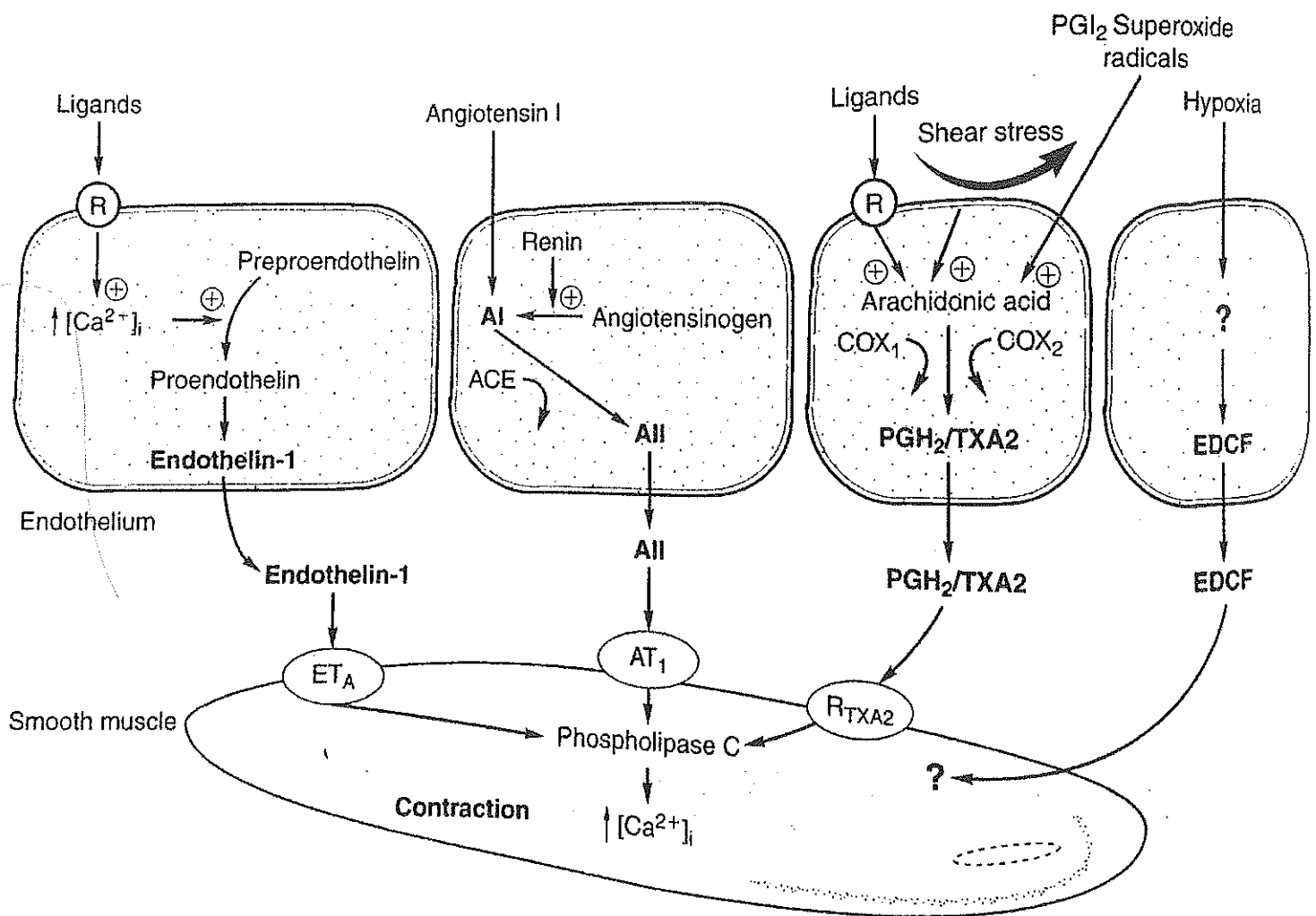


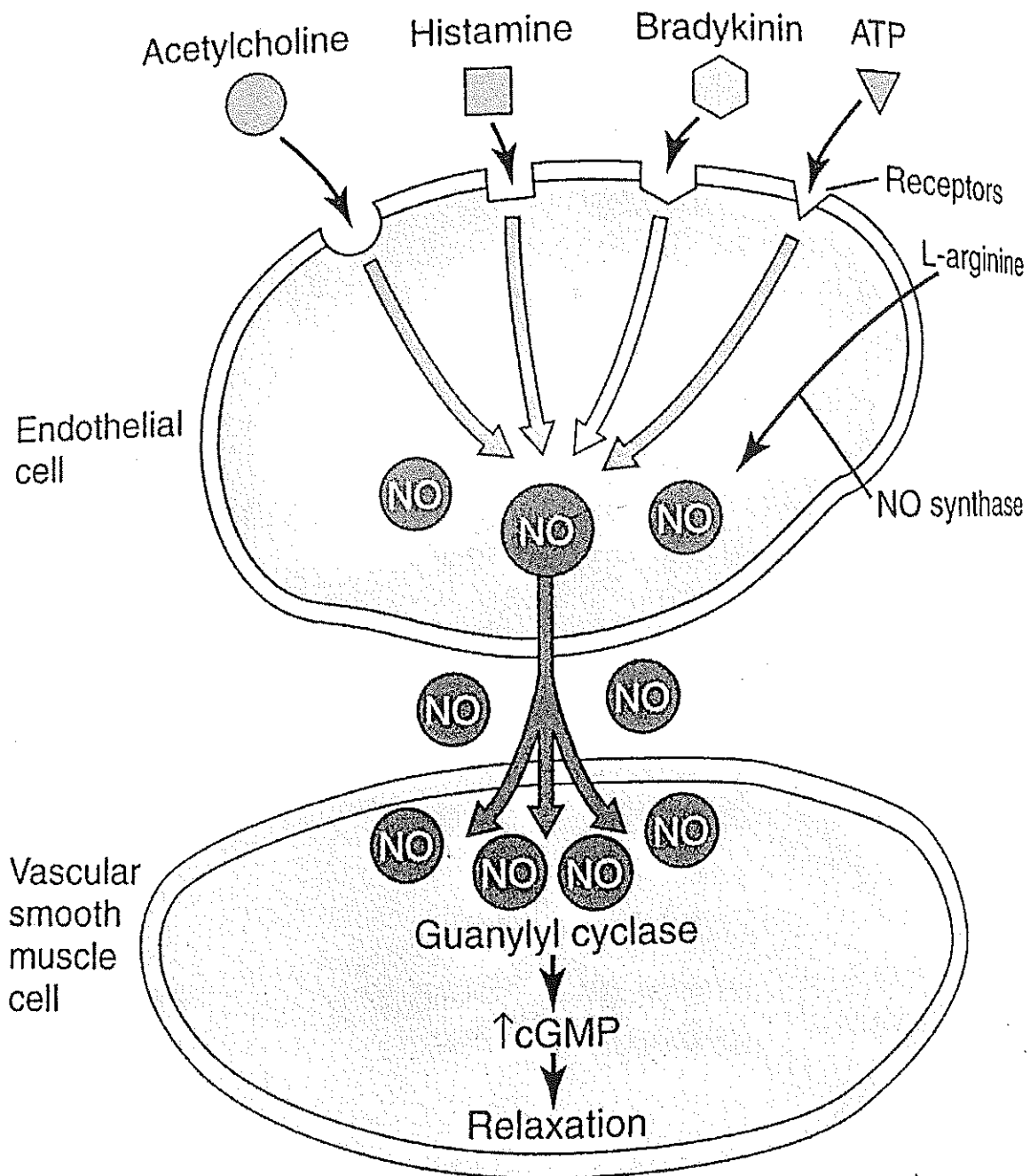
FIGURE 31-7

Contracting factors derived from vascular endothelium. *Endothelin-1* is generated from the precursor preproendothelin. It is a powerful vasoconstrictor that is preferentially released toward the luminal side of endothelial cells. *Angiotensin II (AII)* is produced in endothelial cells because they are the locus for angiotensin-converting enzyme (ACE). *Prostaglandin H<sub>2</sub>* and *thromboxane A<sub>2</sub> (TXA<sub>2</sub>)* both bind to the TXA<sub>2</sub> receptor (R<sub>TXA2</sub>). They are produced when cyclooxygenase is activated by mechanical or various chemical stimuli. Preference for production of one or the other is determined by local concentrations of promoters and inhibitors. PGH<sub>2</sub> has an extremely short half-life. *Endothelium-derived contracting factor (EDCF)* is responsible for hypoxic vasoconstriction. Its chemical nature has not yet been identified. AI = angiotensin I; AII = angiotensin II; ACE = angiotensin-converting enzyme; AT<sub>1</sub> = type 1 angiotensin II receptor; COX = cyclooxygenase; ET<sub>A</sub> = A-type endothelin receptor; PGI<sub>2</sub> = prostaglandin I<sub>2</sub> (prostacyclin); PGH<sub>2</sub> = prostaglandin H<sub>2</sub>; TXA<sub>2</sub> = thromboxane A<sub>2</sub>.



## Phosphodiesterase Type 5 Inhibitors

Sildenafil, tadalafil, and vardenafil are selective inhibitors of cGMP-specific PDE type 5, which is found in high concentrations in the penile corpus cavernosum and is



**FIGURE 24-3** Endothelium-dependent relaxation produced by vasodilators. These substances act on endothelial cells at their respective receptors to release NO. The latter diffuses into the vascular smooth muscle cell, increases soluble guanylyl cyclase activity and cGMP concentration, and promotes relaxation. L-Arginine is converted to NO by NO synthases.

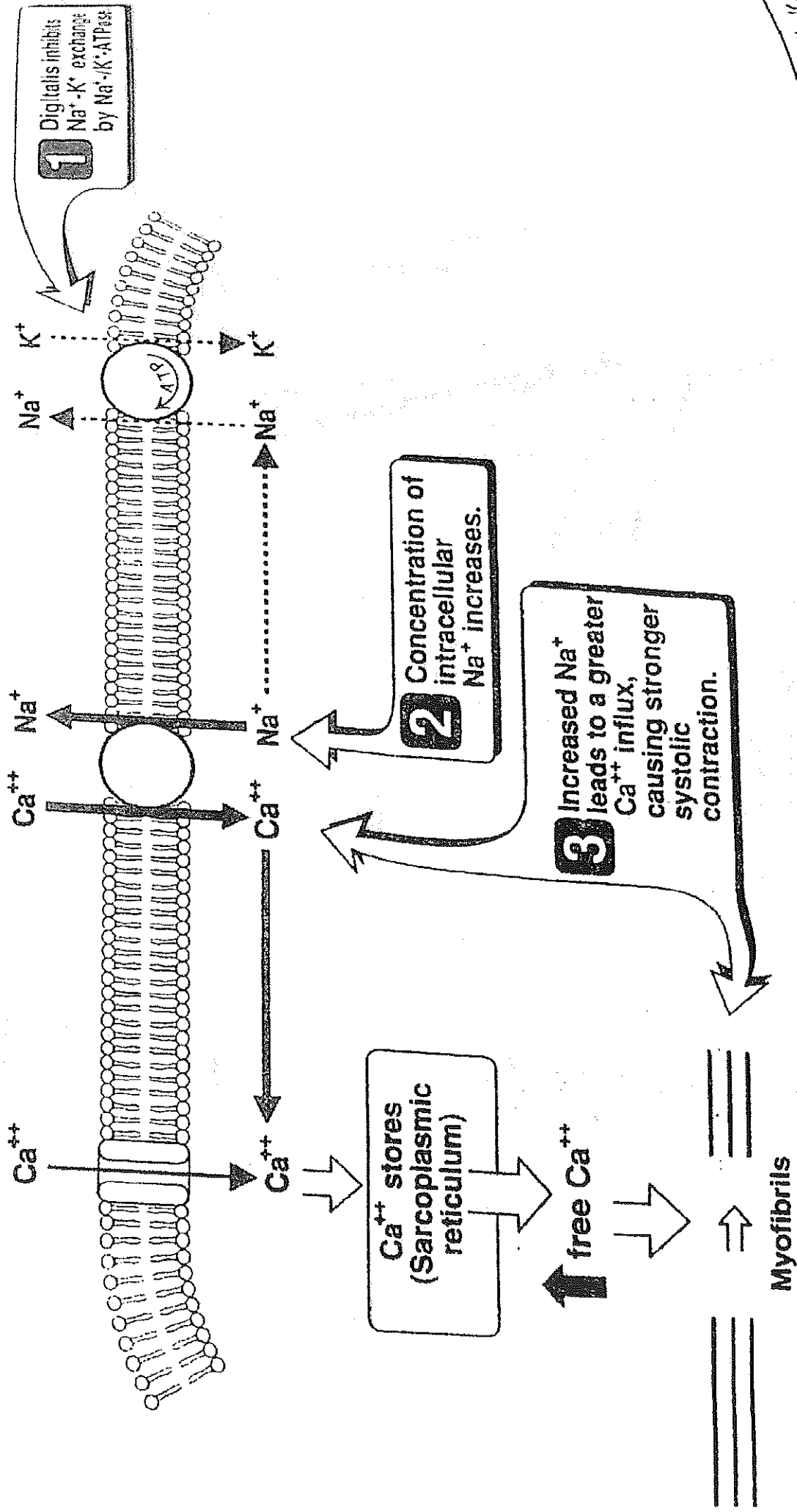
TABLE 33-1

## Major Ion Fluxes during the Cardiac Action Potential

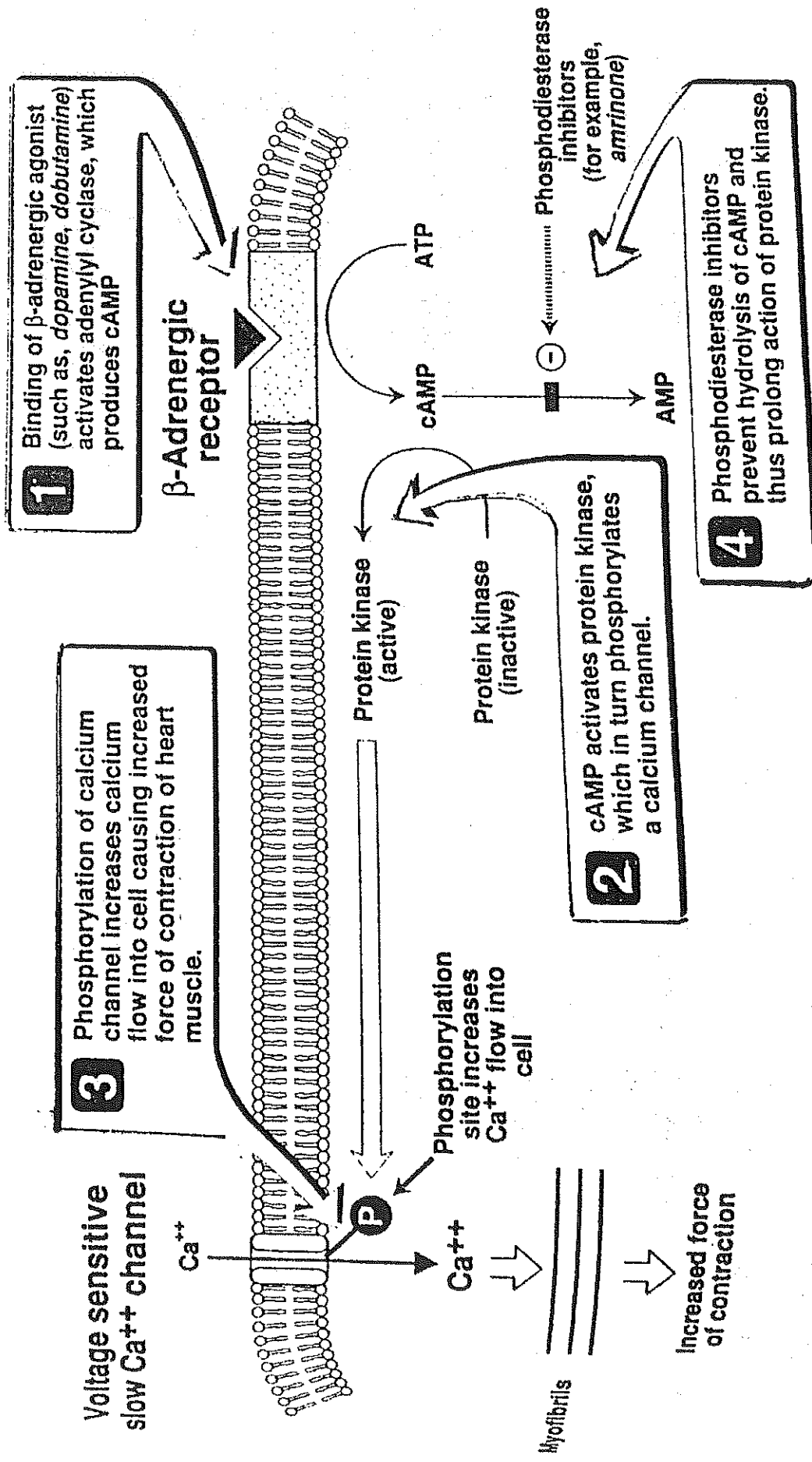
Name	Ion	Current	Phase of Action Potential	
$I_{Na}$	$Na^+$	Inward	0	(depolarization)
$I_{to}$	$K^+$	Outward	1	(rapid repolarization)
$I_{Ca}$	$Ca^{2+}$	Inward	2	(plateau)
$I_{Kr}$	$K^+$	Outward	2, 3	(repolarization)
$I_{Ks}$	$K^+$	Outward	2, 3	(repolarization)
$I_{Kur}^*$	$K^+$	Outward	2	(repolarization)
$I_{K1}^\dagger$	$K^+$	Outward	3, 4	(repolarization, diastole)
$I_f$	$Na^+$	Inward	4	(spontaneous depolarization)

\* $I_{Kur}$  is only identified in atrial tissue.

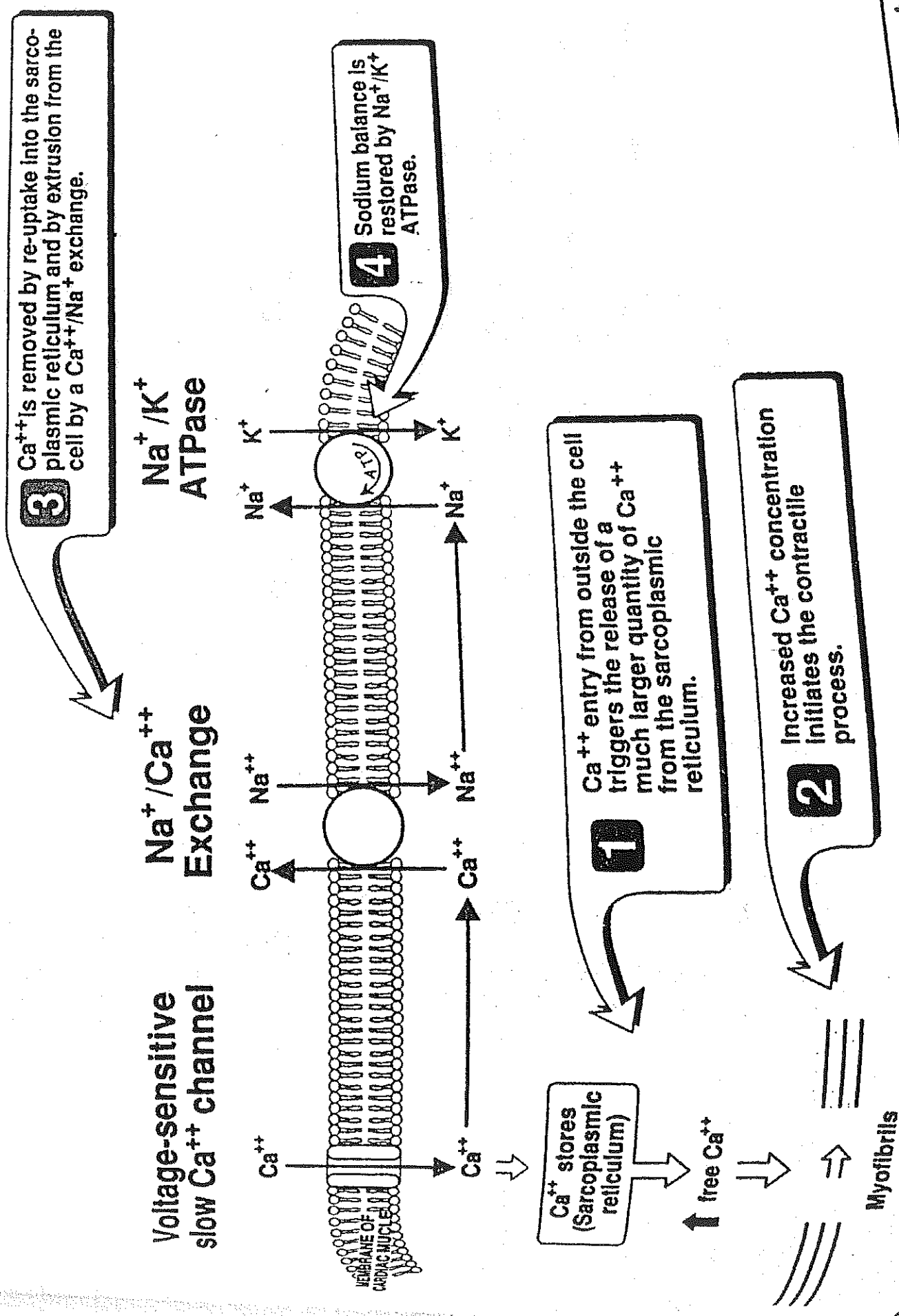
$I_{K1}^\dagger$  during the resting phase (diastole in cells without spontaneous depolarization) maintains the equilibrium responsible for the resting potential at or near the Nernst potential.



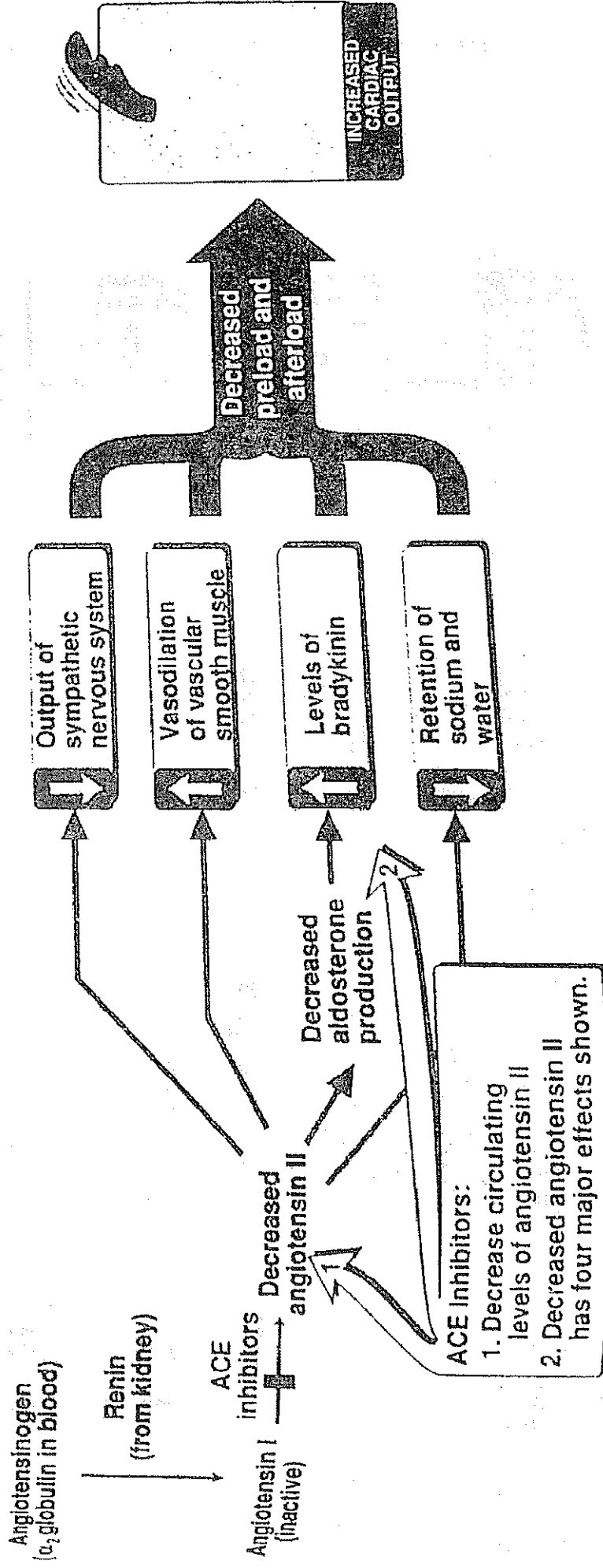
الشكل (7.16) : آلية تأثير الغلو كوزيدات القلبية (الديجيتالية)



الشكل (11-16) : أماكن تأثيرات مقلدات  $\beta$  على العضلة القلبية



الشكل (3-16) : حركة الشوارد خلال تقلص العضلة القلبية



الشكل (5.16) : تأثيرات مضادات الخثرة القابلة للإنجيوتنسين

أ - م - د - ه - ز - ح - ط - ث - ج - ب - أ