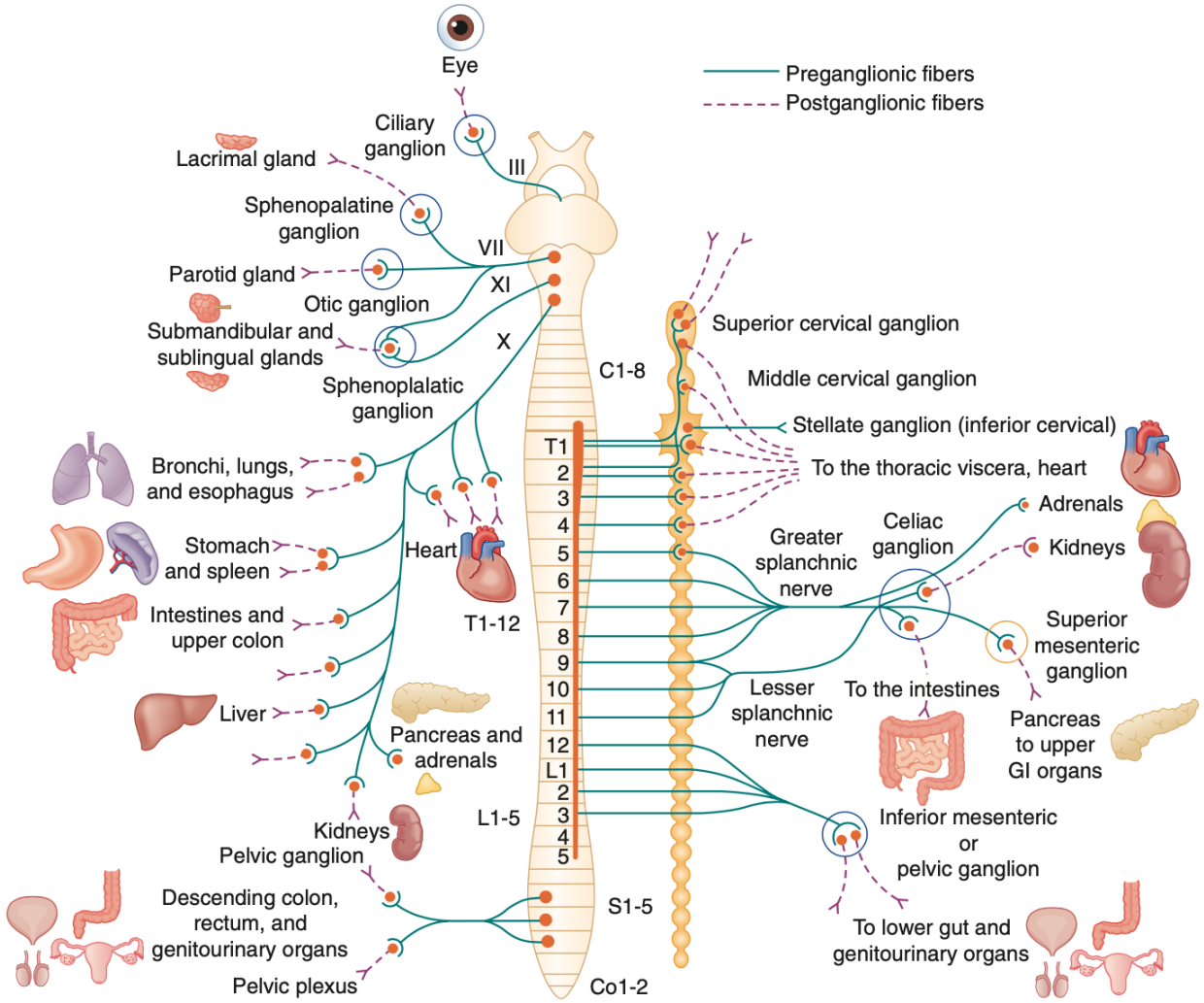
Farmaci di emergenza, vasopressori, inotropi

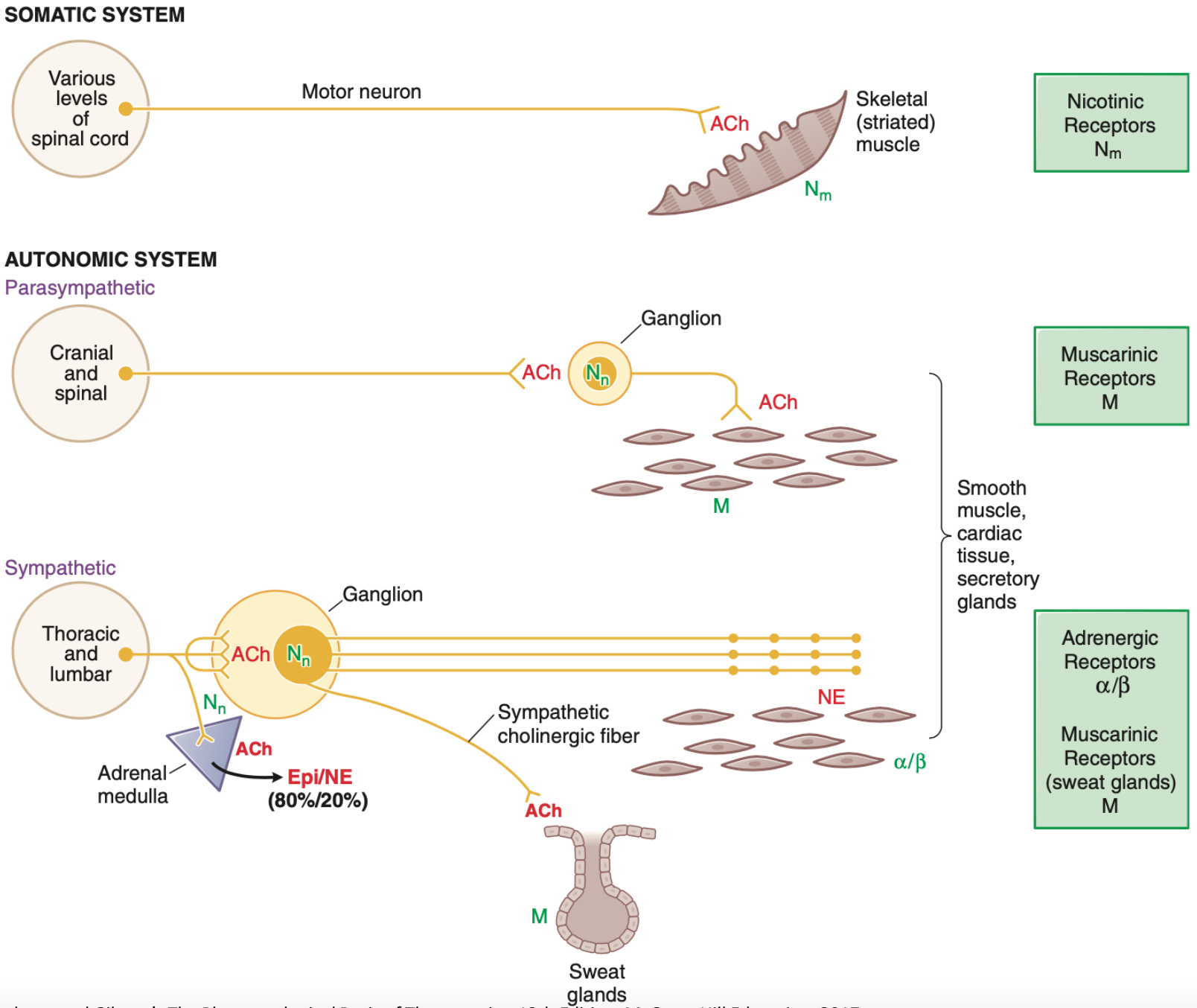
Salvatore Sardo
Università degli studi di Cagliari
salvatore.sardo@unica.it

Sistema nervoso autonomo



• **Fig. 13.1** Schematic representation of the autonomic nervous system consisting of the parasympathetic nervous system *(PNS, left)* and the sympathetic nervous system *(SNS, right)*.

Hemmings HC, Egan TD. Pharmacology and Physiology for Anesthesia E-Book: Foundations and Clinical Application. Elsevier Health Sciences; 2018.



Hilal-Dandan R, Knollmann B, Brunton L. Goodman and Gilman's The Pharmacological Basis of Therapeutics, 13th Edition. McGraw-Hill Education; 2017.

Effetti della stimolazione del SNA

TABLE 8–1 ■ RESPONSES OF EFFECTOR ORGANS TO AUTONOMIC NERVE IMPULSES

ORGAN SYSTEM	SYMPATHETIC EFFECT[a]	ADRENERGIC RECEPTOR SUBTYPE[b]	PARASYMPATHETIC EFFECT[a]	CHOLINERGIC RECEPTOR SUBTYPE[b]
Heart[c]				
Sinoatrial node	↑ heart rate++	$\beta_1 > \beta_2$	↓ heart rate+++	$M_2 >> M_3$
Atria	↑ contractility and conduction velocity++	$\beta_1 > \beta_2$	↓ contractility++ and shortened AP duration	$M_2 >> M_3$
Atrioventricular node	↑ automaticity and conduction velocity++	$\beta_1 > \beta_2$	↓ conduction velocity; AV block+++	$M_2 >> M_3$
His-Purkinje system	↑ automaticity and conduction velocity	$\beta_1 > \beta_2$	Little effect	$M_2 >> M_3$
Ventricle	↑ contractility, conduction velocity, automaticity, and rate of idioventricular pacemakers+++	$\beta_1 > \beta_2$	Slight ↓ in contractility	$M_2 >> M_3$

Hilal-Dandan R, Knollmann B, Brunton L. Goodman and Gilman's The Pharmacological Basis of Therapeutics, 13th Edition. McGraw-Hill Education; 2017.

TABLE 8–1 ■ RESPONSES OF EFFECTOR ORGANS TO AUTONOMIC NERVE IMPULSES

ORGAN SYSTEM	SYMPATHETIC EFFECT[a]	ADRENERGIC RECEPTOR SUBTYPE[b]	PARASYMPATHETIC EFFECT[a]	CHOLINERGIC RECEPTOR SUBTYPE[b]
Blood vessels				
Arteries and arterioles[d]				
Coronary	Constriction+; dilation[e]++	α_1, α_2; β_2	No innervation[h]	—
Skin and mucosa	Constriction+++	α_1, α_2	No innervation[h]	—
Skeletal muscle	Constriction; dilation[e,f]++	α_1; β_2	Dilation[h] (?)	—
Cerebral	Constriction (slight)	α_1	No innervation[h]	—
Pulmonary	Constriction+; dilation	α_1; β_2	No innervation[h]	—
Abdominal viscera	Constriction+++; dilation+	α_1; β_2	No innervation[h]	—
Salivary glands	Constriction+++	α_1, α_2	Dilation[h]++	M_3
Renal	Constriction++; dilation++	α_1, α_2; β_1, β_2	No innervation[h]	
(Veins)[d]	Constriction; dilation	α_1, α_2; β_2		
Endothelium	—	—	↑ NO synthase[h]	M_3
Lung				
Tracheal and bronchial smooth muscle	Relaxation	β_2	Contraction	$M_2 = M_3$
Bronchial glands	↓ secretion, ↑ secretion	α_1	Stimulation	M_2, M_3
		β_2		

Hilal-Dandan R, Knollmann B, Brunton L. Goodman and Gilman's The Pharmacological Basis of Therapeutics, 13th Edition. McGraw-Hill Education; 2017.

TABLE 8–1 ■ RESPONSES OF EFFECTOR ORGANS TO AUTONOMIC NERVE IMPULSES

ORGAN SYSTEM	SYMPATHETIC EFFECT[a]	ADRENERGIC RECEPTOR SUBTYPE[b]	PARASYMPATHETIC EFFECT[a]	CHOLINERGIC RECEPTOR SUBTYPE[b]
Autonomic nerve endings				
Sympathetic terminal				
Autoreceptor	Inhibition of NE release	$\alpha_{2A} > \alpha_{2C}(\alpha_{2B})$		
Heteroreceptor	—		Inhibition of NE release	M_2, M_4
Parasympathetic terminal				
Autoreceptor	—	—	Inhibition of ACh release	M_2, M_4
Heteroreceptor	Inhibition ACh release	$\alpha_{2A} > \alpha_{2C}$	—	—

Hilal-Dandan R, Knollmann B, Brunton L. Goodman and Gilman's The Pharmacological Basis of Therapeutics, 13th Edition. McGraw-Hill Education; 2017.

Recettori adrenergici α_1

	G PROTEIN COUPLING	PRINCIPLE EFFECTORS	TISSUE LOCALIZATION	DOMINANT EFFECTS[b]
α_{1A}	$G\alpha_q$ ($\alpha_{11}/\alpha_{14}/\alpha_{16}$)	↑ PLC, ↑ PLA_2 ↑ Ca^{2+} channels ↑ Na^+/H^+ exchanger Modulation of K^+ channels ↑ MAPK Signaling	Heart, lung Liver Smooth muscle Blood vessels Vas deferens, prostate Cerebellum, cortex Hippocampus	• Dominant receptor for contraction of vascular smooth muscle • Promotes cardiac growth and structure • Vasoconstriction of large resistant arterioles in skeletal muscle
α_{1B}	$G\alpha_q$ ($\alpha_{11}/\alpha_{14}/\alpha_{16}$)	↑ PLC, ↑ PLA_2 ↑ Ca^{2+} channels ↑ Na^+/H^+ exchanger Modulation of K^+ channels ↑ MAPK signaling	Kidney, lung Spleen Blood vessels Cortex Brainstem	• Most abundant subtype in heart • Promotes cardiac growth and structure
α_{1D}	$G\alpha_q$ ($\alpha_{11}/\alpha_{14}/\alpha_{16}$)	↑ PLC, ↑ PLA_2 ↑ Ca^{2+} channels ↑ Na^+/H^+ exchanger Modulation of K^+ channels ↑ MAPK signaling	Platelets, aorta Coronary artery Prostate Cortex Hippocampus	• Dominant receptor for vasoconstriction in aorta and coronaries

Hilal-Dandan R, Knollmann B, Brunton L. Goodman and Gilman's The Pharmacological Basis of Therapeutics, 13th Edition. McGraw-Hill Education; 2017.

Recettori adrenergici α_2

α_{2A}	$G\alpha_i$ $G\alpha_o$ (α_{o1},/α_{o2})	↓ AC-cAMP-PKA pathway	Platelets Sympathetic neurons Autonomic ganglia Pancreas Coronary/CNS vessels Locus ceruleus Brainstem, spinal cord	• Dominant inhibitory receptor on sympathetic neurons • Vasoconstriction of precapillary vessels in skeletal muscle
α_{2B}	$G\alpha_i$ $G\alpha_o$ (α_{o1},/α_{o2})	↓ AC-cAMP-PKA pathway	Liver, kidney Blood vessels Coronary/CNS vessels Diencephalon Pancreas, platelets	• Dominant mediator of α_2 vasoconstriction
α_{2C}	$G\alpha_i$ (α_{11}/α_{12}/α_{13}) $G\alpha_o$ (α_{o1}/α_{o2})	↓ AC-cAMP-PKA pathway	Basal ganglia Cortex, cerebellum Hippocampus	• Dominant receptor modulating DA neurotransmission • Dominant receptor inhibiting hormone release from adrenal medulla

Hilal-Dandan R, Knollmann B, Brunton L. Goodman and Gilman's The Pharmacological Basis of Therapeutics, 13th Edition. McGraw-Hill Education; 2017.

Recettori adrenergici β

β_1	$G\alpha_s$	↑ AC-cAMP-PKA pathway ↑ L-type Ca^{2+} channels	Heart, kidney Adipocytes Skeletal muscle Olfactory nucleus Cortex, brainstem Cerebellar nuclei Spinal cord	• Dominant mediator of positive inotropic and chronotropic effects in heart
β_2[c]	$G\alpha_s$	↑ AC-cAMP-PKA pathway ↑ Ca^{2+} channels	Heart, lung, kidney Blood vessels Bronchial smooth muscle GI smooth muscle Skeletal muscle Olfactory bulb Cortex, hippocampus	• Smooth muscle relaxation • Skeletal muscle hypertrophy
β_3[c,d]	$G\alpha_s$	↑ AC-cAMP-PKA pathway ↑ Ca^{2+} channels	Adipose tissue GI tract, heart	• Metabolic effects

Hilal-Dandan R, Knollmann B, Brunton L. Goodman and Gilman's The Pharmacological Basis of Therapeutics, 13th Edition. McGraw-Hill Education; 2017.

Agonisti adrenergici

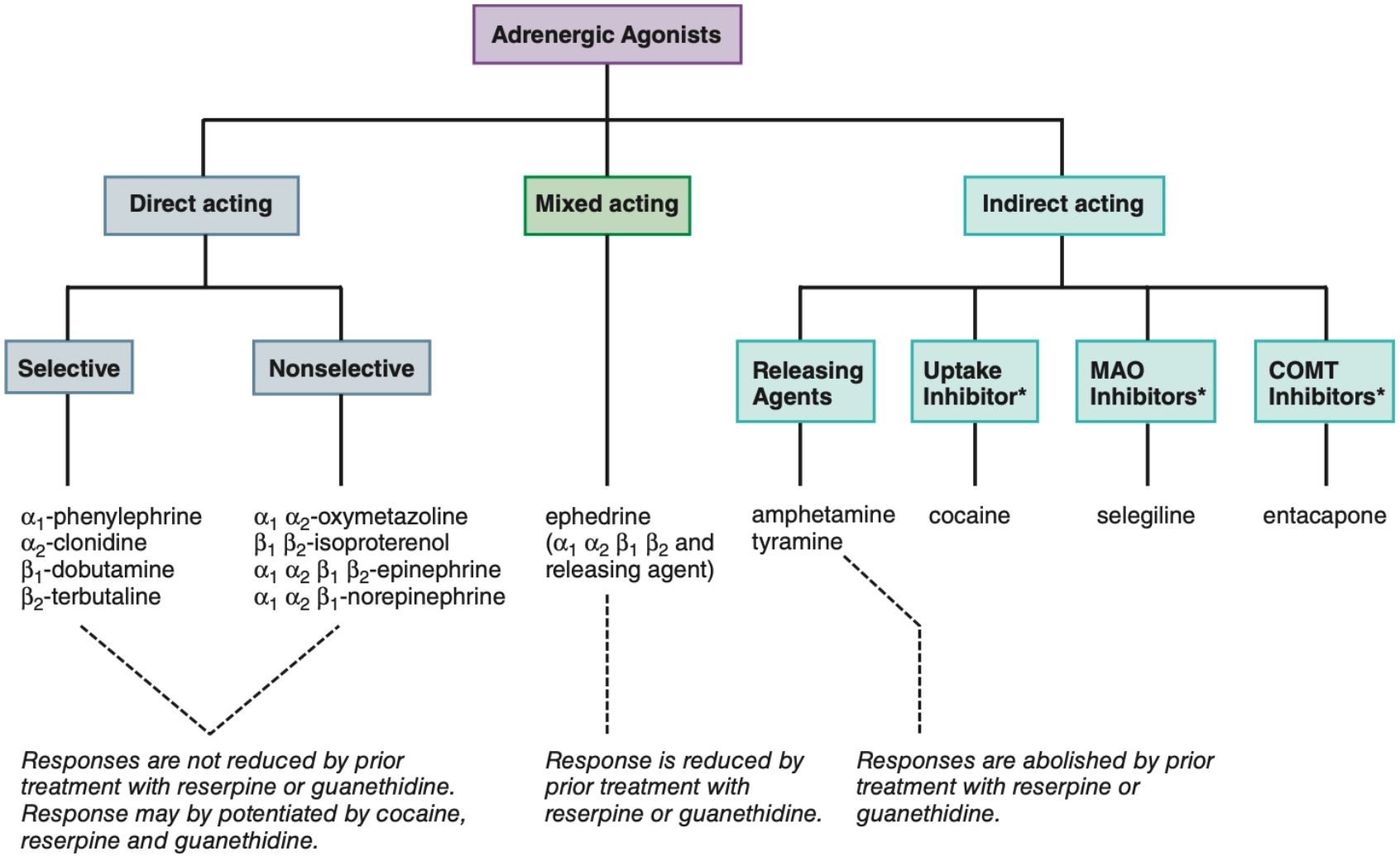


Figure 12–1 *Classification of adrenergic receptor agonists (sympathomimetic amines) or drugs that produce sympathomimetic-like effects.* For each category, a prototypical drug is shown. (*Not actually sympathetic drugs but produce sympathomimetic-like effects.)

Hilal-Dandan R, Knollmann B, Brunton L. Goodman and Gilman's The Pharmacological Basis of Therapeutics, 13th Edition. McGraw-Hill Education; 2017.

Biosintesi degli agonisti adrenergici endogeni

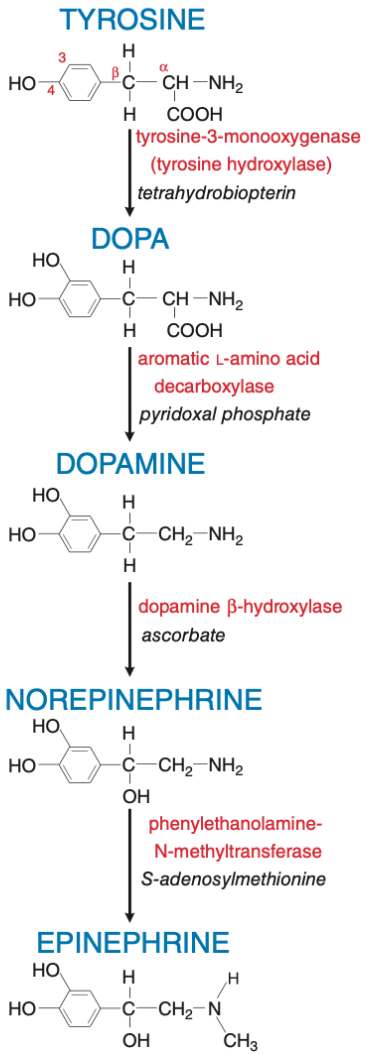


Figure 8–7 *Steps in the enzymatic synthesis of dopamine, norepinephrine and epinephrine.* The enzymes involved are shown in red; essential cofactors in italics. The final step occurs only in the adrenal medulla and in a few epinephrine-containing neuronal pathways in the brainstem.

Hilal-Dandan R, Knollmann B, Brunton L. Goodman and Gilman's The Pharmacological Basis of Therapeutics, 13th Edition. McGraw-Hill Education; 2017.

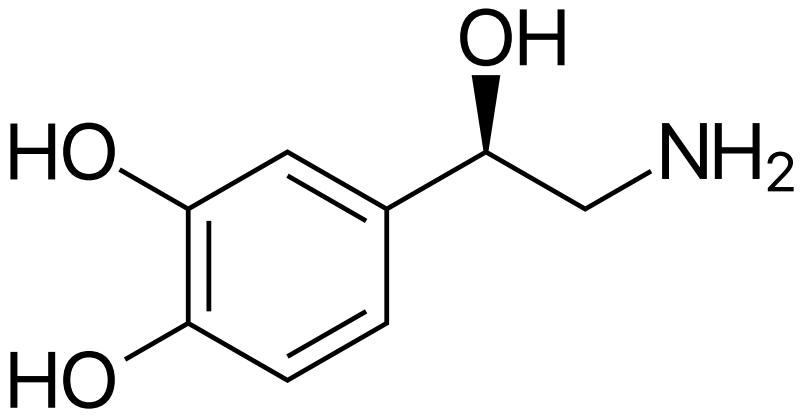
Domande o interventi?

Catecolamine: uso clinico

Drug	Clinical Indication	Dose Range	Receptor Binding				Major Side Effects
			α1	β1	β2	DA	
Catecholamines							
Dopamine	Shock (cardiogenic, vasodilatory) HF Symptomatic bradycardia unresponsive to atropine or pacing	2.0 to 20 μg · kg^{-1} · min^{-1} (max 50 μg · kg^{-1} · min^{-1})	+++	++++	++	+++++	Severe hypertension (especially in patients taking nonselective β-blockers) Ventricular arrhythmias Cardiac ischemia Tissue ischemia/gangrene (high doses or due to tissue extravasation)
Dobutamine	Low CO (decompensated HF, cardiogenic shock, sepsis-induced myocardial dysfunction) Symptomatic bradycardia unresponsive to atropine or pacing	2.0 to 20 μg · kg^{-1} · min^{-1} (max 40 μg · kg^{-1} · min^{-1})	+	+++++	+++	N/A	Tachycardia Increased ventricular response rate in patients with atrial fibrillation Ventricular arrhythmias Cardiac ischemia Hypertension (especially nonselective β-blocker patients) Hypotension
Norepinephrine	Shock (vasodilatory, cardiogenic)	0.01 to 3 μg · kg^{-1} · min^{-1}	+++++	+++	++	N/A	Arrhythmias Bradycardia Peripheral (digital) ischemia Hypertension (especially nonselective β-blocker patients)
Epinephrine	Shock (cardiogenic, vasodilatory) Cardiac arrest Bronchospasm/anaphylaxis Symptomatic bradycardia or heart block unresponsive to atropine or pacing	Infusion: 0.01 to 0.10 μg · kg^{-1} · min^{-1} Bolus: 1 mg IV every 3 to 5 min (max 0.2 mg/kg) IM: (1:1000): 0.1 to 0.5 mg (max 1 mg)	+++++	++++	+++	N/A	Ventricular arrhythmias Severe hypertension resulting in cerebrovascular hemorrhage Cardiac ischemia Sudden cardiac death
Isoproterenol	Bradyarrhythmias (especially torsade des pointes) Brugada syndrome	2 to 10 μg/min	0	+++++	+++++	N/A	Ventricular arrhythmias Cardiac ischemia Hypertension Hypotension
Phenylephrine	Hypotension (vagally mediated, medication-induced) Increase MAP with AS and hypotension Decrease LVOT gradient in HCM	Bolus: 0.1 to 0.5 mg IV every 10 to 15 min Infusion: 0.4 to 9.1 μg · kg^{-1} · min^{-1}	+++++	0	0	N/A	Reflex bradycardia Hypertension (especially with nonselective β-blockers) Severe peripheral and visceral vasoconstriction Tissue necrosis with extravasation

Overgaard CB, Dzavík V. Inotropes and vasopressors: review of physiology and clinical use in cardiovascular disease. Circulation. 2008;118(10):1047-1056. doi:10.1161/CIRCULATIONAHA.107.728840

Noradrenalina



4–12 µg/min effetti α1 e β dose dipendenti

nessun effetto significativo β2

a bassa dose prevale effetto β1, ↑CO

a dose maggiore effetto α1 vasocostrizione arteriosa e venosa nel circolo epatico, muscolare, splancnico, renale,

↓HR e CO (bradicardia riflessa)

Meno aritmogena di ADR

Dosaggio

0.01 – 1 µg/kg/min (IBW)

Diluzione

8 mg NAD Tartrato / 50 ml Glucosio 5%

Hemmings HC, Egan TD. Pharmacology and Physiology for Anesthesia E-Book: Foundations and Clinical Application. Elsevier Health Sciences; 2018

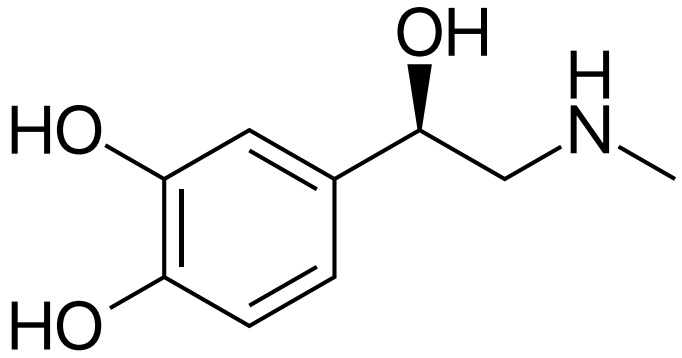
Noradrenalina

DOSE EQUIVALENCE AND CONVERSION

- 1 mg of noradrenaline base is equivalent to 2 mg of noradrenaline acid tartrate. **Doses expressed as the base.**

BNF 80: September 2020-March 2021. (2020). Regno Unito: Pharmaceutical Press.

Adrenalina



Monoamina endogena (cellule cromaffini surrenali)

Secrezione basale di ADR 0.2 µg/kg/min e NAD 0.05 µg/kg/min

2–10 µg/min → effetto prevalente β1 – β2 ↑HR, ↑contrattilità, venocostrizione, vasodilatazione muscolare, ↑metabolismo x2, ↑glicogenolisi

>10 µg/min → stimolo α vasocostrizione di cute, mucose, rene, muscoli, letto splancnico

Dosaggio

- 0.01 – 1 µg /kg/min IV (IBW)
- bolo 1 mg IV per ARRESTO CARDIOCIRCOLATORIO
- bolo 0.5 mg IM / 20–50 **µg IV** per anafilassi

Diluzione 2 mg/ 50 ml (4 **µg** /ml) in Glucosio 5%

Hemmings HC, Egan TD. Pharmacology and Physiology for Anesthesia E-Book: Foundations and Clinical Application. Elsevier Health Sciences; 2018
Lott C, Truhlář A, Alfonzo A, et al. European Resuscitation Council Guidelines 2021: Cardiac arrest in special circumstances. Resuscitation. 2021;161:152-219. doi:10.1016/j.resuscitation.2021.02.011 .

Adrenalina e Noradrenalina

TABLE 12–2 ■ COMPARATIVE EFFECTS OF INFUSIONS OF EPINEPHRINE AND NOREPINEPHRINE IN HUMAN BEINGS[a]

EFFECT	EPI	NE
Cardiac		
Heart rate	+	–[b]
Stroke volume	++	++
Cardiac output	+++	0, –
Arrhythmias	++++	++++
Coronary blood flow	++	++
Blood pressure		
Systolic arterial	+++	+++
Mean arterial	+	++
Diastolic arterial	+, 0, –	++
Mean pulmonary	++	++
Peripheral circulation		
Total peripheral resistance	–	++
Cerebral blood flow	+	0, –
Muscle blood flow	+++	0, –
Cutaneous blood flow	–	–
Renal blood flow	–	–
Splanchnic blood flow	+++	0,+
Metabolic effects		
Oxygen consumption	++	0, +
Blood glucose	+++	0, +
Blood lactic acid	+++	0, +
Eosinopenic response	+	0
CNS		
Respiration	+	+
Subjective sensations	+	+

+, increase; 0, no change; –, decrease. Data from Goldenberg M, et al. *Arch Intern Med.* **1950**;*86*:823.

[a]0.1–0.4 μg/kg per minute.

[b]After atropine.

Hilal-Dandan R, Knollmann B, Brunton L. Goodman and Gilman's The Pharmacological Basis of Therapeutics, 13th Edition. McGraw-Hill Education; 2017.

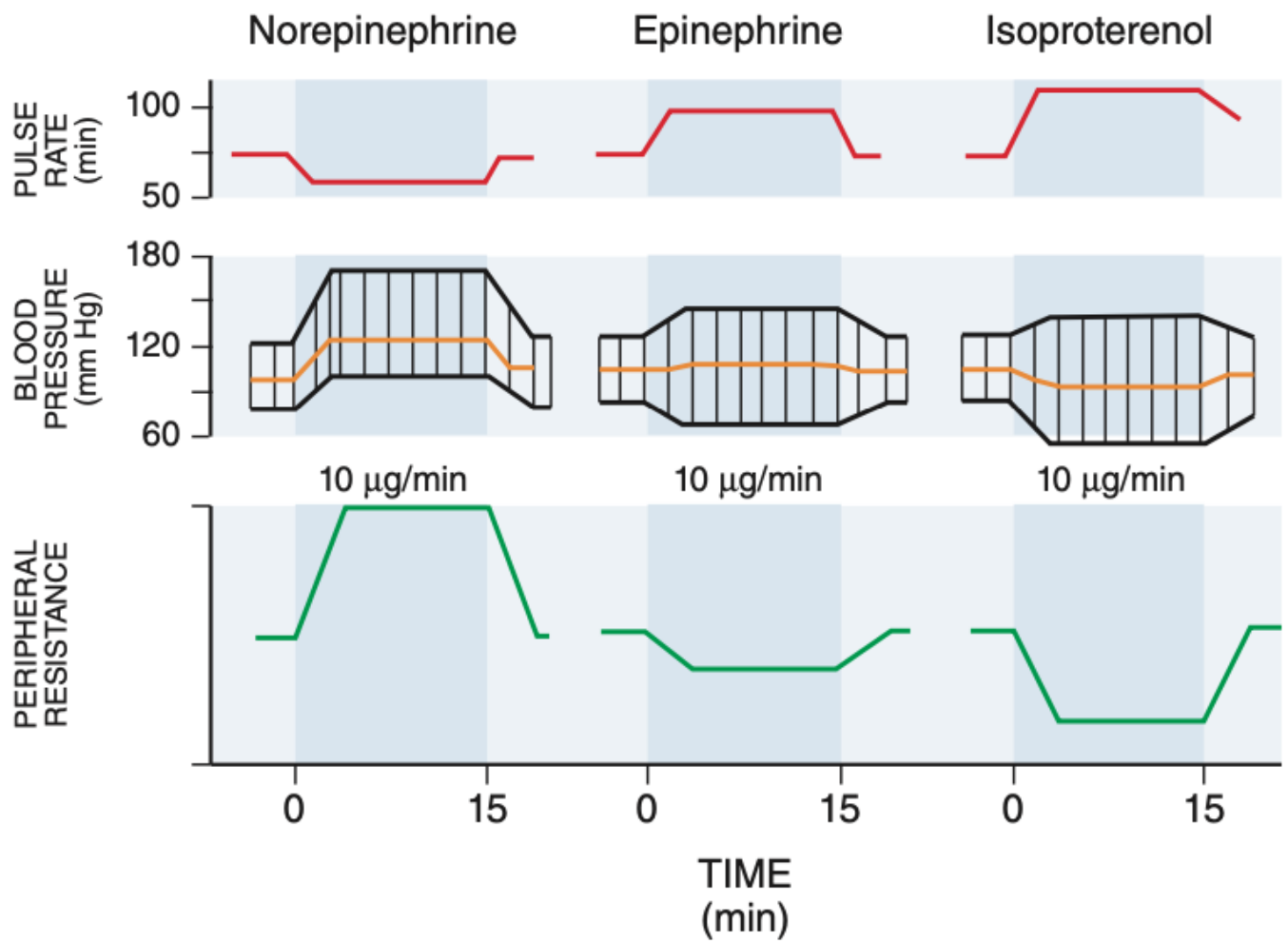


Figure 12–2 *Comparative effects of intravenous infusion of NE, EPI, and INE.* (Reproduced with permission from Allwood MJ, Cobbold AF, Ginsberg J. Peripheral vascular effects of noradrenaline, isopropylnoradrenaline, and dopamine. *Br Med Bull.* **1963**;*19*:132–136. With permission from Oxford University Press.)

Hilal-Dandan R, Knollmann B, Brunton L. Goodman and Gilman's The Pharmacological Basis of Therapeutics, 13th Edition. McGraw-Hill Education; 2017.

Dopamina

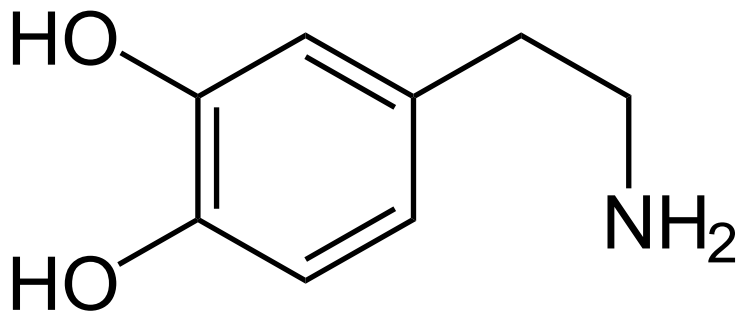
1–3 µg/kg/min → stimolo D1-like receptor → vasodilatazione coronarica, mesenterica, renale

3–10 µg/kg/min → stimolo β1 → inotropismo +, ↑HR, ↑rilascio e ↓reuptake NAD

>10 µg/kg/min → stimolo α1 → vasocostrizione periferica e renale

Diluizione

200 mg/50 ml



Hemmings HC, Egan TD. Pharmacology and Physiology for Anesthesia E-Book: Foundations and Clinical Application. Elsevier Health Sciences; 2018.

Efedrina

Isolato da Ephedra sinica

Agonista misto, non-catecolaminico
Compete con NAD per reuptake nelle vescicole sinaptiche

- ↑ HR, BP, CO 10-15 per 10-15 minuti
- Dosi ripetute: tachifilassi per deplezione di NAD
- Broncodilatatore
- Stimolante SNC

Generalmente disponibile in fiale da 10 o 25 mg
Diluizione a 5 o 2.5 mg/ml in NaCl 0.9%

Ephedrine hydrochloride

04-Dec-2019

INDICATIONS AND DOSE

Reversal of hypotension from spinal or epidural anaesthesia

▶ BY SLOW INTRAVENOUS INJECTION

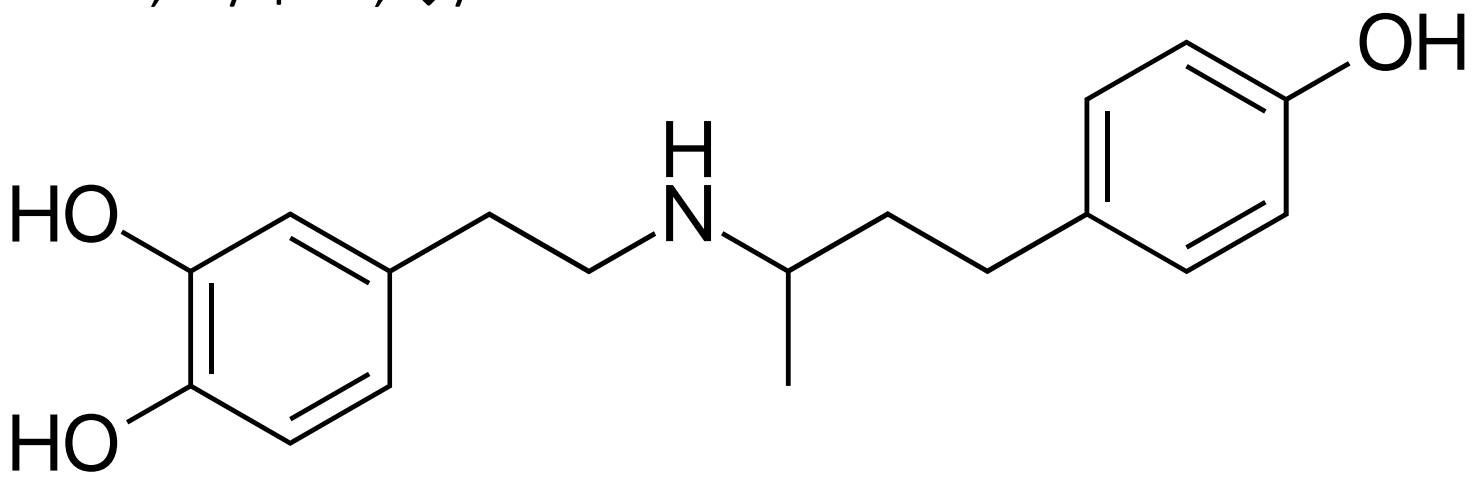
▶ Adult: 3–6 mg every 3–4 minutes (max. per dose 9 mg), adjusted according to response, injection solution to contain ephedrine hydrochloride 3 mg/ml; maximum 30 mg per course

Hemmings HC, Egan TD. Pharmacology and Physiology for Anesthesia E-Book: Foundations and Clinical Application. Elsevier Health Sciences; 2018.
BNF 80: September 2020-March 2021. (2020). Regno Unito: Pharmaceutical Press.

Dobutamina

Catecolamina sintetica (gruppo aromatico su dopamina)

Miscela racemica (+) isomero potente agonista β1/ antagonista α1, (−) isomero agonista α1
Ino-dilatatore
2–20 µg/kg/min →↑CO, ↓LV filling pressure, ↗/↑HR, ↓/= SVR



Inattivata da soluzioni alcaline

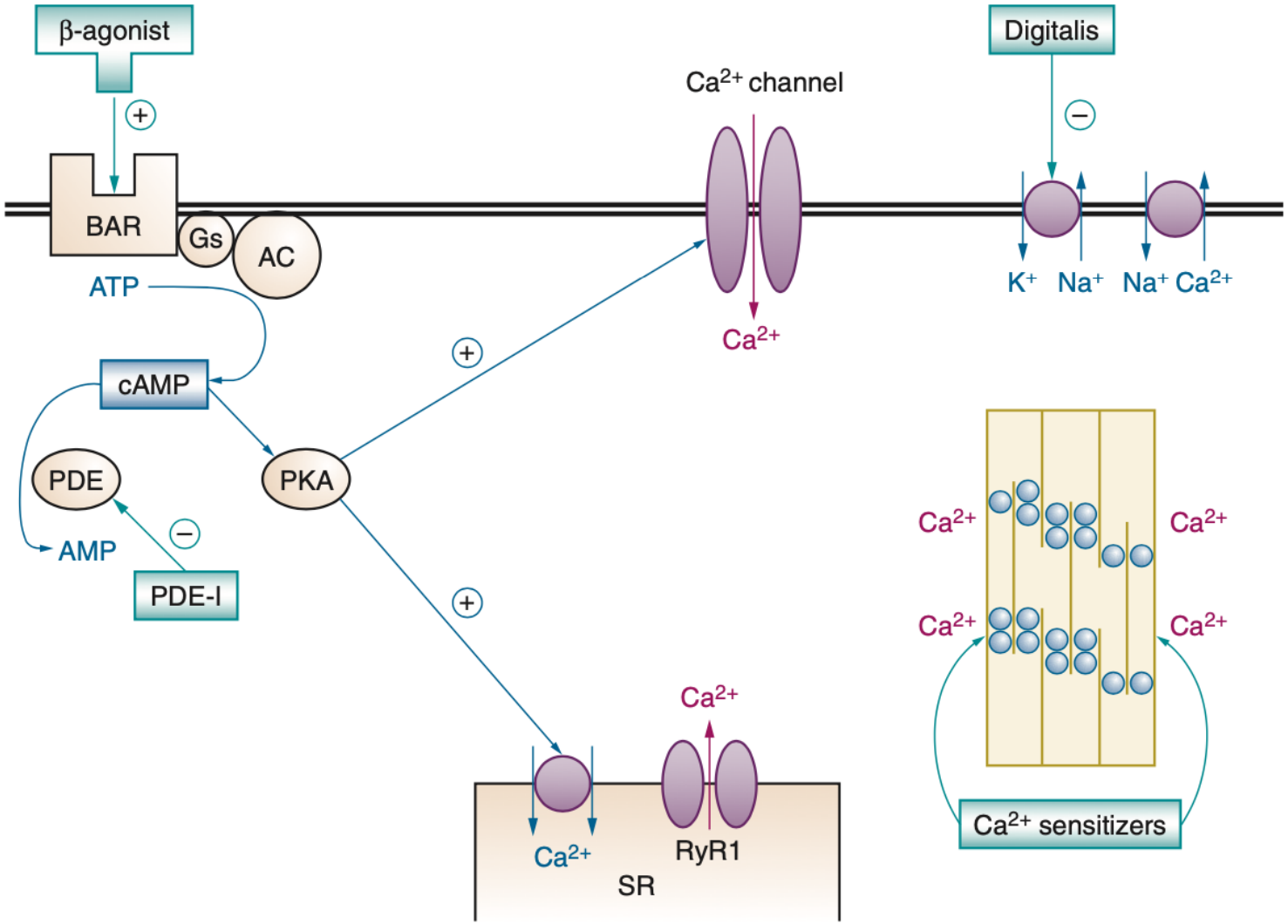
Diluizione in glucosio 5%
250 mg / 50ml

Tachifilassi per infusioni prolungate >72h

Hemmings HC, Egan TD. Pharmacology and Physiology for Anesthesia E-Book: Foundations and Clinical Application. Elsevier Health Sciences; 2018.

Domande o interventi?

Milrinone/Enoximone



Hemmings HC, Egan TD. Pharmacology and Physiology for Anesthesia E-Book: Foundations and Clinical Application. Elsevier Health Sciences; 2018.

Milrinone/Enoximone

Milrinone

27-Jan-2020

- **DRUG ACTION** Milrinone is a phosphodiesterase type-3 inhibitor that exerts most effect on the myocardium; it has positive inotropic properties and vasodilator activity.

- **INDICATIONS AND DOSE**

Short-term treatment of severe congestive heart failure unresponsive to conventional maintenance therapy (not immediately after myocardial infarction) | Acute heart failure, including low output states following heart surgery

▶ INITIALLY BY INTRAVENOUS INJECTION

▸ Adult: Initially 50 micrograms/kg, given over 10 minutes, followed by (by intravenous infusion) 375–750 nanograms/kg/minute usually given following surgery for up to 12 hours or in congestive heart failure for 48-72 hours; maximum 1.13 mg/kg per day

Enoximone

10-Mar-2020

- **DRUG ACTION** Enoximone is a phosphodiesterase type-3 inhibitor that exerts most effect on the myocardium; it has positive inotropic properties and vasodilator activity.

- **INDICATIONS AND DOSE**

Congestive heart failure where cardiac output reduced and filling pressures increased

▶ BY SLOW INTRAVENOUS INJECTION

▸ Adult: Initially 0.5–1 mg/kg, rate not exceeding 12.5 mg/minute, then 500 micrograms/kg every 30 minutes until satisfactory response or total of 3 mg/kg given; maintenance, initial dose of up to 3 mg/kg may be repeated every 3–6 hours as required

▶ BY INTRAVENOUS INFUSION

▸ Adult: Initially 90 micrograms/kg/minute, dose to be given over 10–30 minutes, followed by 5–20 micrograms/kg/minute, dose to be given as either a continuous or intermittent infusion; maximum 24 mg/kg per day

BNF 80: September 2020-March 2021. (2020). Regno Unito: Pharmaceutical Press.

Milrinone/Enoximone

- Before staring a PDEI, hypovolaemia must be corrected to prevent a further significant fall in blood pressure. Preloading the circulation may be necessary, although volume loading may be given at the same time as starting treatment with a PDEI. Volume loading should be undertaken with care in patients recently weaned from cardiopulmonary bypass since left ventricular compliance is usually abnormal, and rapid transfusion resulting in excessive left ventricular volume may cause a precipitate rise in left ventricular pressure, subendocardial compression, and myocardial ischaemia.
- Vasopressors may be needed and should be prepared for use at the same time as PDEI administration. A norepinephrine infusion is commonly used; vasopressin has been described as an alternative.[8] For those patients already on a vasopressor, an increase in dosage requirements should be expected.

Feneck R. Phosphodiesterase inhibitors and the cardiovascular system. Continuing Education in Anaesthesia Critical Care & Pain. 2007;7(6):203-207. doi:10.1093/bjaceaccp/mkm039

Levosimendan

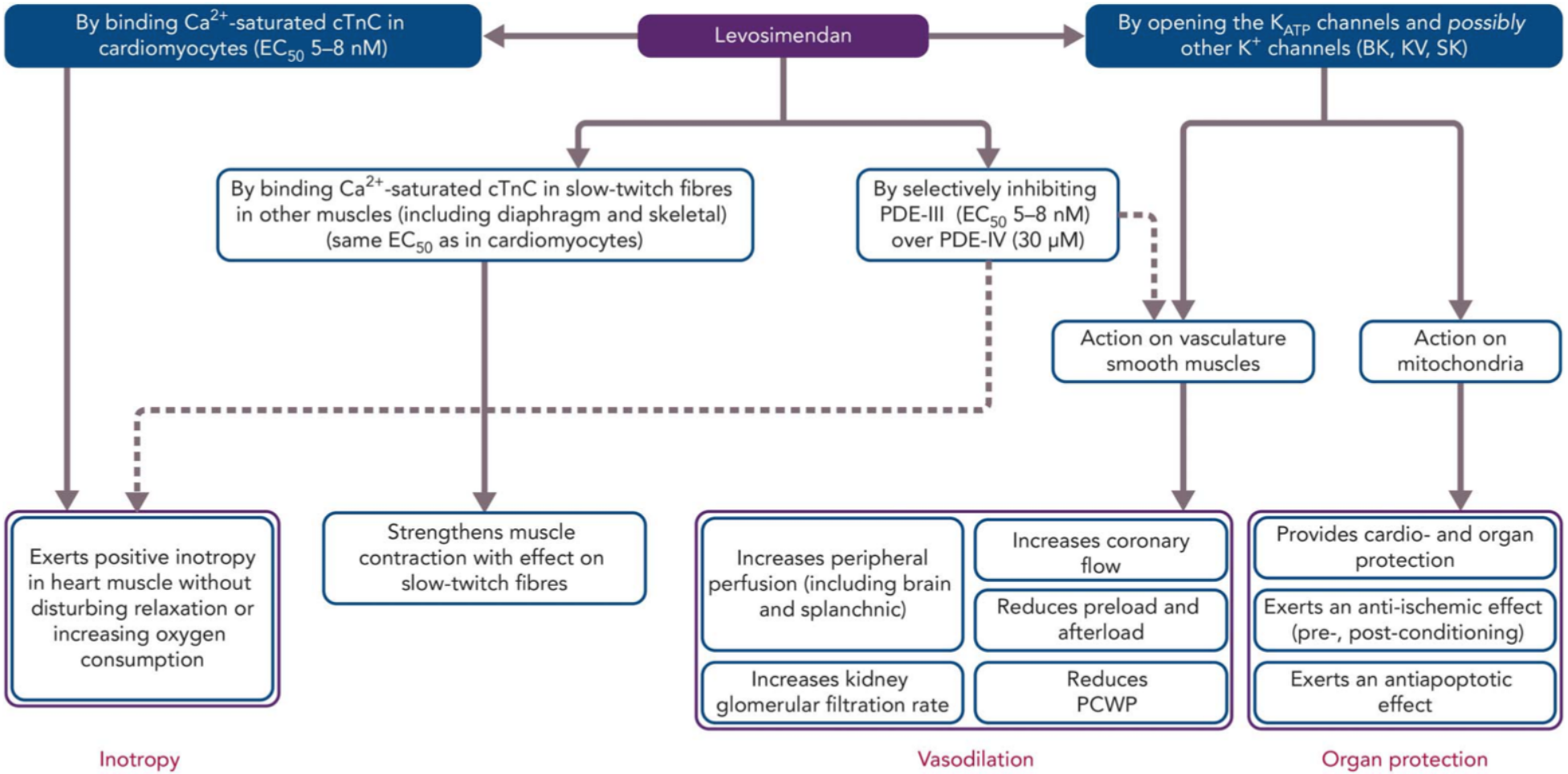
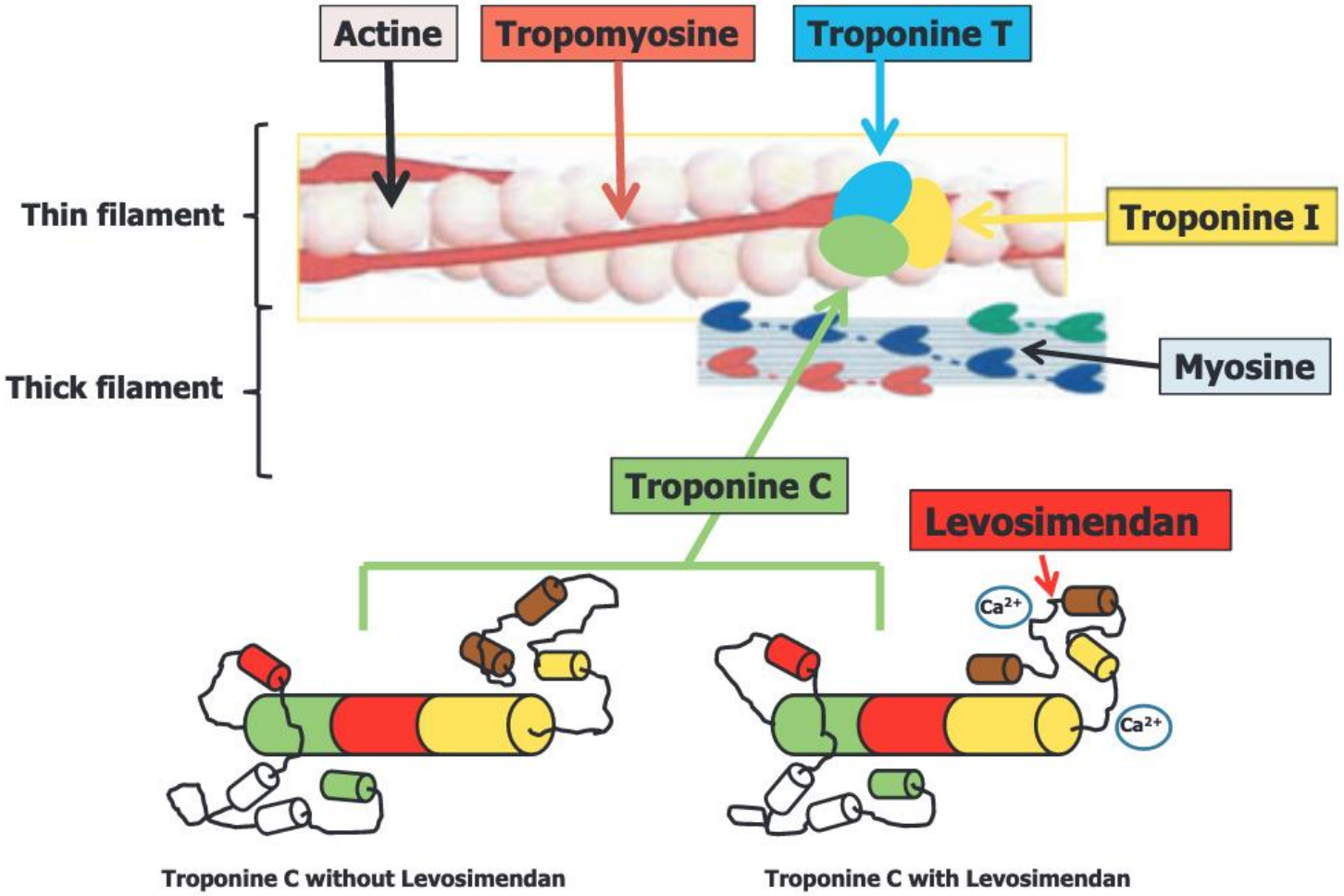


FIGURE 2. Mode of actions and pharmacologic effects of levosimendan: The mechanisms of action in the blue boxes contribute to the cardiovascular effects of the drug. Dotted lines mark pathways that are still not fully elucidated. EC_{50}, half maximal effective concentration; K_{ATP}, adenosine triphosphate–dependent potassium channels; PDE III, IV, phosphodiesterase isoforms in cardiac tissue. Adapted from: Al-Chalabi et al[216] Used with permission from Wolters Kluwer Health.

Papp Z, Agostoni P, Alvarez J, et al. Levosimendan Efficacy and Safety: 20 Years of SIMDAX in Clinical Use. Journal of Cardiovascular Pharmacology. 2020;76(1).

Levosimendan



Pathak A, Lebrin M, Vaccaro A, Senard JM, Despas F. Pharmacology of levosimendan: inotropic, vasodilatory and cardioprotective effects. J Clin Pharm Ther. 2013;38(5):341-349. doi:10.1111/jcpt.12067

Levosimendan

Dosaggio 0.05 – 0.2 µg/kg/min

sensibilizza cardiomiociti al Ca^{2+}

stabilizza la troponina C favorendo la contrazione

effetto lusitropo

vasodilatazione tramite canali ATP-sensitive K+

↓precarico, ↓postcarico, ↑DO_2/MVO_2 miocardio, ↑perfusione coronarica e renale

↑CO, ↑SV, ↑HR, ↓SVR, ↓SBP, ↓PCWP, ↓PAP

debole inibitore cAMP-PDE

Durata 7-9 giorni per metaboliti attivi

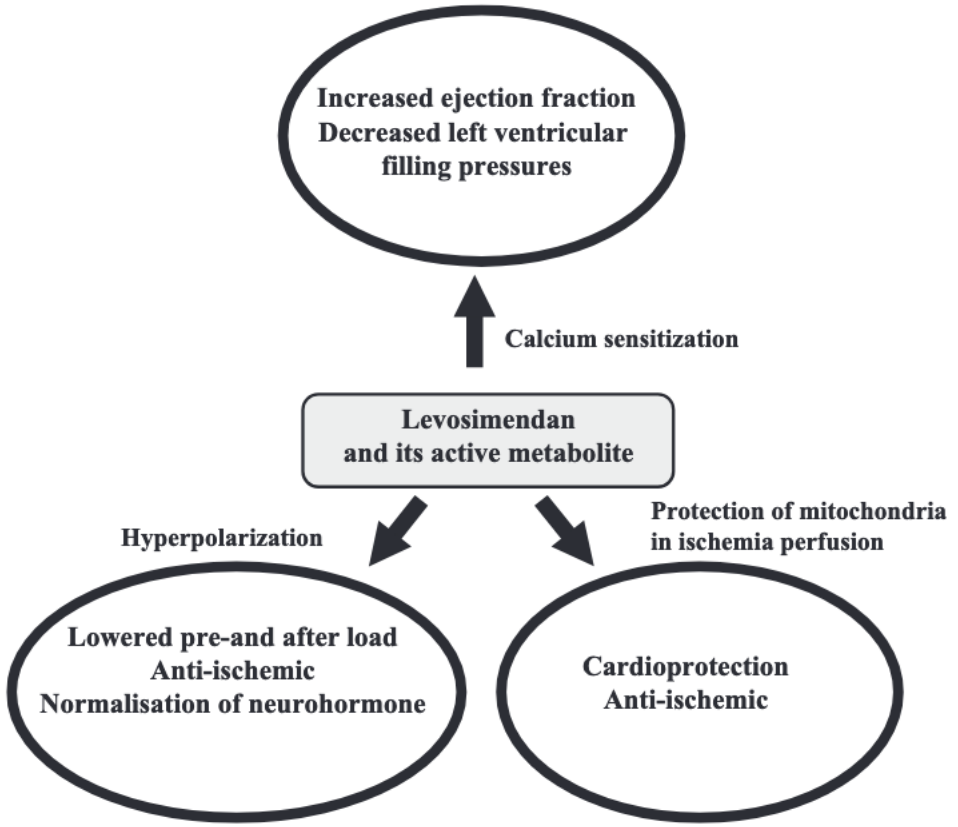
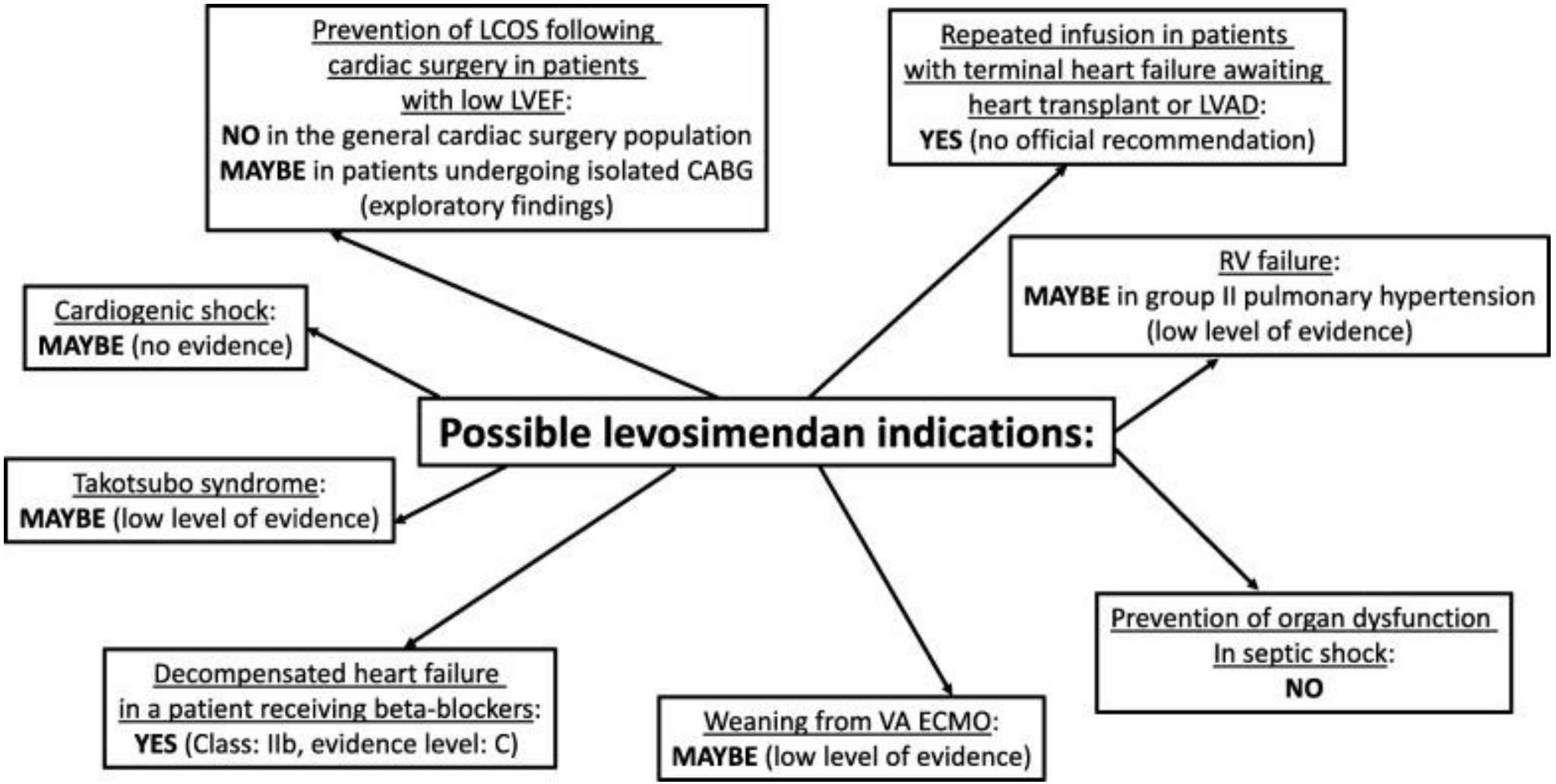


Fig. 2. Major biological mechanisms and haemodynamic effects of levosimendan.

Pathak A, Lebrin M, Vaccaro A, Senard JM, Despas F. Pharmacology of levosimendan: inotropic, vasodilatory and cardioprotective effects. J Clin Pharm Ther. 2013;38(5):341-349. doi:10.1111/jcpt.12067

Levosimendan



Cholley B, Levy B, Fellahi JL, et al. Levosimendan in the light of the results of the recent randomized controlled trials: an expert opinion paper. Crit Care. 2019;23(1):385. doi:10.1186/s13054-019-2674-4

Terlipressina

Dosaggio 0.05-0.2 mg/h

Diluizione 1 mg/50 ml

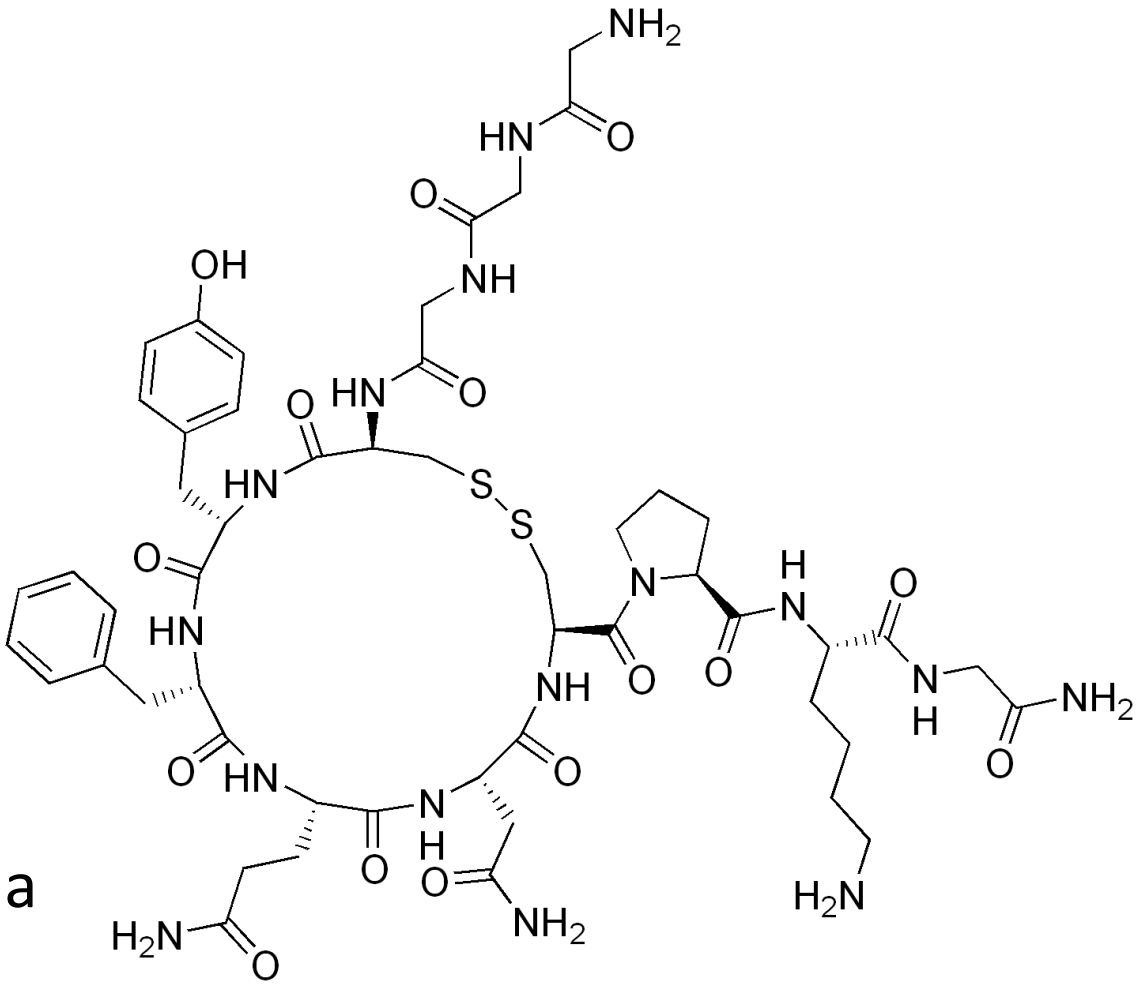
Agonista recettori V1

Potente vasocostrittore periferico

Vasodilatatore polmonare

t ½ 50 min, conversione a lisin-vasopressina

durata effetto vasoattivo 8 h



Hemmings HC, Egan TD. Pharmacology and Physiology for Anesthesia E-Book: Foundations and Clinical Application. Elsevier Health Sciences; 2018.

SIAARTI
PRO VITA CONTRA DOLOREM SEMPER

BUONE PRATICHE CLINICHE

USO DELLA VASOPRESSINA E DEI SUOI ANALOGHI NEI PAZIENTI CRITICI

Coordinatori del gruppo di lavoro:
Massimo Girardis
Dipartimento di Anestesia, Rianimazione e Terapia Intensiva,
Azienda Ospedaliero-Universitaria Policlinico di Modena
Luigi Tritapepe
UOC Anestesia e Rianimazione,
Azienda Ospedaliera San Camillo-Forlanini, Roma;
Università Sapienza di Roma

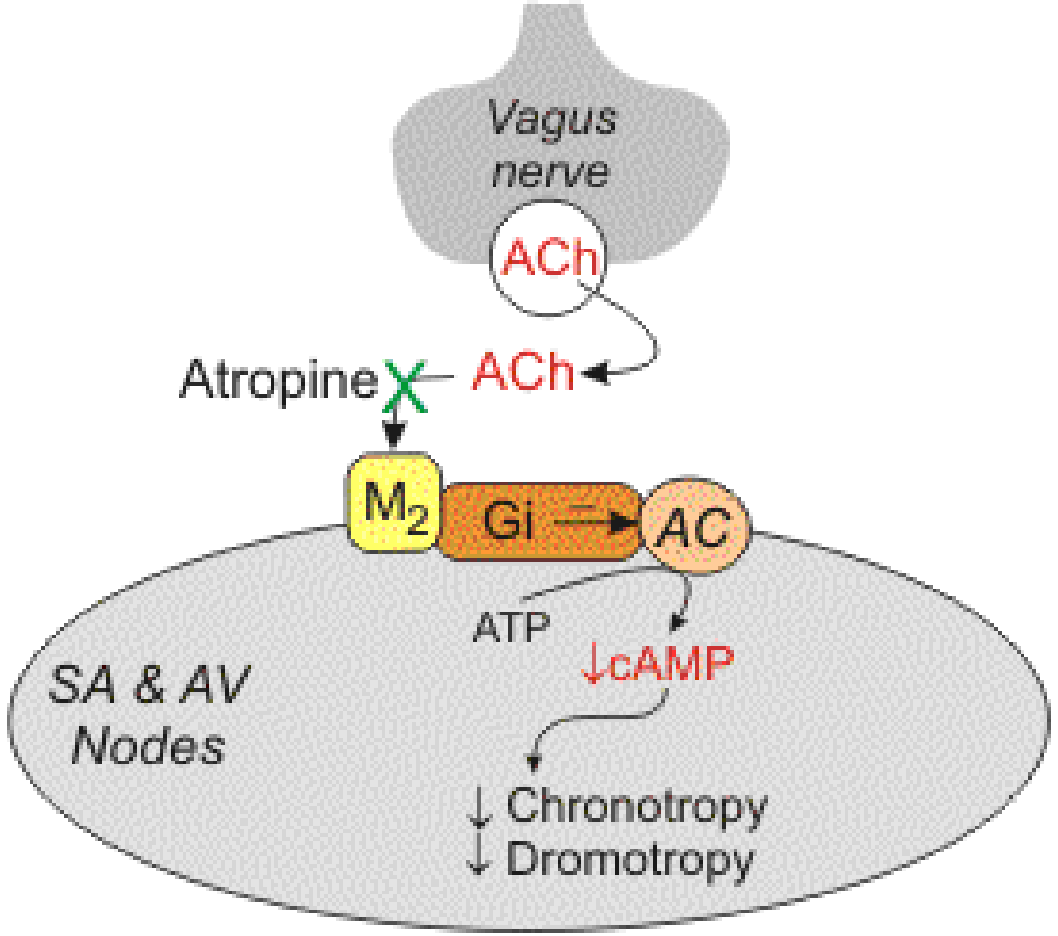
- **Nei pazienti con shock settico non è appropriato l'utilizzo di vasopressina come vasopressore di prima linea**
- **Nei pazienti con shock settico refrattario, che ricevono già noradrenalina, è appropriata l'associazione di vasopressina/terlipressina come vasopressore di seconda linea per raggiungere una pressione arteriosa media di 65 mmHg**
- **Nei pazienti con shock settico che ricevono noradrenalina con indicazione ad associare vasopressina/terlipressina, l'infusione continua di vasopressina è da preferire rispetto all'utilizzo della Terlipressina**
- **Nei pazienti con shock settico meno severo (dosaggi di noradrenalina da 5 a 14 mcg/min), è appropriata la precoce associazione di vasopressina alla noradrenalina per ridurre la mortalità**
- **Nei pazienti con shock settico trattati con noradrenalina e steroidi, l'appropriatezza di associare la vasopressina per trarre vantaggio dall'attività sinergica non è chiara**
- **Nei pazienti con shock settico, è appropriata l'associazione della vasopressina alla noradrenalina per ridurre l'incidenza di danno renale acuto**
- **Nei pazienti con shock settico l'appropriatezza dell'utilizzo precoce di vasopressina per ridurre l'incidenza di fibrillazione atriale non è chiara.**
- **Nei pazienti con shock settico e cirrosi epatica è appropriato l'utilizzo precoce di vasopressina/terlipressina per ridurre le complicanze e la mortalità**

Domande o interventi?

Atropina

Premedicazione
0.4-0.6 mg IV/IM/SC 30-60 prima dell'anestesia
q4-6hr PRN

Bradicardia
0.5-1 - 0.04 mg/kg IV q5min, dose massima 3 mg



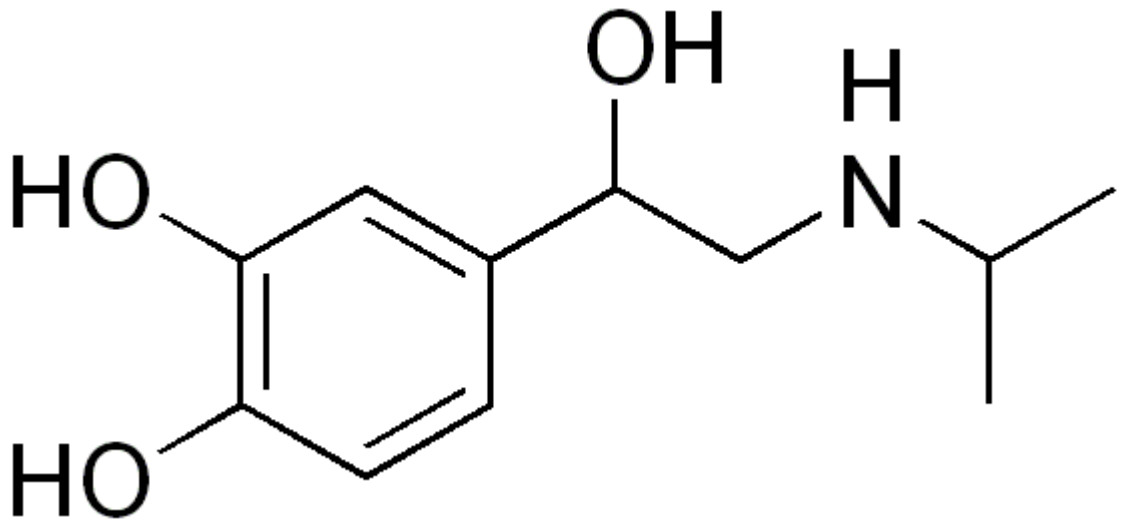
Abbreviations: ACh, acetylcholine; M2, muscarinic receptor; AC, adenylate cyclase; SA, sinoatrial; AV, atrioventricular

https://reference.medscape.com/drug/atropen-atropine-iv-im-343093
https://cvpharmacology.com/antiarrhy/atropine

Isoproterenolo

Agonista **beta-1 and beta-2 adrenergic**

- ↑ HR
- ↑ contrattilità
- Rilassamento muscolatura bronchiale, intestinale. uterina
- Vasodilatazione periferica



Bradicardia

0.05 - 1 µg/kg/min

https://www.ncbi.nlm.nih.gov/books/NBK526042/

Domande o interventi?