Urgências e emergências cardíacas em cardiologia pediátrica

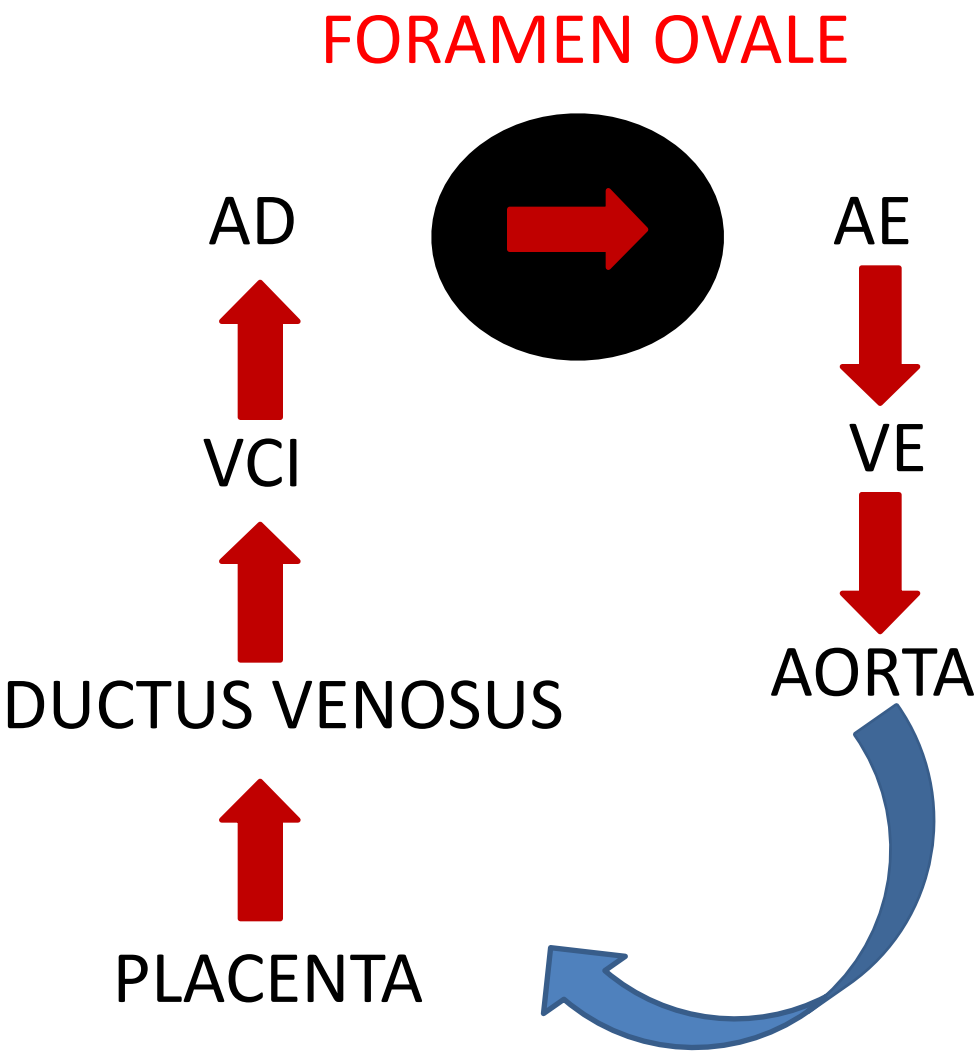
3ª Curso Básico de Cardiologia Pediátrica

Serviço de Cardiologia Pediátrica
Hospital Pediátrico de Coimbra, CHUC

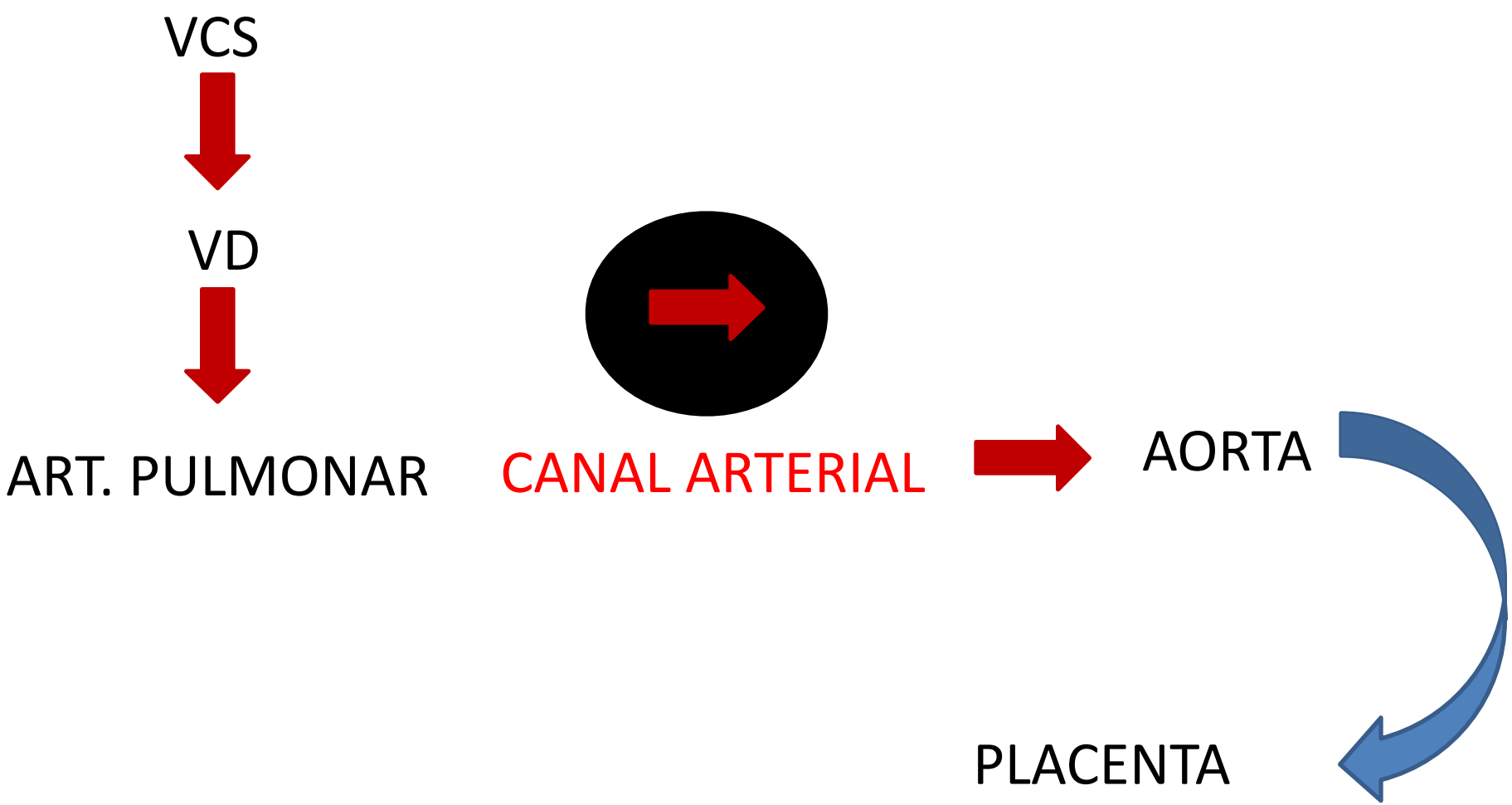
Etiologia

- Patologias canal dependente
- Patologias ‘foramen ovale ’ dependente
- Crise cianótica
- Arritmias

Circulação fetal

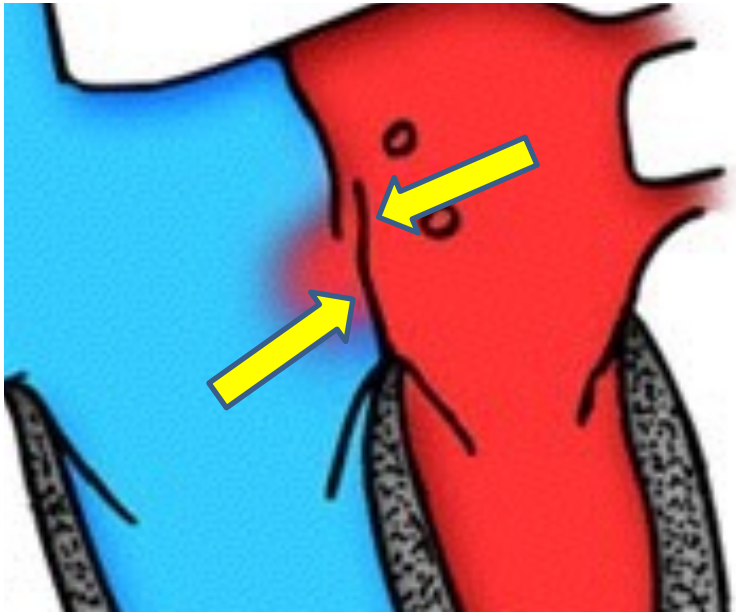


Circulação fetal

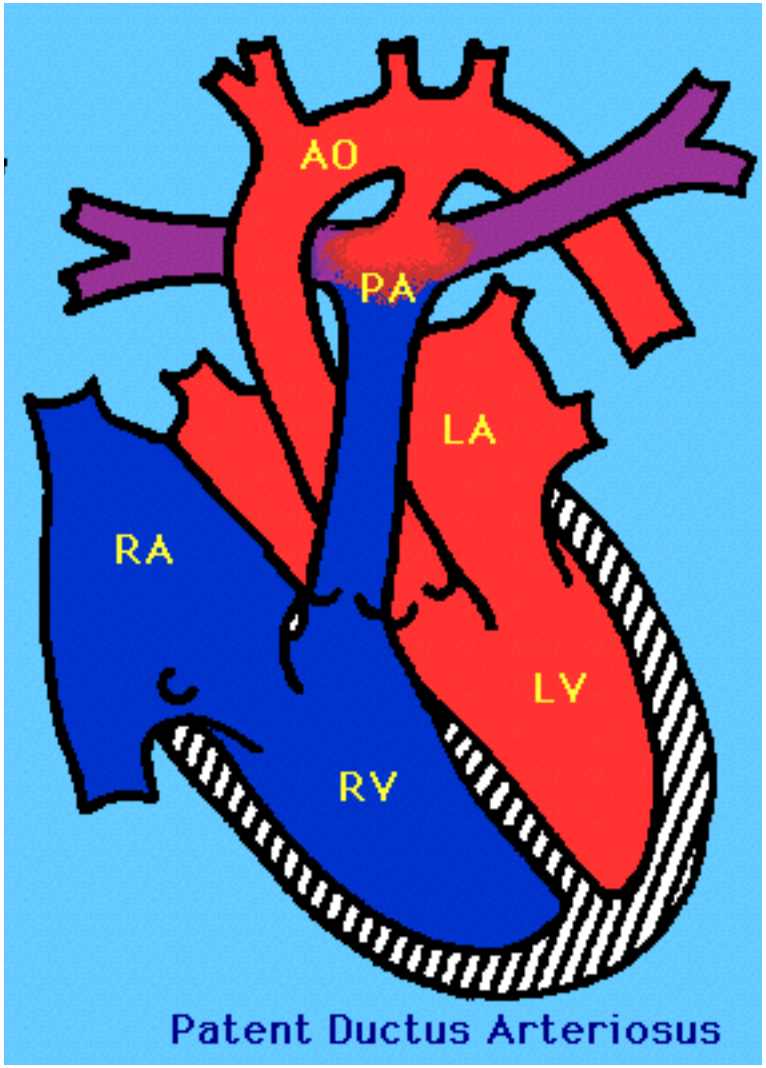


Foramen ovale

- Patente durante a vida fetal: AD> AE
- Após o nascimento resistência vascular sistémica aumenta
- pressão AE > AD
- Foramen ovale encerra



Canal arterial



Canal arterial

- Encerramento funcional < 24 horas de vida
- Contração do músculo liso
- Encerramento anatómico 3 semanas de vida
- Depende:

Gestação

PO_2

Prostaglandinas

Canal arterial:O_2

- Potente estímulo para o encerramento do canal arterial
- Resposta vigorosa do músculo liso ao O_2

menos intensa em prematuros

Prostaglandinas

- Níveis diminuem após nascimento

  placenta fonte de PGE

  fluxo pulmonar “lava” PGE

- Prematuros:

  produção PGE/ metabolismo

  resposta mais “ávida” ao PGE

Canal arterial patente

Cardiopatias canal-dependente

- Cianose

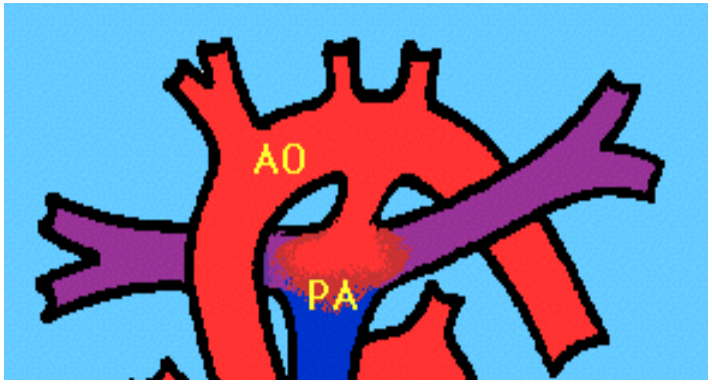
*ausência de resposta à oferta de O_2

- Choque cardiogénico
- Misto

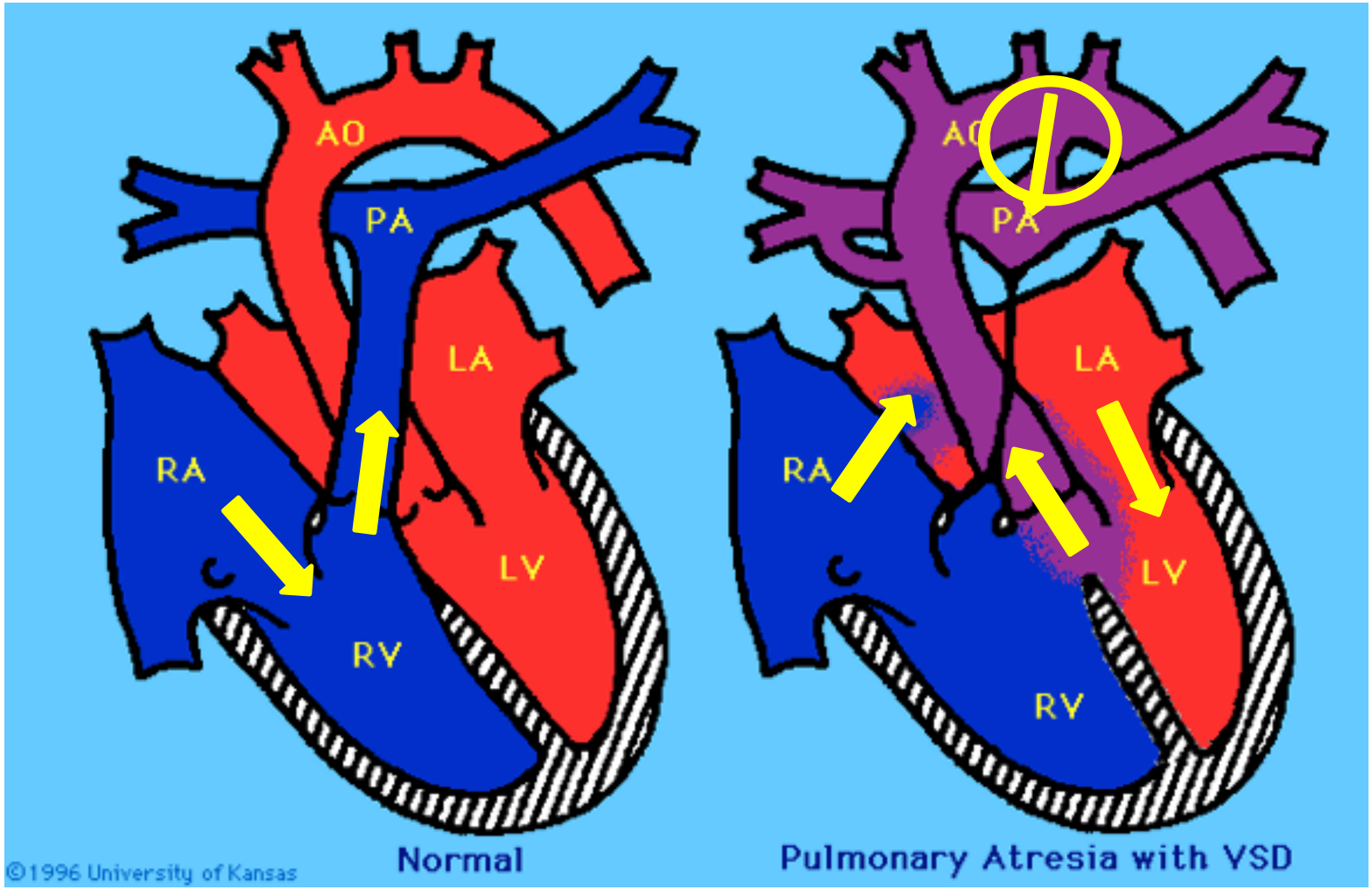
Porquê?

- Cianose: diminuição do débito pulmonar
- Choque cardiogénico:diminuição do débito sistémico

Clínica coincide com o encerramento do canal arterial



Cardiopatia com cianose

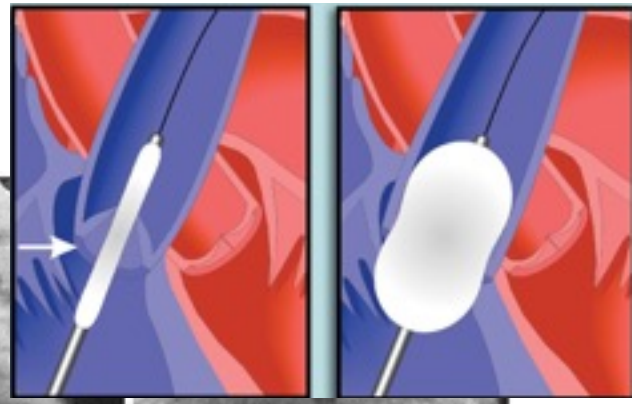


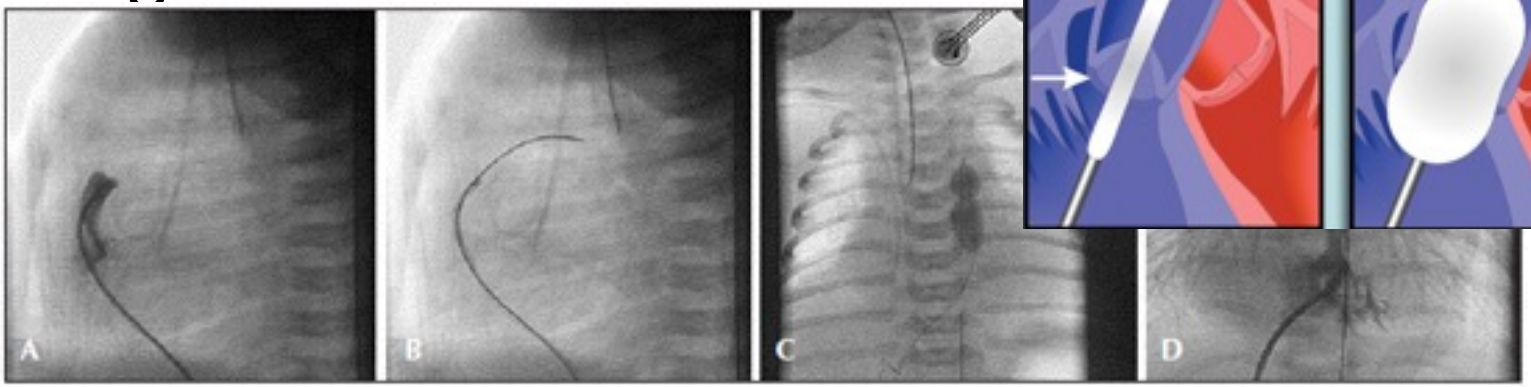
Tratamento

- Manter o canal arterial patente

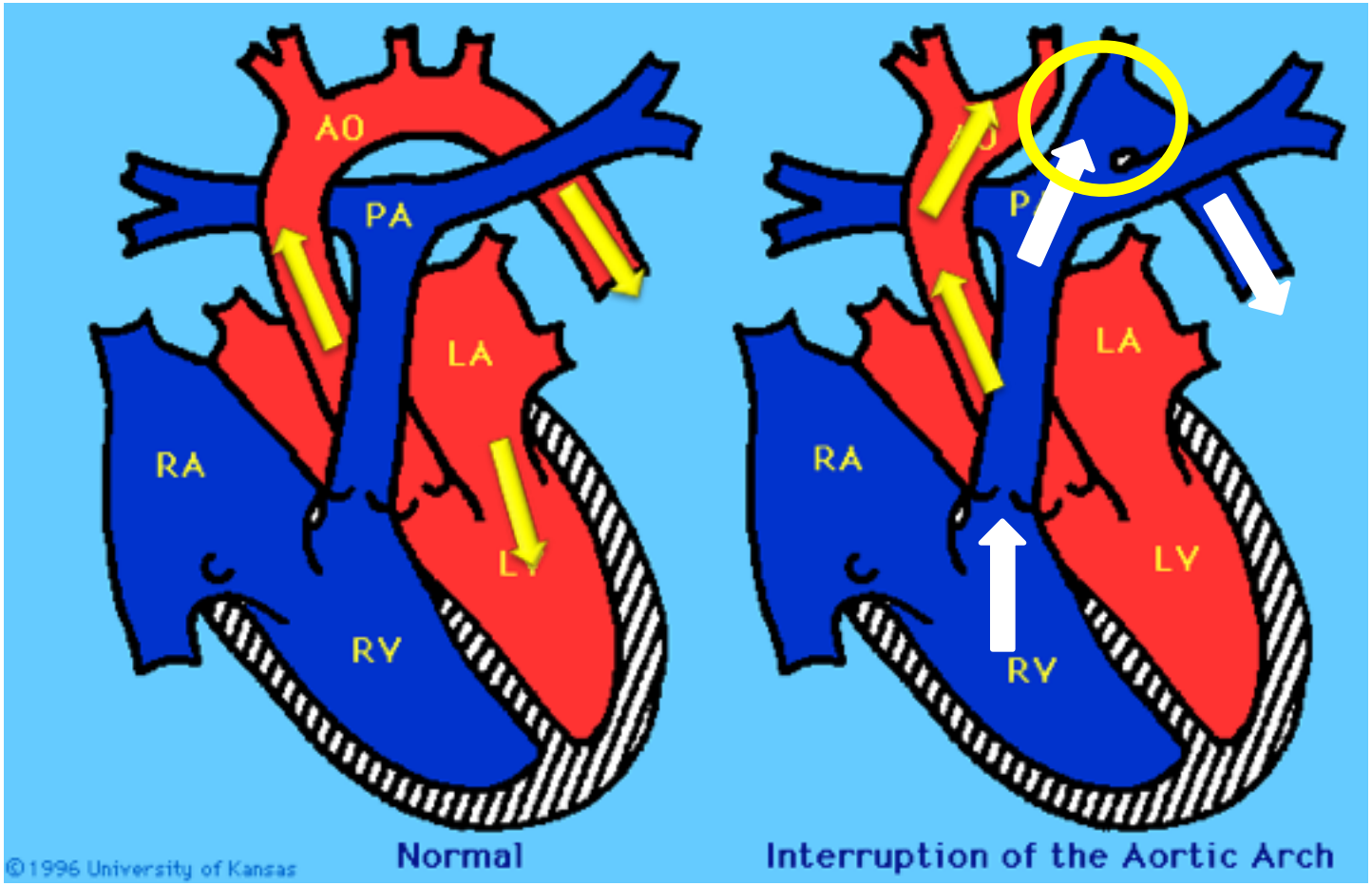
  PGE_1

- Perfuração da válvula:
- Por via percutânea: rádio-frequência + valvuloplastia
- cirurgia





Cardiopatia com choque cardiogénico:interrupção do arco aórtico



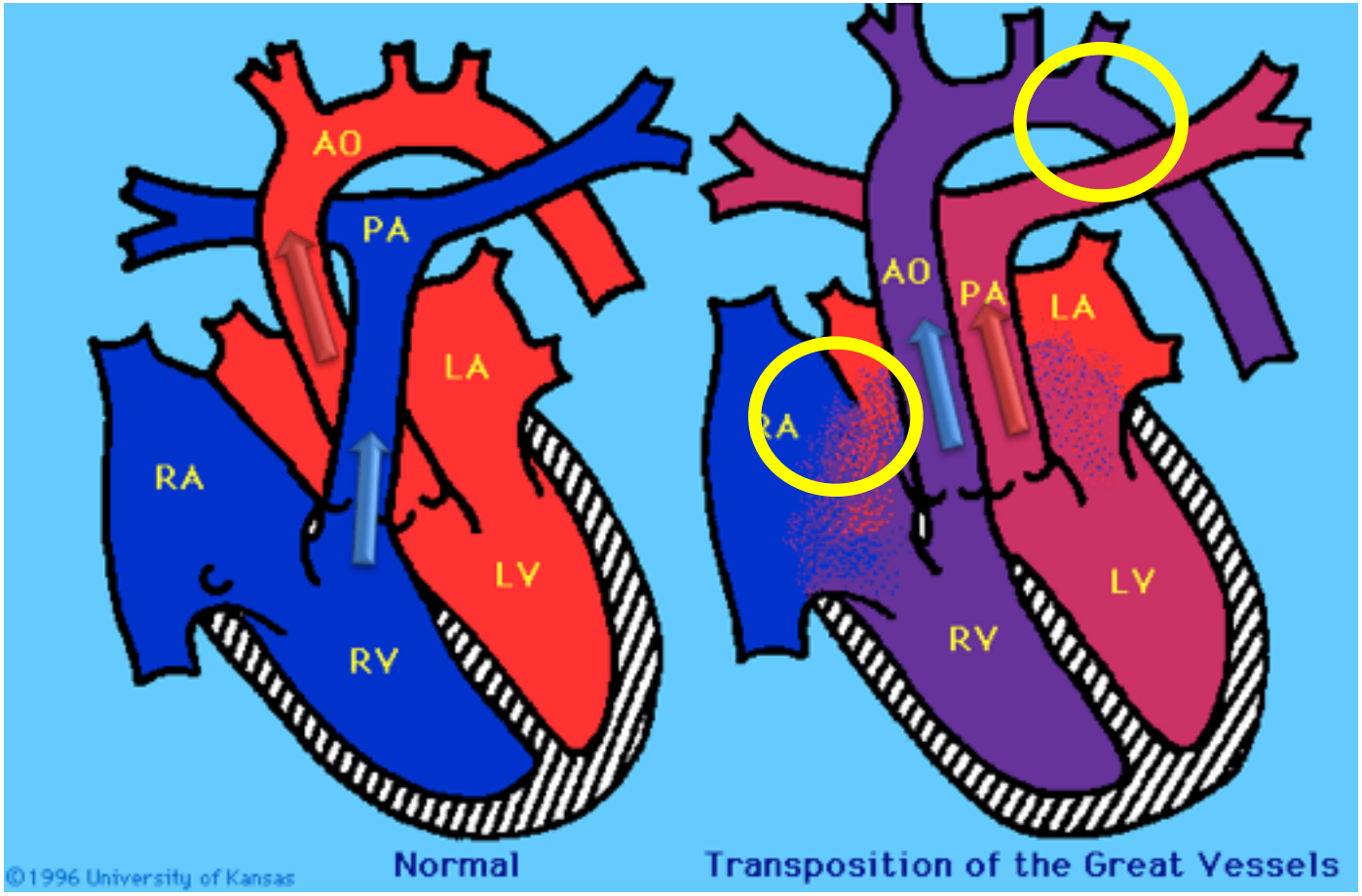
Tratamento

- Manter o canal arterial patente

  PGE_1

- cirurgia

Cardiopatia ‘FO’ dependente:cianose

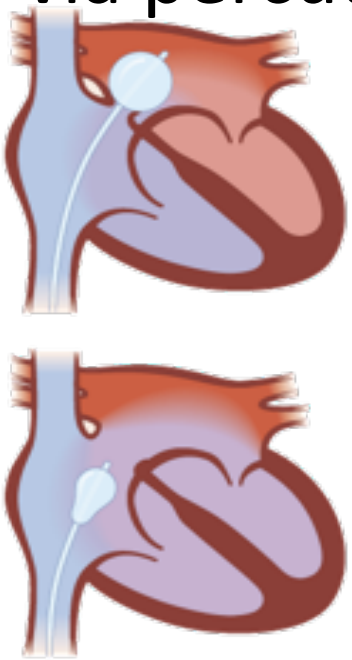


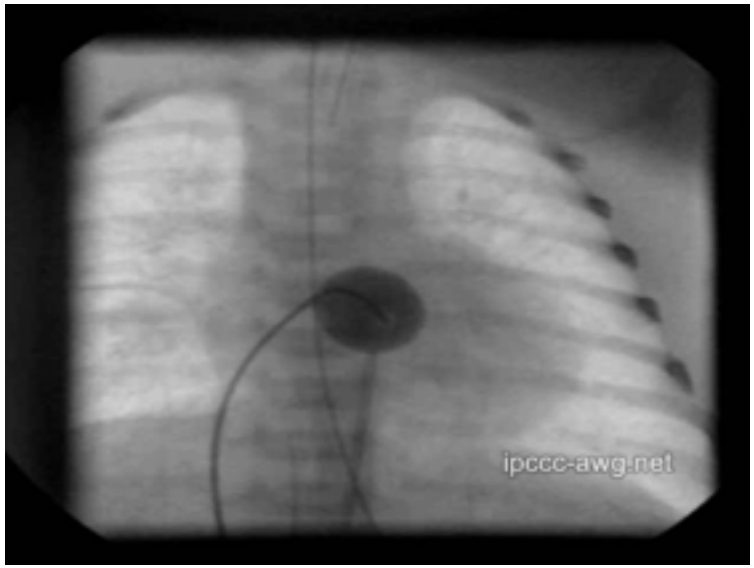
Tratamento

- Manter o canal arterial patente

  PGE_1

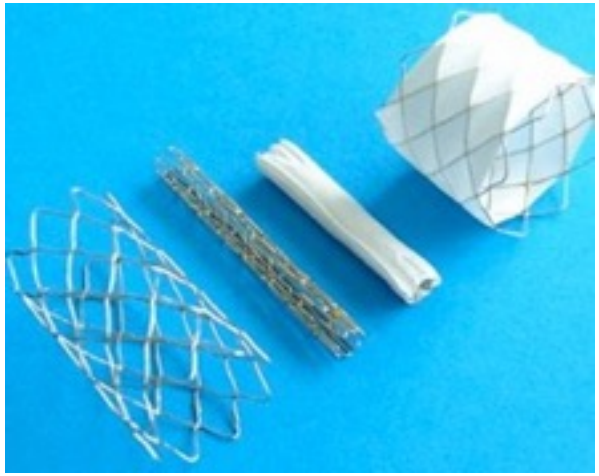
- Abrir o FO por via percutânea: procedimento de Rashkind





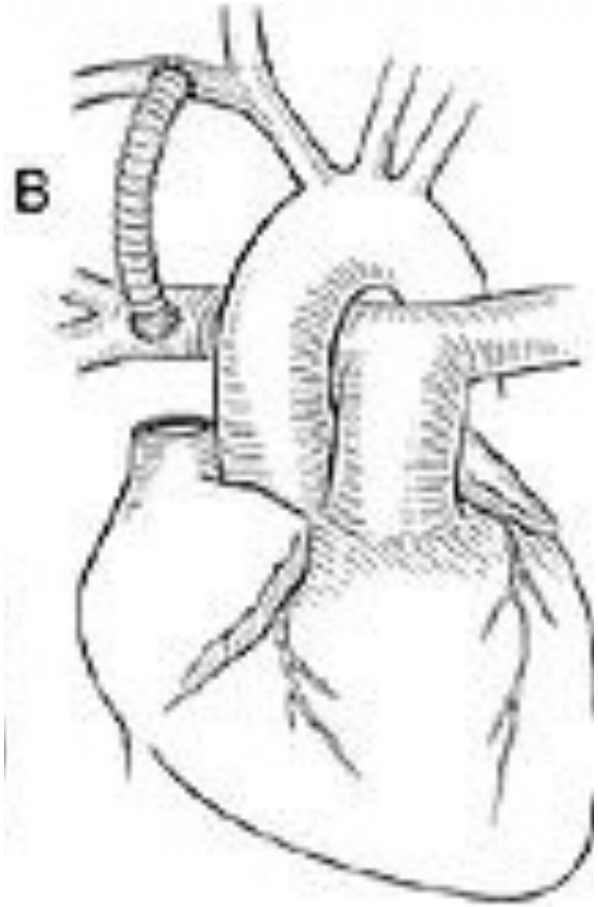
Canal arterial: PGE1

- Apneias
- Febre
- Alterações gastro-intestinais
- Dores articulares
- Hipotensão

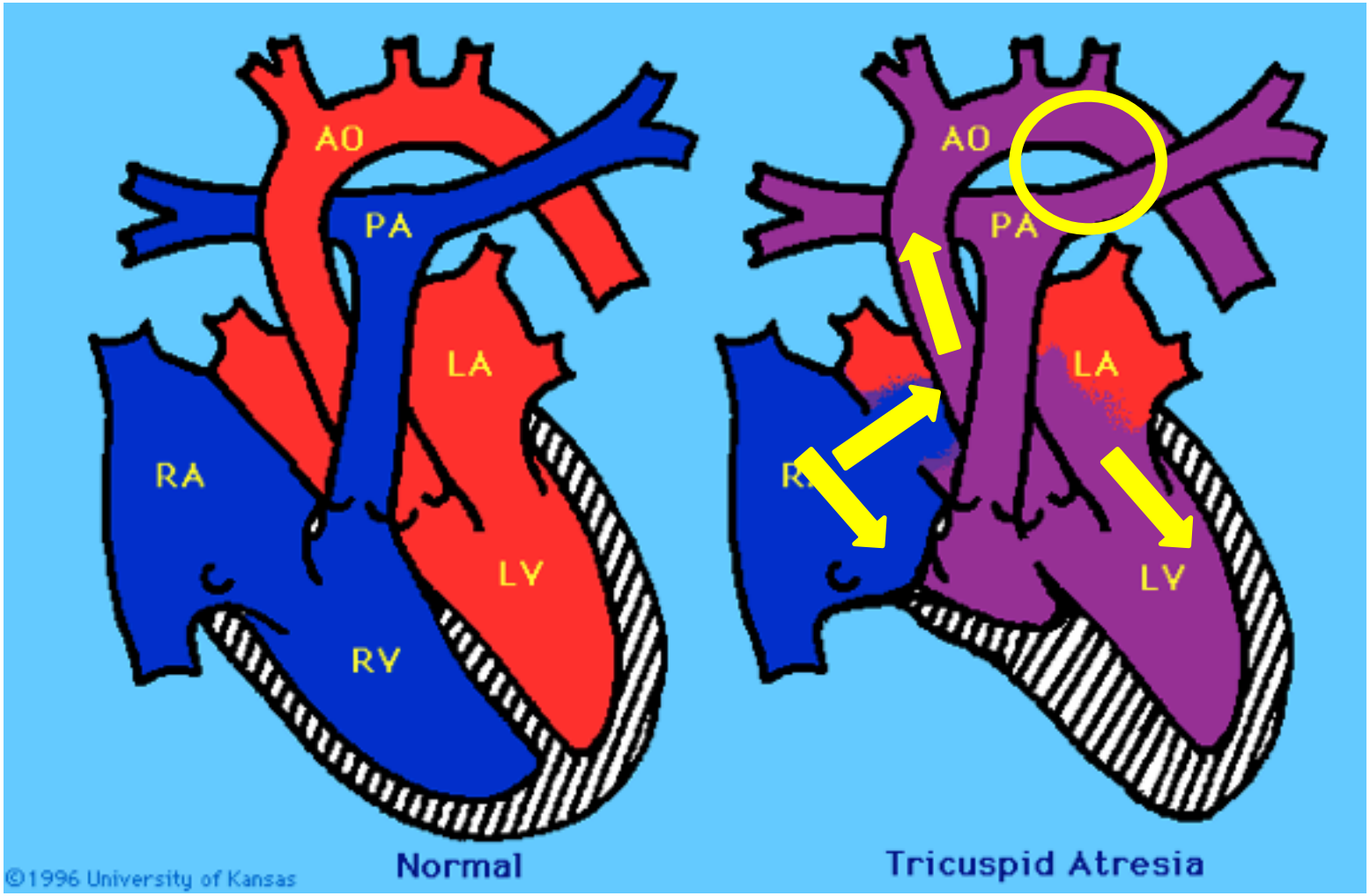


Canal arterial: cirurgia

- Shunt Blalock-Taussig



cardiopatia:cianose + choque cardiogénico



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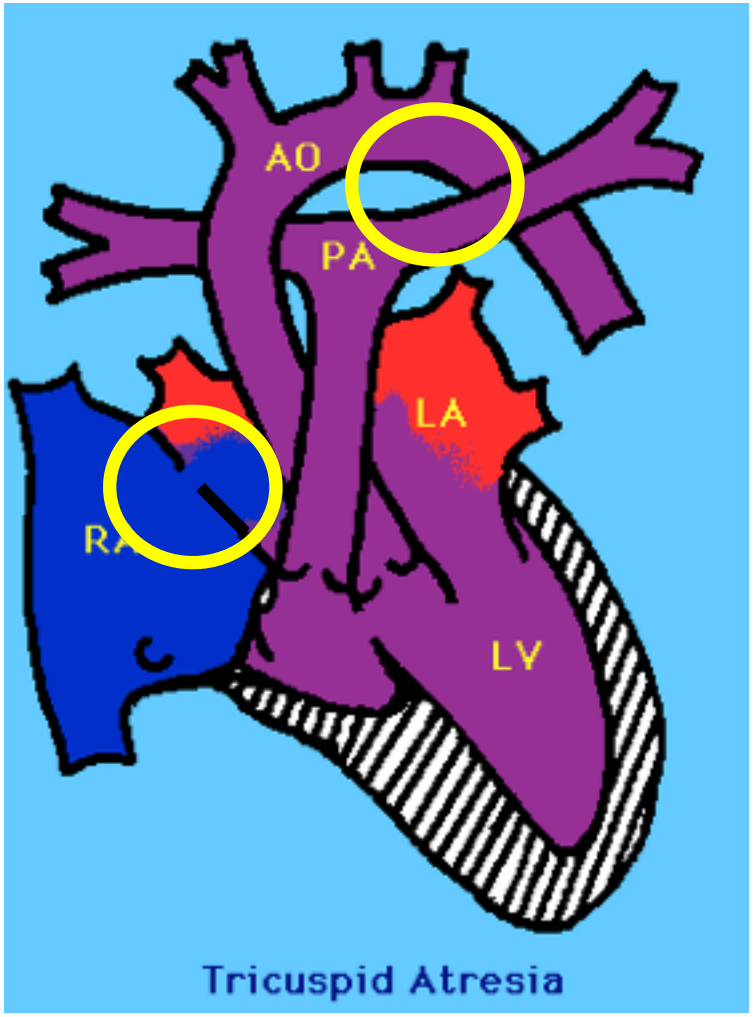
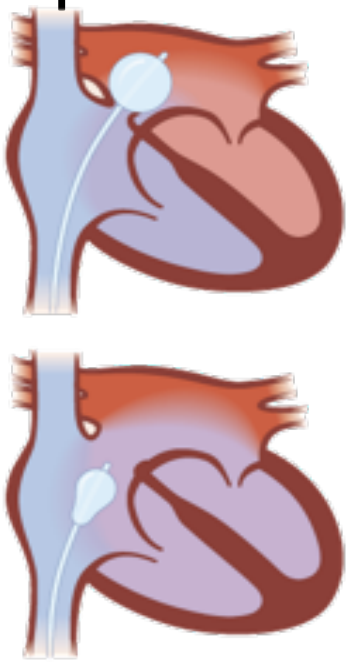


Tratamento

- Manter o canal arterial patente

  PGE_1

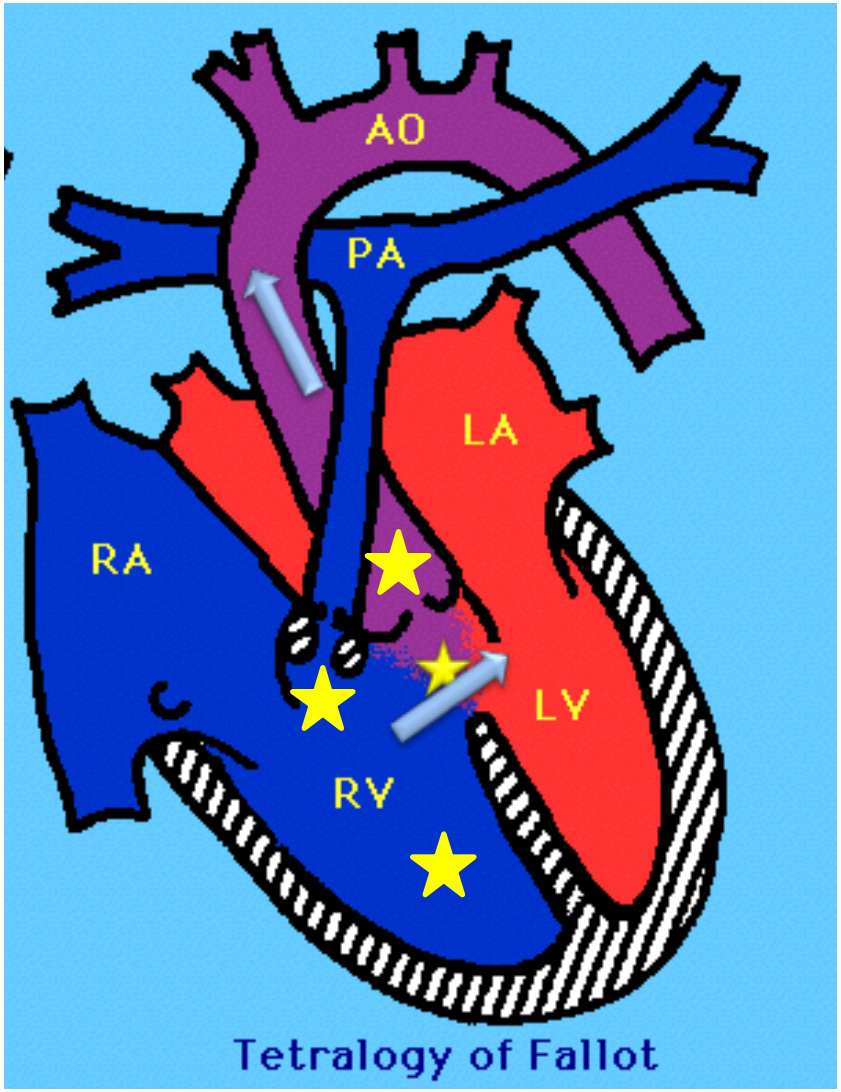
- Abrir o FO por via percutânea: procedimento de Rashkind



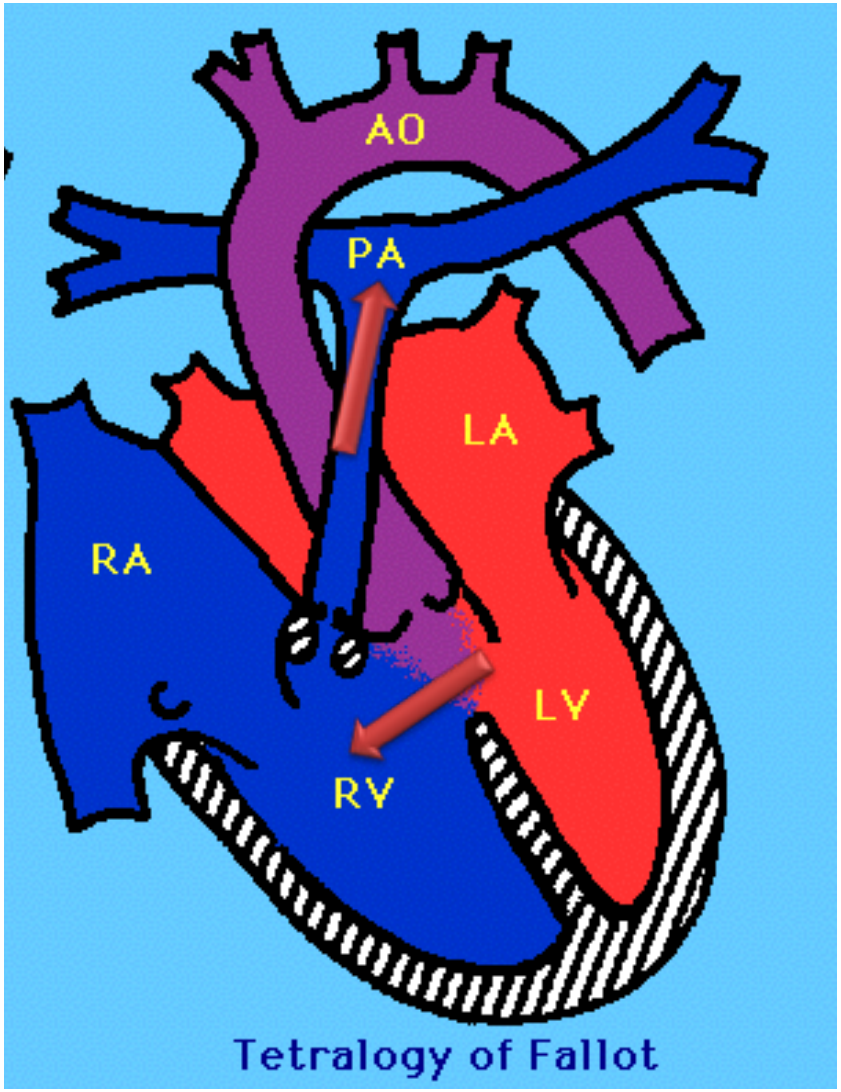
Crise cianótica

- T.Fallot

Pressão
VD=VE



Crise cianótica



HIPÓXIA

↓

PO2 ↓

ACIDOSE — PCO2 ↑

pH ↓

↓

AGITAÇÃO/TAQUIPNEIA

MORTE

Tratamento

- Oxigénio:

  vasodilatador pulmonar

- Posição genu-peitoral

  Aumenta a resistência vascular sistémica

Tratamento

- Reduzir agitação

  morfina

- Tratar a acidose

  Bic. Sódio

- Aumentar a RVS

  Ketamina

- Espasomo infundibular(?)

  Propranolol

Ritmo sinusal

- Nódulo sinusal
- QRS precedido de onda “p”
- Intervalos normais para a idade
- FC ~ idade

Arritmia sinusal

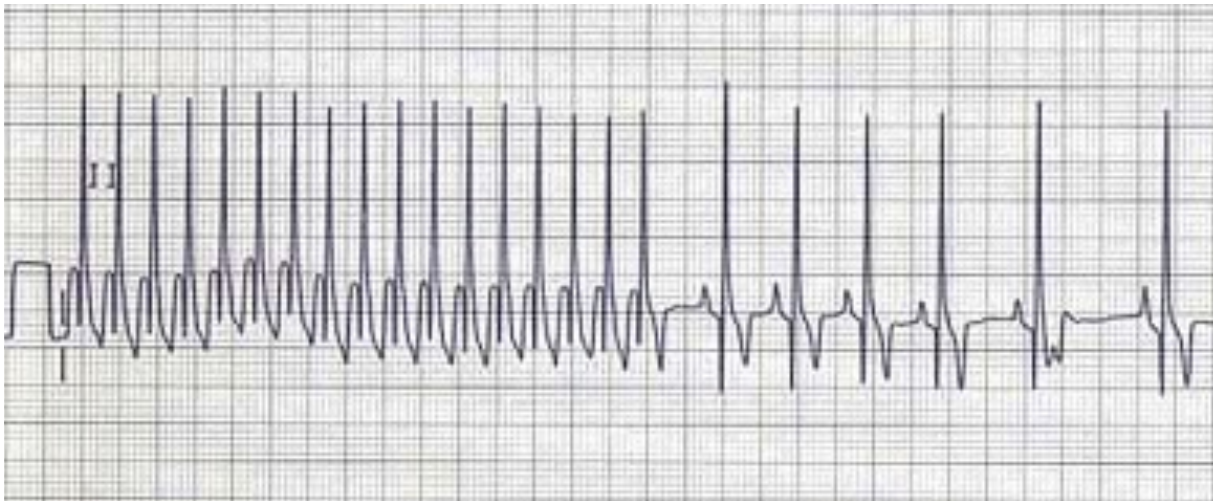
Inspiração **Expiração**

Ciclo respiratório

Activação/Inibição do Vagus

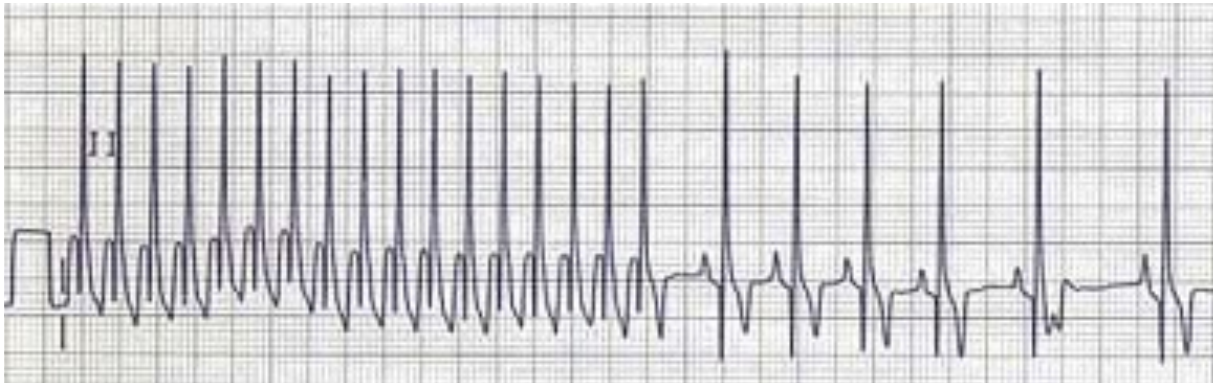
Desaparece c/ apneia

Taquicardia supraventricular



Baixo débito

Tratamento



MANOBRAS VAGAIS

60-90% RESOLUÇÃO ESPONTÂNEA

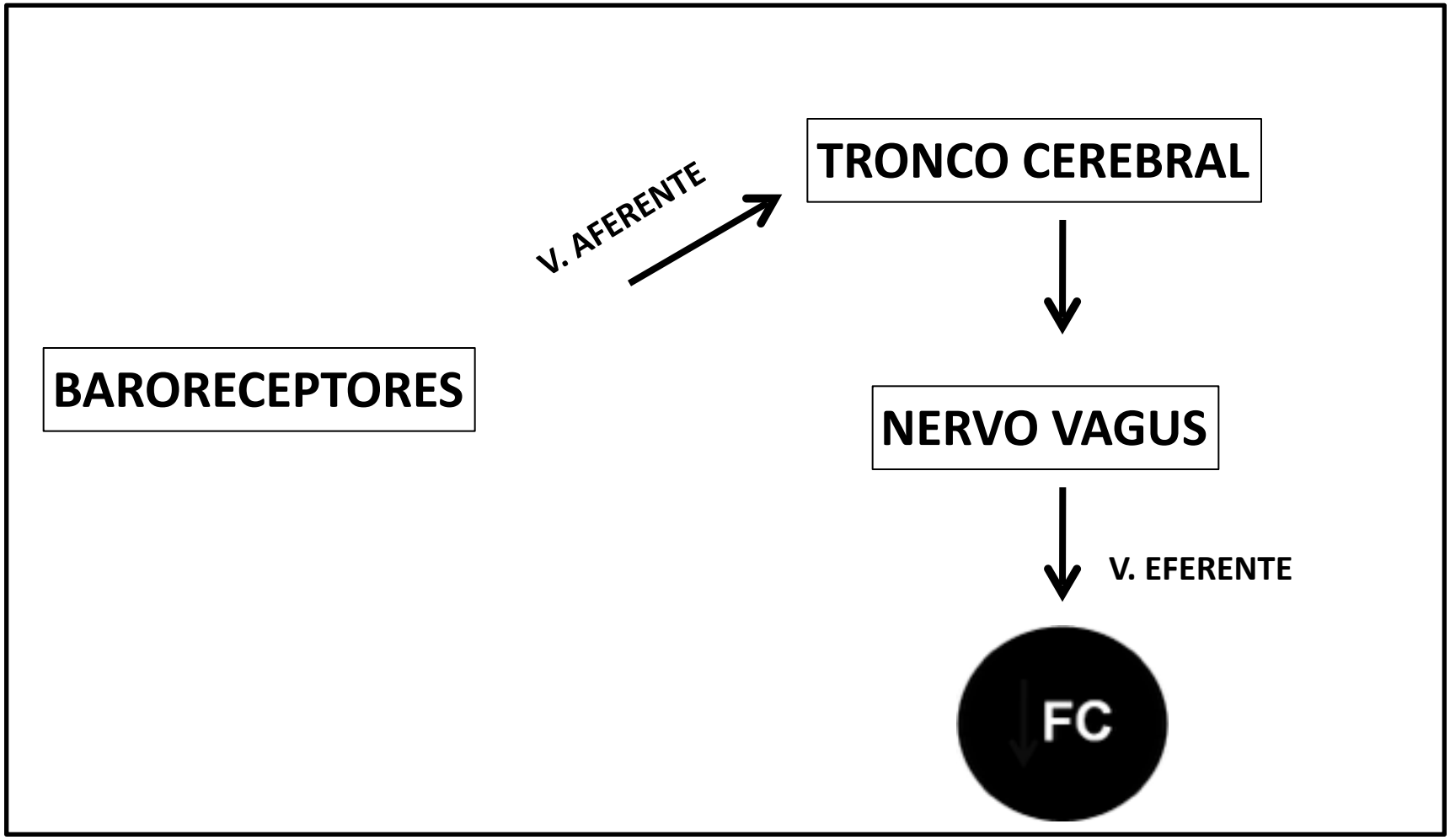
1/3 REACIVAÇÃO

ADENOSINA

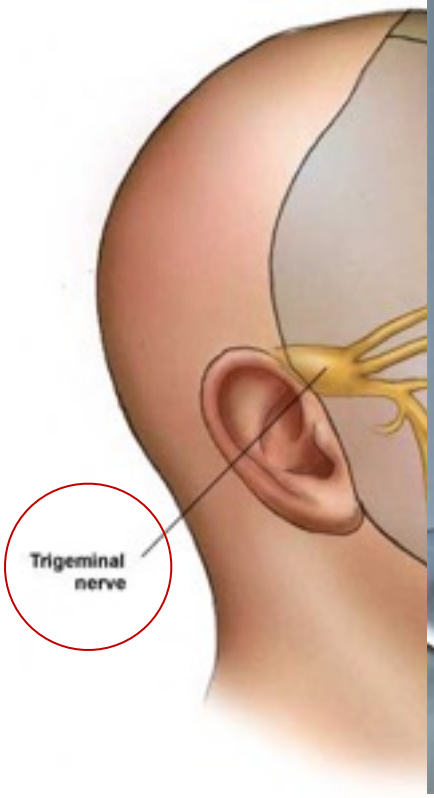
CARDIOVERSÃO

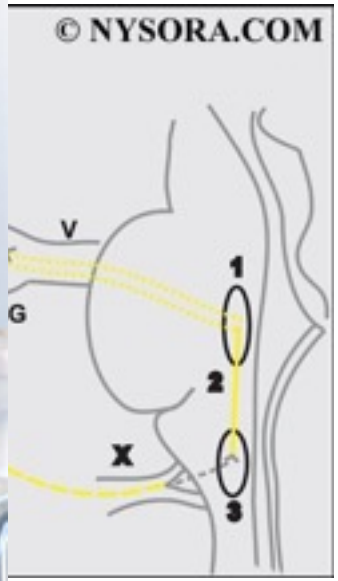
30%

Manobras vagais



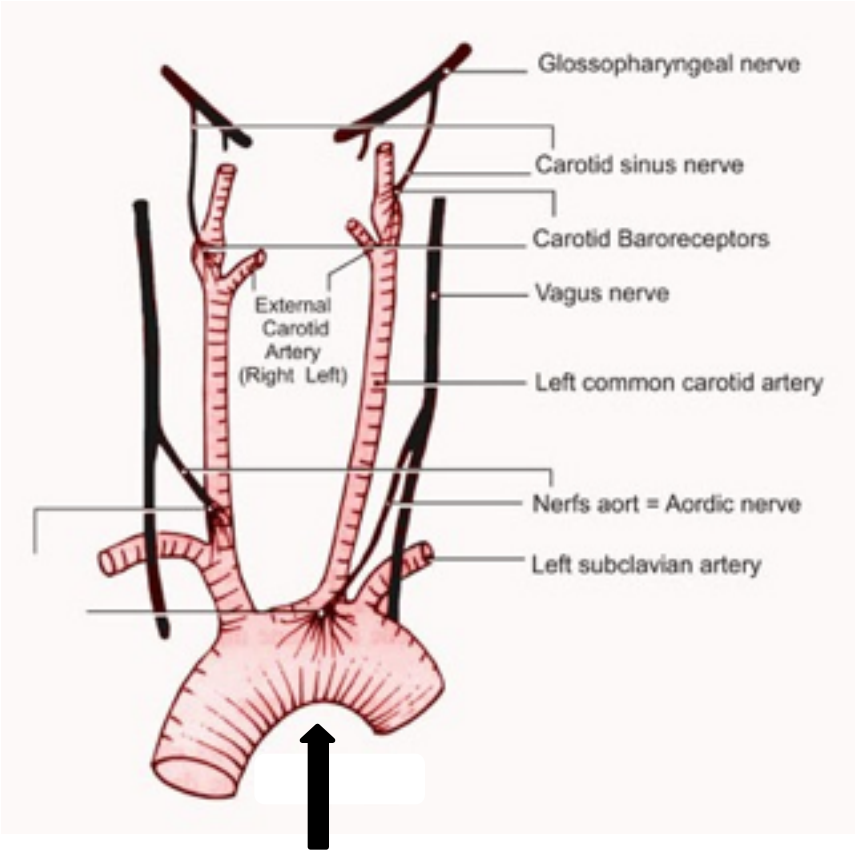
Reflexo do mergulho





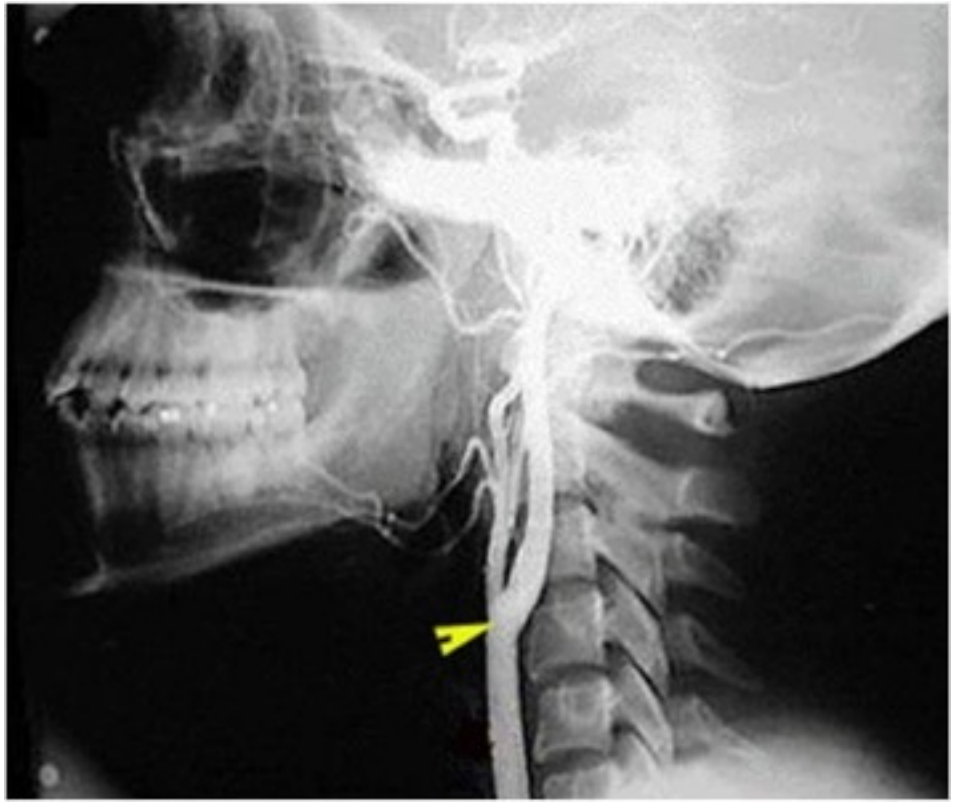
ACTIVAÇÃO DE AFERENTES SENSORIAIS

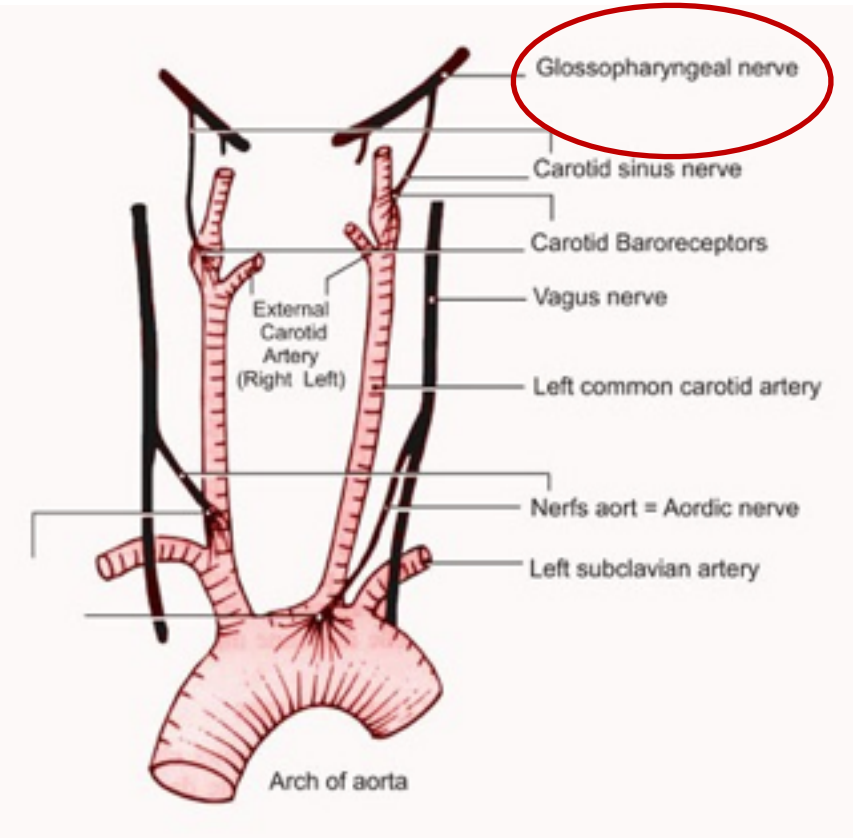
Manobra de Valsalva



PRESSÃO INTRATORÁCICA

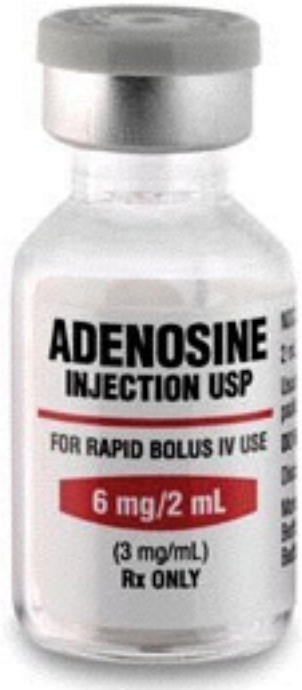
Massagem do seio carotídeo





Massagem unilateral; 5 Segundos

Adenosina



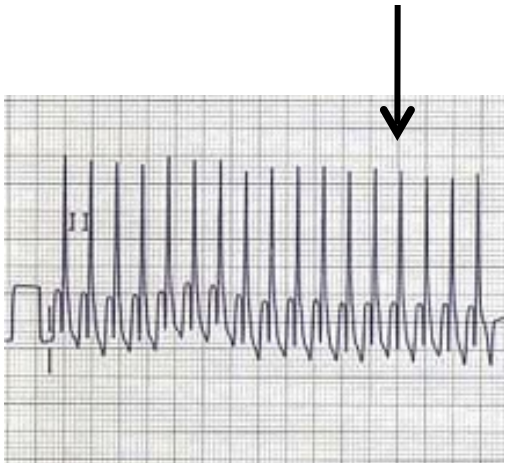
$T^{1/2}$ < 10 seg

90%

IV/IO: 0,1mg/kg/dose (máx. 6mg) – 0,2mg/kg/ dose (máx. 12mg)

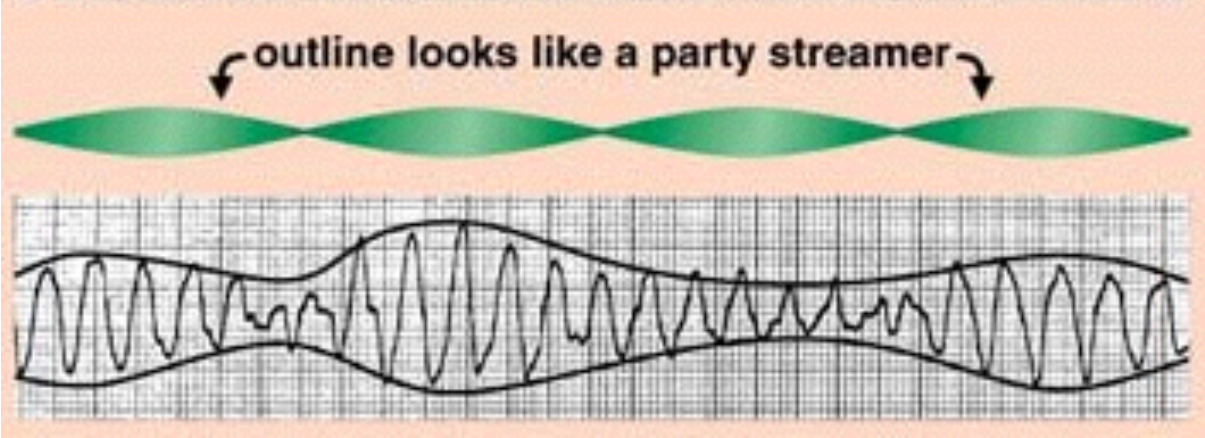
Cardioversão

SINC



0,5-2 j/kg

Torsades de Pointes



S. QT Longo

Sulfato de Magnésio 100mg/kg

Cardioversão

Obrigado!