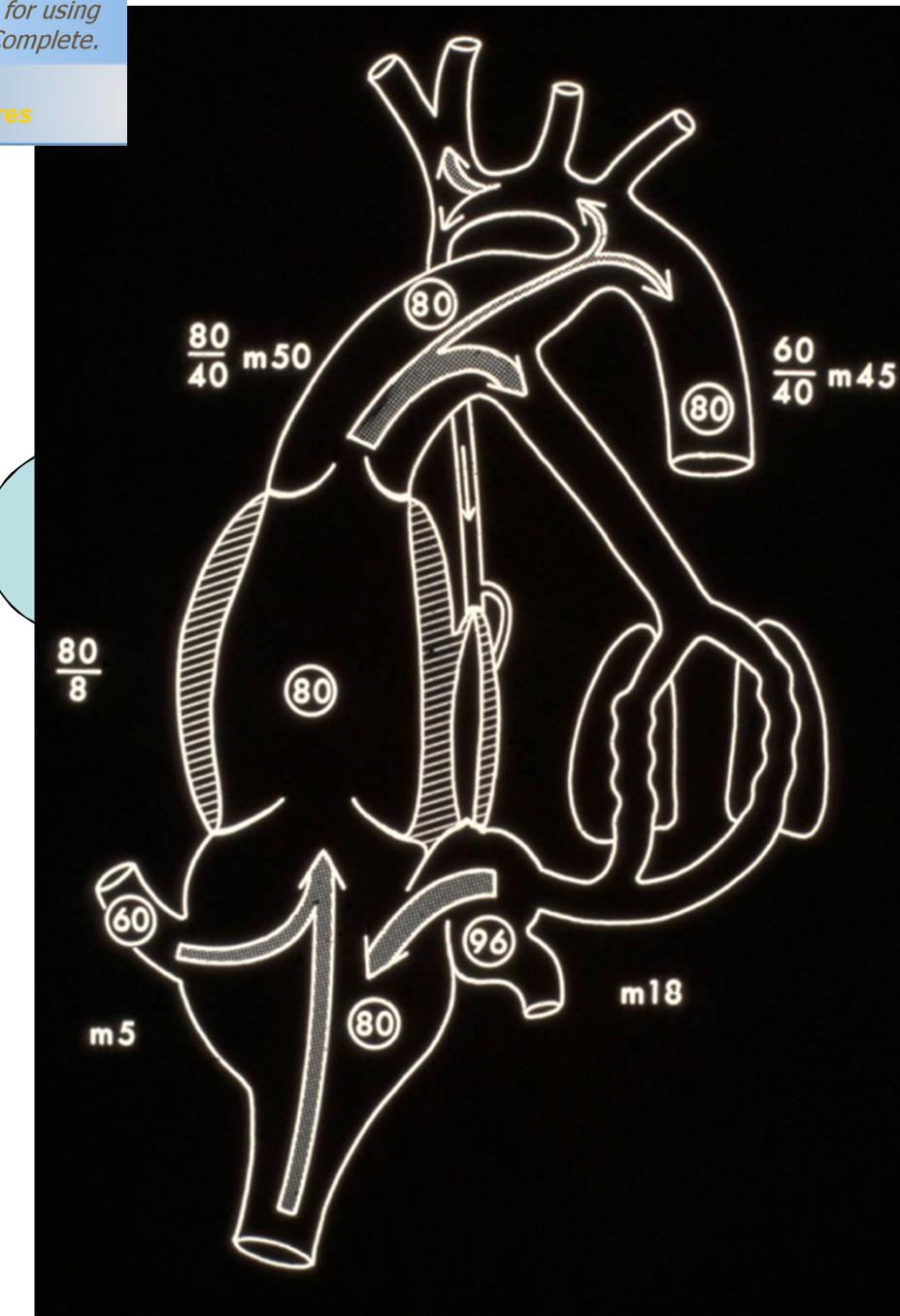
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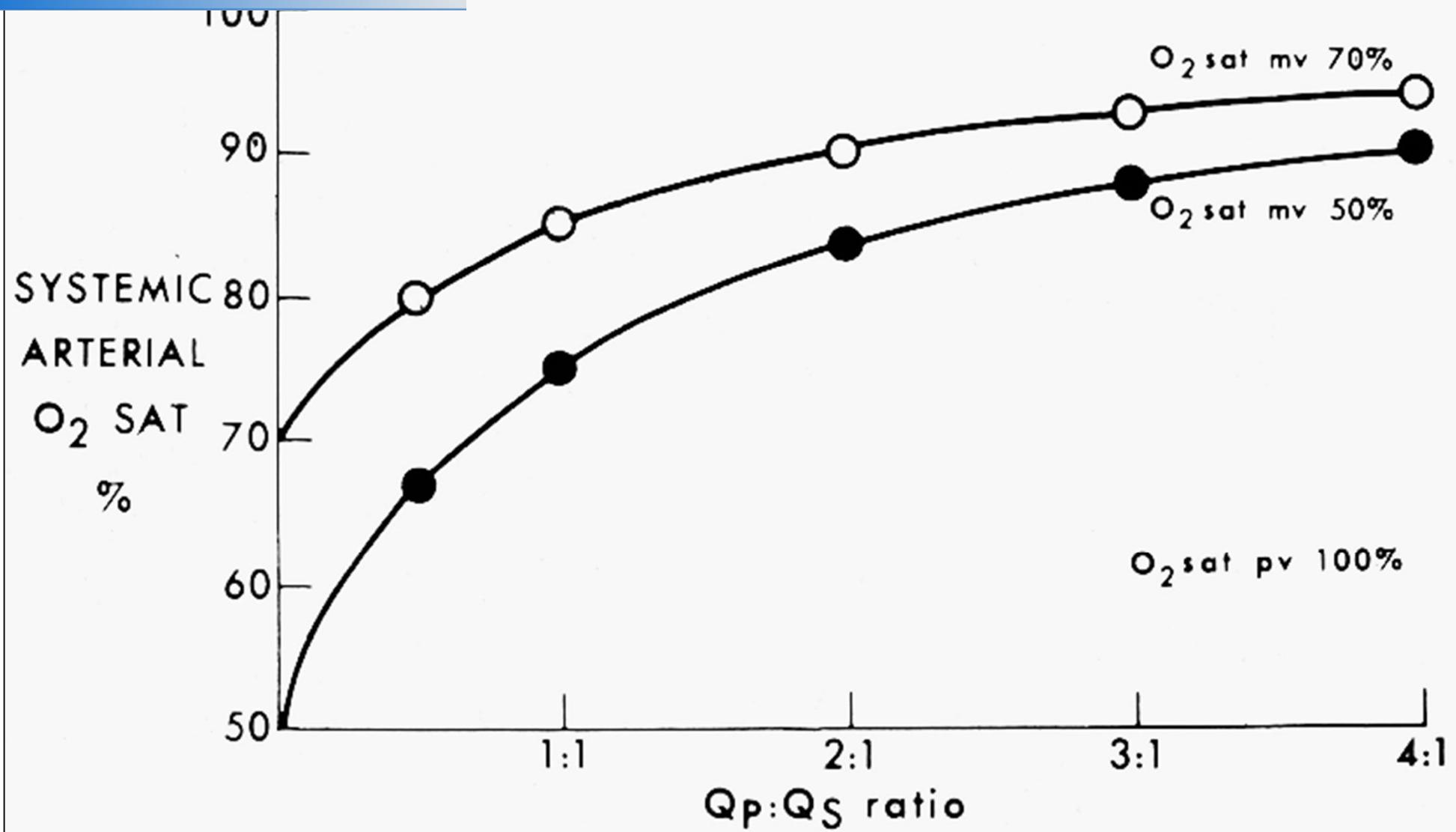


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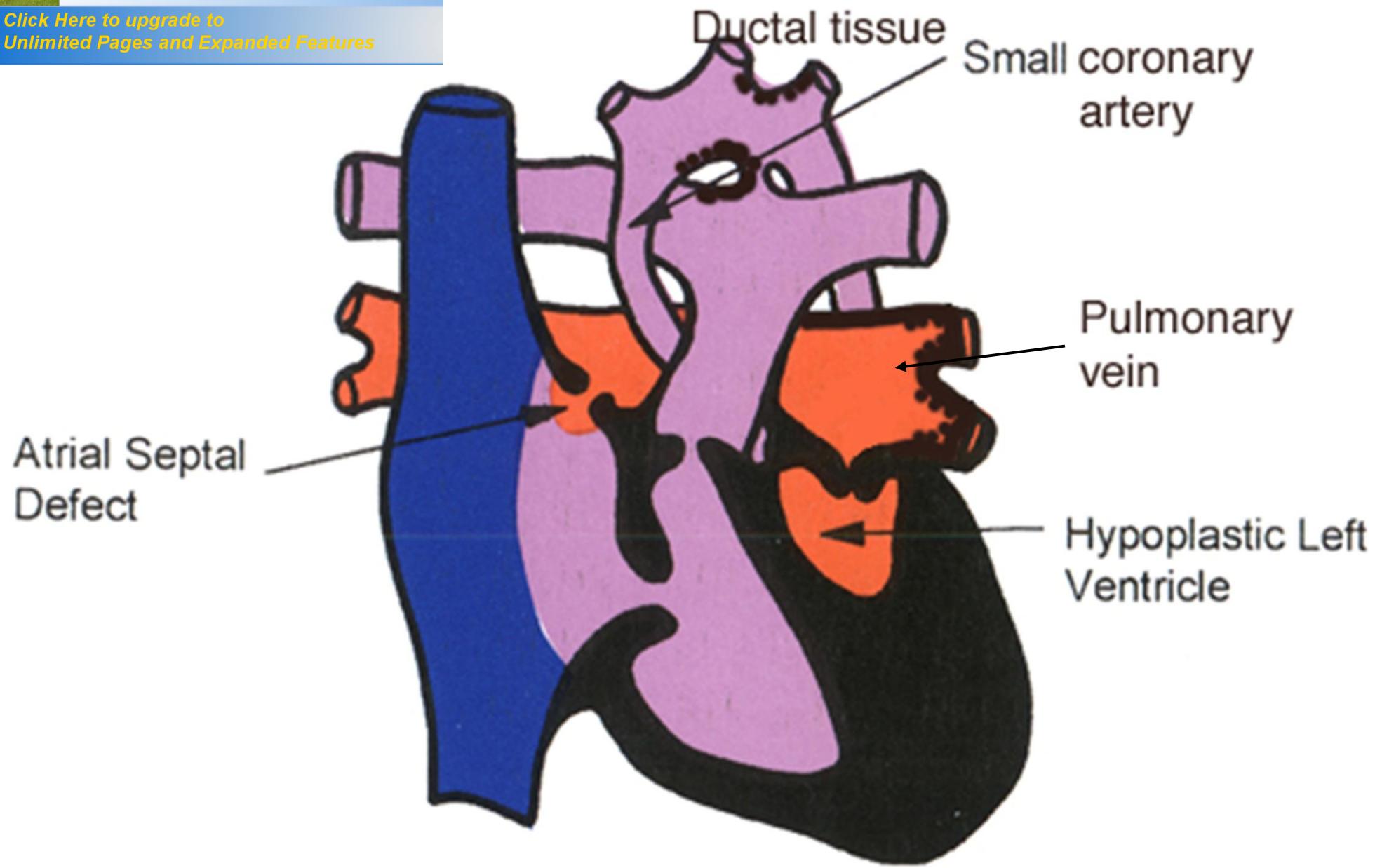
Ao

SPTI

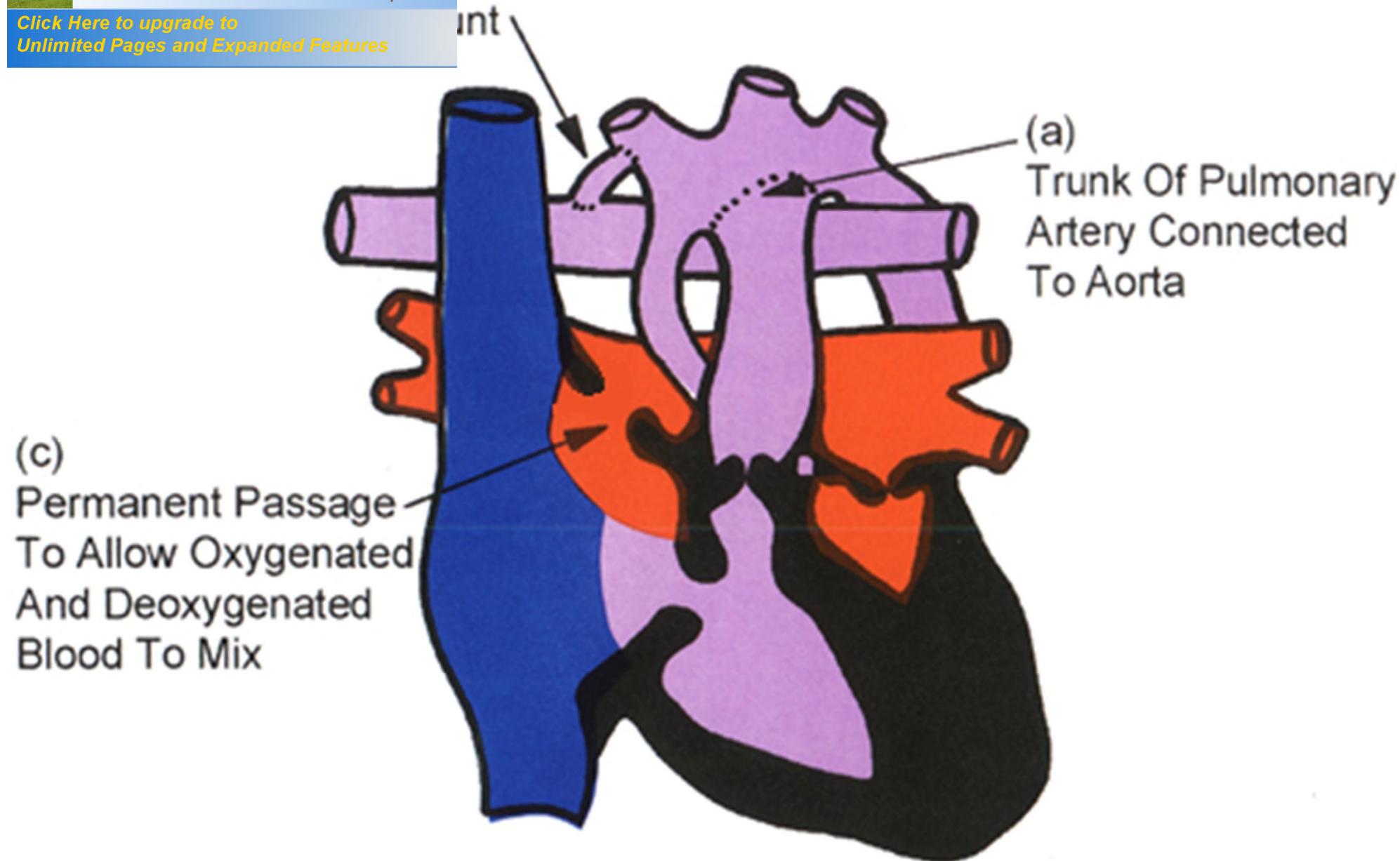
DPTI

LV

Normal



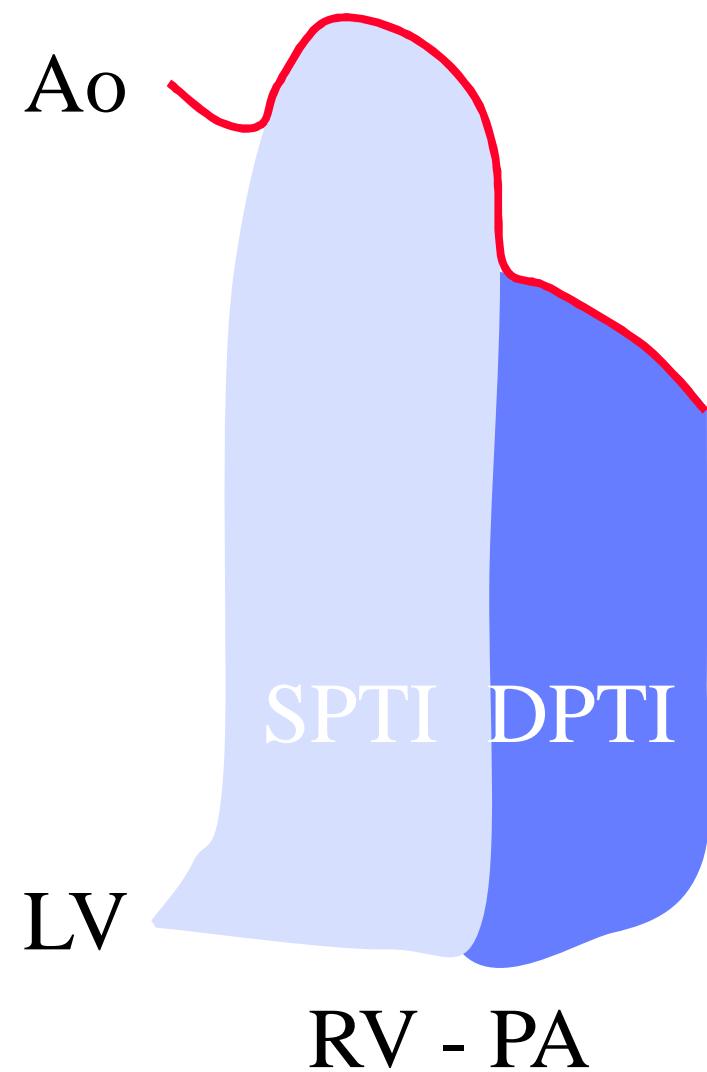
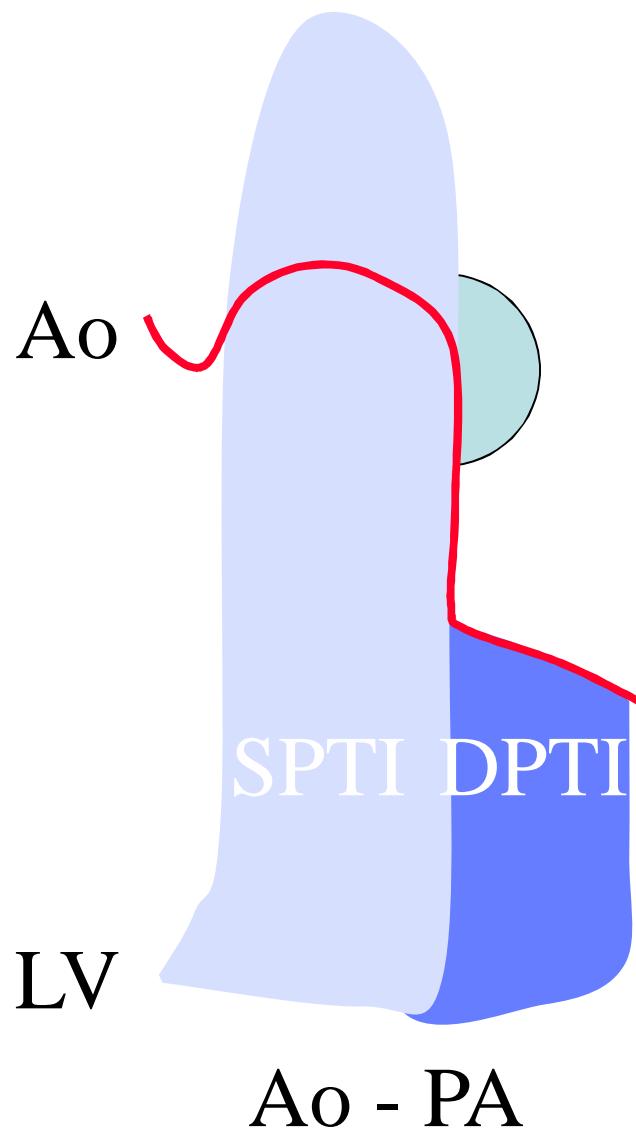
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# Norwood: Ao/PA or RV/PA ?

- “ For the PA bed: Ao/PA better than RV/PA
  - “ Better QP for the same PA sys pr.
  - “ No PA regurgitation
  - “ *despite less pulsatility that may stimulate growth?*
- “ For RV function: RV/PA better than Ao/PA
  - “ Better coronary diastolic perf usion
  - “ less RV afterload (\ Ao sys pr)
    - . diastolic leak flow through Ao/PA shunt  $\rightarrow \uparrow$  Ao syst pr
    - . less flow (and gradient) through the plastified Asc Ao
  - “ *Despite the RV scar and the PA/RV regurgitation*

# Norwood



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