

Chapter 13

Vasodilating Agents

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ABSTRACT

This chapter discusses the salient features of arterial and venous dilating agents commonplace in the management of the post-cardiotomy surgical patient. A keen understanding of the underlying cellular mechanism, pharmacology, indication, safety profile, and controversies of clinical utility of vasodilating agents is imperative for routine use. The evidenced-based examination of each therapeutic modality will strengthen the practitioner's fund of knowledge for management of each pathophysiological state.

INTRODUCTION

Vasodilating agents have a wide scope of therapeutic utility both intraoperatively and in the ICU setting. The rapid onset of vasodilators aide in treatment of aneurysm propagation, aneurysm dissection, and hypertensive crises. Pulmonary vasodilators are the crux of right-heart support with provision of inotropy, decreasing right ventricular preload, and decreasing pulmonary vascular resistance.

Intraoperatively, vasodilator-mediated hypotensive anesthetic technique preserves chief quality outcome measures. Bodies of evidence demonstrate 2-4 fold reduction of intraoperative blood loss(Sharrock, 1998), 50% reduction of in-hospital transfusion, decreased intraoperative time (Dolman, Bentley & Head, 2000).

Vasodilator drugs are classified based upon their site of activity. Dilation of the capacitance – venous – system will reduce preload, allowing an unloading of the myocardium. Decreased wall tension reduces myocardial oxygen demand, and optimizes overall Starling function. Dilation of the resistance – arteriole – system results in afterload reduction, improving effective myocardial contractility. The net result is augmentation of stroke volume and cardiac output.

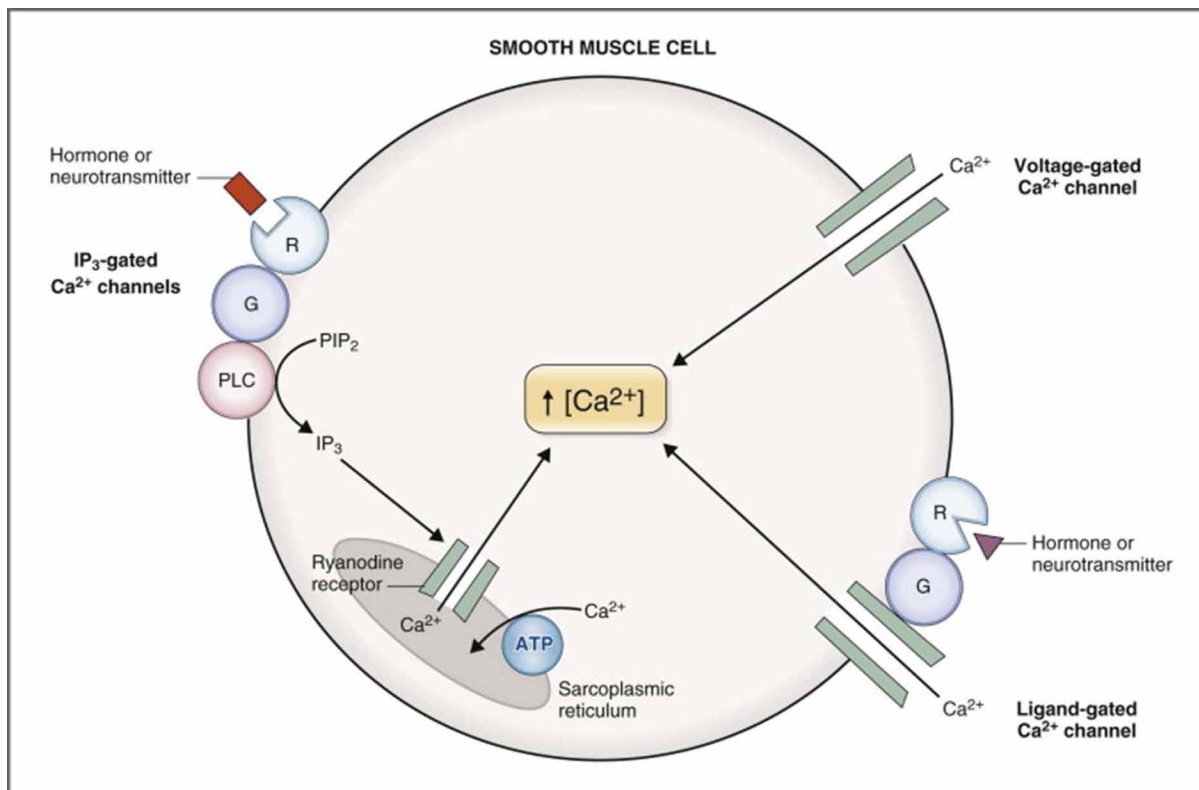
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MOLECULAR MECHANISMS: SMOOTH MUSCLE

Vascular smooth muscle constriction is dependent upon the translation of the synaptic action potential to muscle tension. Excitation-coupling describes the rise of intracellular calcium following receptor interaction with a neurotransmitter. Intracellular calcium rises trifold (see figure 1): (i) direct influx through voltage-gated calcium channels, (ii) g-protein ligand gated calcium channels, and (iii) IP₃-phospholipase system. The latter, GTP binding protein – G Protein – is activated, and couples with phospholipase C to upregulate IP₃ (inositol 1,4,5-triphosphate) production (Costanzo, 2014). The net effect is increased intracellular Calcium, liberated from the sarcoplasmic reticulum. Intracellular calcium-calmodulin complexes activate myosin cross-bridges, resulting in vasoconstriction. Increased systemic vascular resistance leads to a higher afterload and mean arterial pressure, and a parasympathetic bradycardic reflex. Alpha-receptor antagonism (*e.g.* phentolamine) perturbs intracellular calcium release, lowering overall systemic vascular resistance in a vasodilated state.

Figure 1. Trifold mechanism that increases intracellular smooth muscle calcium

This figure was published in Physiology, Fifth Edition. Costanzo, Linda. Figure 1-30 Mechanisms for increasing intracellular [Ca²⁺] in smooth muscle. Copyright Elsevier 2014.



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