POST HEART TRANSPLANT DENERVATION

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- Direct sympathetic and parasympathetic innervation is absent in the post transplant donor heart. The donor heart also evidences an altered response to the baroreceptors.
- Normally, tonic vagal afferent outflow from the atria and ventricles is reduced in response to decreased filling volumes, and central sympathetic outflow increases. Hypotensive activation of carotid and aortic baroreceptors also increases central sympathetic outflow. However, there is no direct sympathetic contact to the denervated transplanted heart.
- In the transplanted heart, cardiac output eventually increases when systemic catecholamine levels increase, but again, there is no direct central sympathetic contact. Compensation for abrupt decreases in blood pressure is slow.
- The baroreceptor response to exogenous catecholamines is also absent, i.e. there is no reflex bradycardia in response to phenylephrine administration.
- Denervation results in presynaptic supersensitivity to catecholamines which evidence reuptake at the adrenergic nerve terminal, such as epinephrine and norepinephrine, but not isoproterenol which has no such reuptake.
- It seems reinnervation of the left ventricle may occur by efferent sympathetics after a year or more post transplant, but vagal reinnervation is unlikely. Some transplant patients evidence angina, supposedly due to sympathetic reinnervation.
- Heart rate in the transplanted heart averages 90 to 110, demonstrating the absence of parasympathetic tone. Heart rate increases in response to systemic catecholamines, but the response is attenuated as compared to normal hearts due a lack of SA node innervation.