Heart Failure in Children: Current Management Strategies

Peter C. Laussen, M.B.,B.S.
Chief, Department of Critical Care,
Hospital for Sick Children
Professor of Anaesthesia,
University of Toronto

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Disclosures

- None

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Epidemiology

CARDIOMYOPATHY

CONGENITAL HEART DISEASE

ACQUIRED

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Pathophysiology

Myocyte structure

Systolic & Diastolic Dysfunction

- Volume load
- Pressure load
- Endothelial interactions

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- **Acute heart failure**
  - Post-operative dysfunction
    - Transient- care focused on short-term support
  - Acute cardiogenic shock
    - Resuscitation followed by recovery or chronic care
  - Exacerbation of chronic heart failure
    - Bridge to “recovery” or transplant

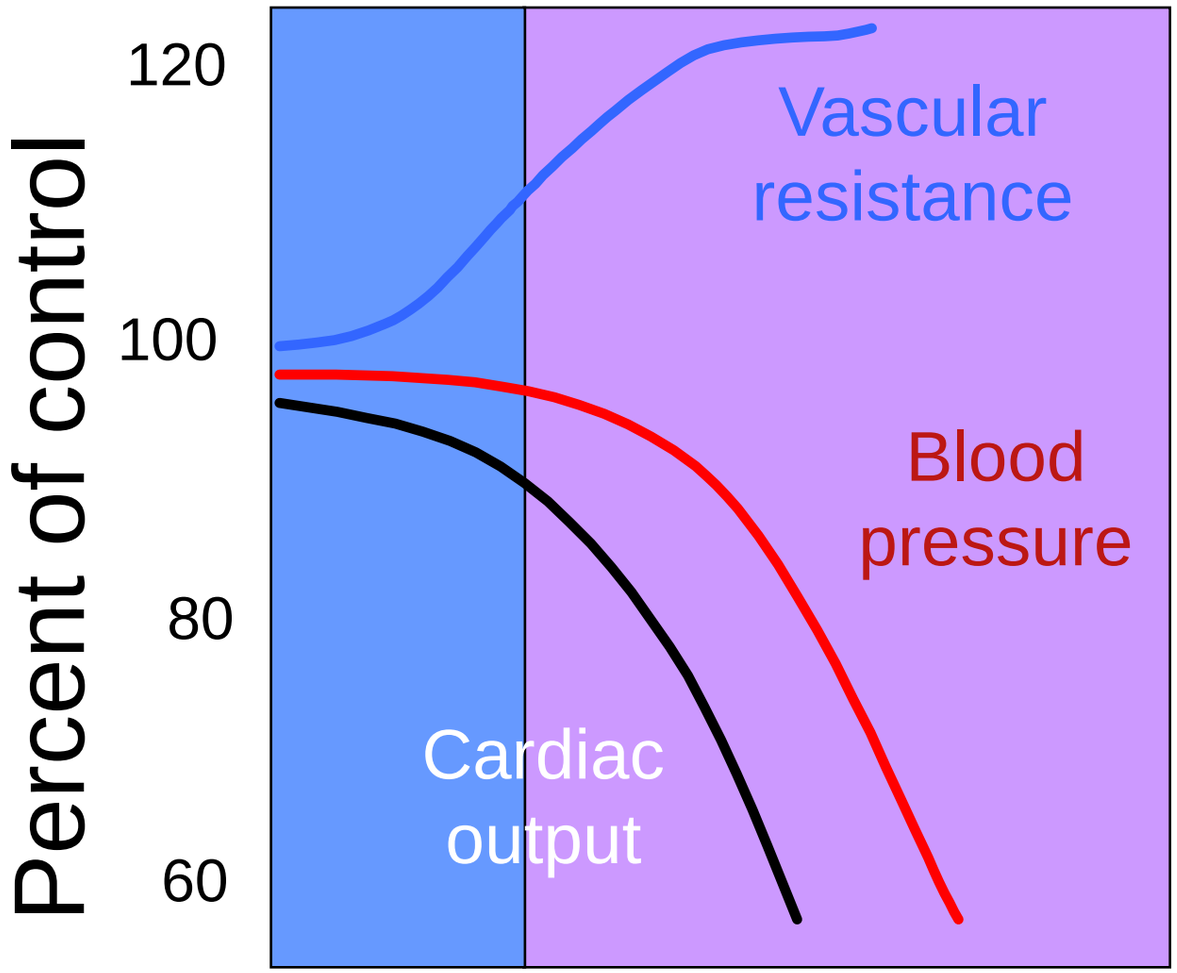
- **Chronic heart failure**
  - Stable but dependent on pharmacology or technology
  - Mechanical support

Blood Pressure ≠ Perfusion

Hypotension occurs late in cardiogenic shock

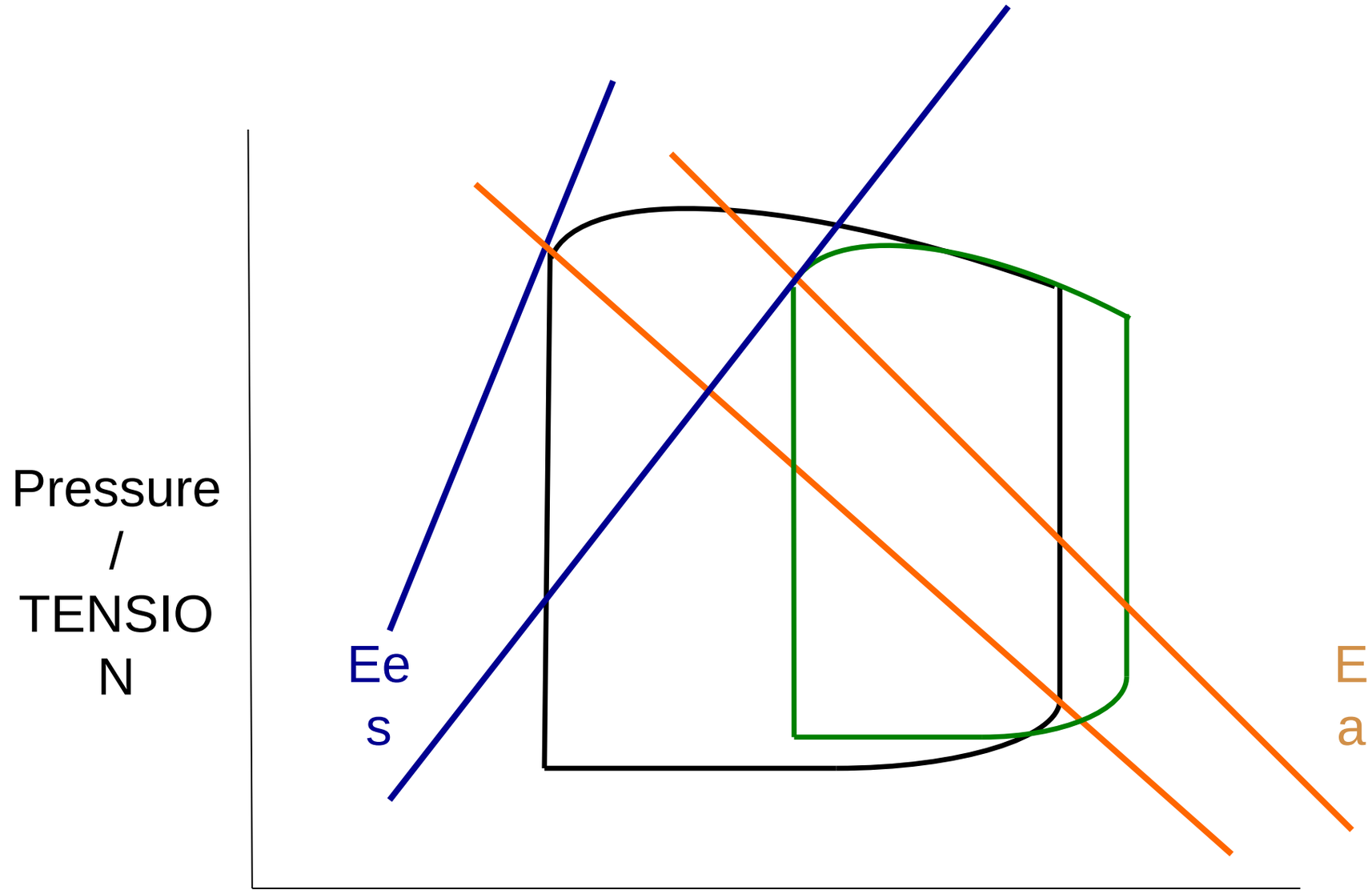
Much easier to manage afterload BEFORE hypotension occurs

Once hypotension is present there are inherent tradeoffs between BP and stroke volume



From Rapid Cardiopulmonary Assessment/Pediatric Advanced Life Support
American Heart Association

Ventricular-Vascular Interactions

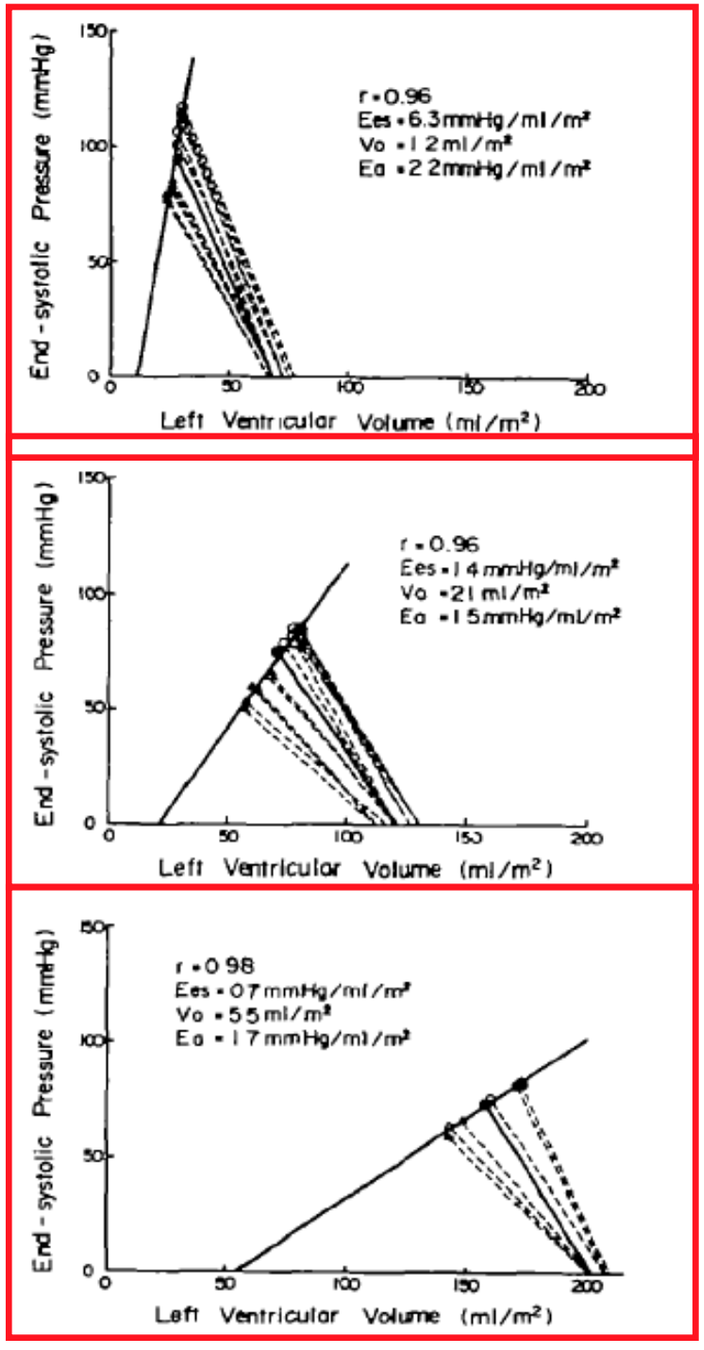


V-A Interactions in Heart Failure

- Normal heart works at peak energy efficiency. Contractility exceeds afterload

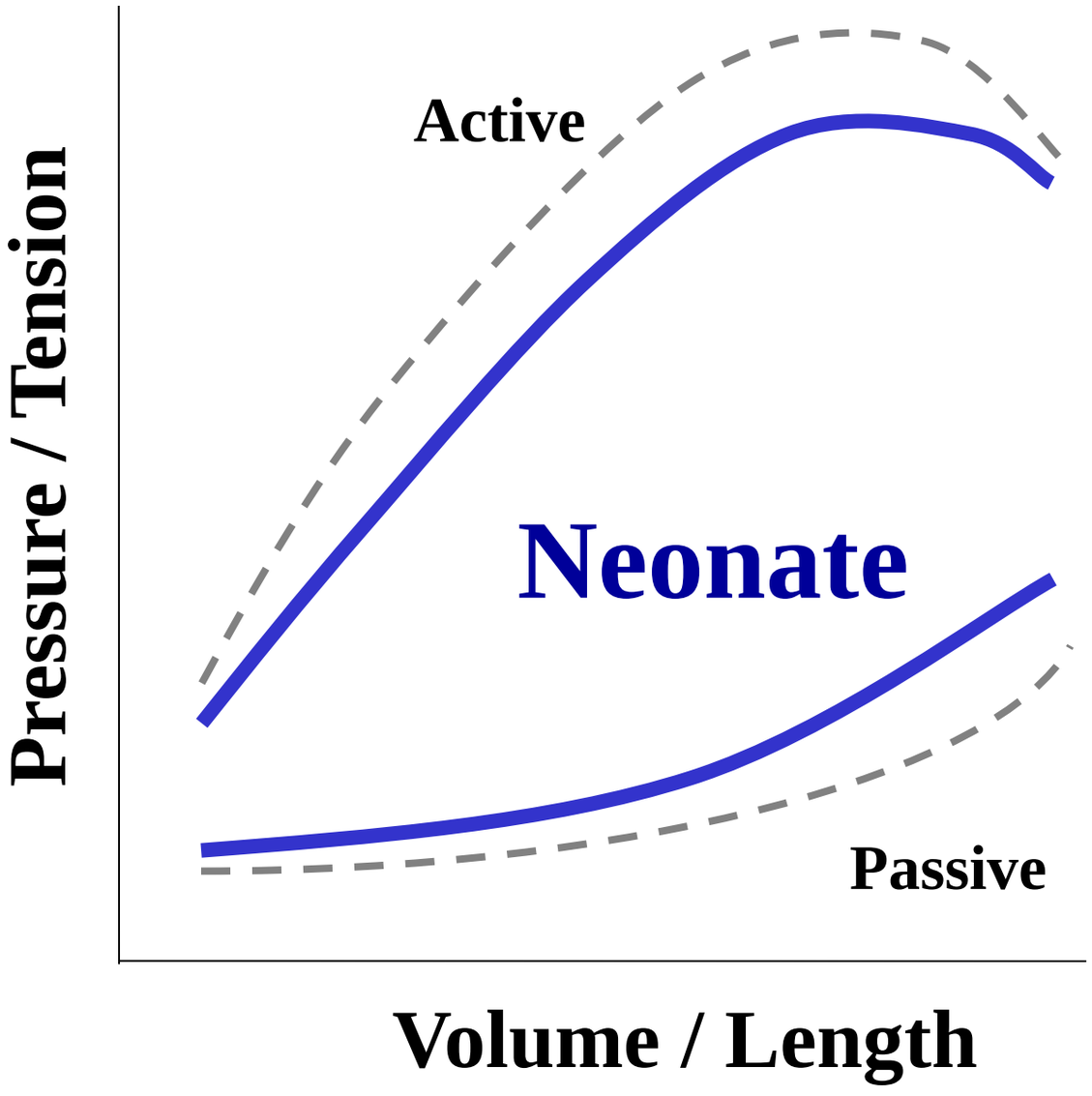
- Compensated failing heart works at peak work efficiency. Contractility matches afterload

- Decompensated heart is inefficient. Contractility cannot match afterload



Asanoi, et al. *Circ Res* 1989;65;483-93

Maturational differences



- Reduced contractile mass,
  $\Rightarrow$ 30% cf. 60% mature

- Expression of Actin & Myosin subtypes

- Intolerant of afterload stress

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Neonatal Myocardium
Calcium handling

- Immature Sarc. retic and T-tubule system
- Cell membrane Na^{+}:Ca^{2+} exchange important for contraction **&** relaxation
- Ca^{2+} influx across membrane initiates and sustains contraction
- Decreased Ca^{2+} pump activity and expression phospholamban (limited phosporylation and Ca^{2+} reuptake)

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LCOS

Balance DO_2 to meet VO_2

- DO_2 well in excess of resting VO_2

Interaction between myocardial performance and endothelial response

Systolic dysfunction
Inadequate fiber shortening

Stretched:

- Volume overload

Decreased fibers:

- Ischemia / myopathy

Inadequate cross-bridging:

- Chemical / receptors
- Developmental
- Edema

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Systolic dysfunction

- Inotropy without excess myocardial VO2

- Volume restriction

- Afterload reduction

- Mechanical ventilation

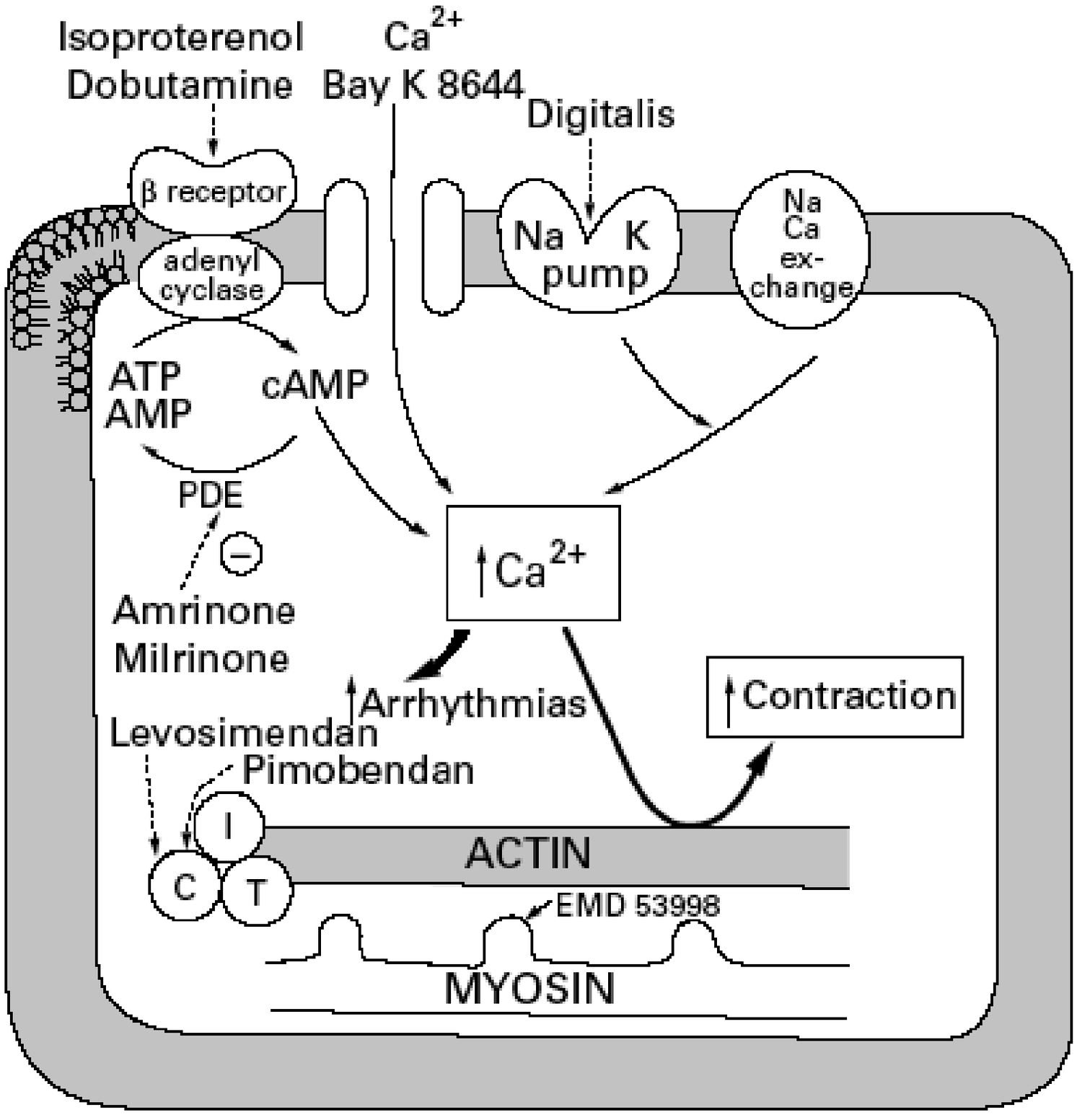
- Mechanical support of the circulation

Traditional Inotropes

- Variable practice
  - Challenge dogma
- Variable patient response
  - Adrenoreceptor polymorphism & down regulation
- Significant consequences related to:
  - impaired calcium homeostasis (ischemia / reperfusion)
  - Arrhythmogenic
  - Impaired diastolic function
- Limited data

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- Optimize handling of intracellular Ca++
- Contractility to exceed or match afterload constraints
- Energy efficient

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Current use

- cAMP mediated increase intracellular Ca^{2+}
  - Adrenergic receptor stimulation
  - PDE III inhibition
- Non-cAMP dependent Ca^{2+} "sensitization"
  - Levosimendan
- Neuro-humoral interactions:
  - Steroids
  - Vasopressin

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Newer approaches

- Novel ways to modify IC Ca++ handling
- Substrate shift: lipid to glucose oxidation
- Endothelium vasoactive peptide: Serelaxin

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Newer approaches

- Receptor and signal polymorphisms

  - Variable expression of AR (particularly α_{2B})
  - Match polymorphism with phenotype
  - Predictive: selection & management

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Mechanical Support of the Circulation

ECMO

VAD:

Pulsatile Vs. Continuous flow

Indications & complications

On the horizon: PumpKIN trial

Specific circumstances

Acute Myocarditis

Single ventricle

Diastolic dysfunction

- **Myocardium:**
  - Hypertrophy
  - Fibrosis
  - Edema: Ischemia / reperfusion
    - Bypass
    - Lymphatic drainage
- **Endocardium**
  - Ischemia
  - Endocardiofibroelastosis EFE
- **Pericardial restriction**

Restrictive physiology

Inotropic function well preserved

Minimize myocardial work / optimize O_2 delivery: Vasopressor / ß-blocker

Caution with afterload reduction, fluid management / diuretics

Mechanical & non-invasive ventilation: wall stress; lung water (redistribution)

Left to right atrial shunt ?

Chronic HF: Treatment

Systolic failure (reduced EF)

FIRST

Diuretic and euvolemia
ACE inhibitors
β Blockers
Aldosterone antagonists

POSSIBLE

Angiotensin receptor blocker
Resynchronization therapy

NOT ROUTINE

Digoxin
Antiarrhythmic
Statins

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Chronic HF: Treatment

Diastolic failure (preserved EF)

FIRST Diuretic : euvolemia

POSSIBLE Non invasive ventilation
Vasopressor (α agonist)

NOT ROUTINE Renin-angiotensin antagonists
Ca channel blockers
Aldosterone blockade
PDE inhibitors
Digoxin
β agonists (dopamine, epi, dobut)

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Summary

- Symptom relief is the primary target

- Need to treat heart failure physiology and biochemistry
  - Diuretics, afterload reduction, positive pressure ventilation
  - ACEI and β-blockers when stable
  - Inotropes for shock

- Early consideration of mechanical support/transplant

Targeted management

Understand what we are trying to treat

Continual reassessment:

- Selected continuous variables

Understand the pharmacokinetics & dynamics, the limitations & complications of the drugs being used