



# Blood circulation and heart

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## DESCRIPTION

Clinical manifestations hinge upon the presence or absence of pulmonary outflow obstruction. Without stenosis, infants with low pulmonary resistance present with signs and symptoms typical of huge left-to-right shunting, ie, congestive heart disease and failure-to-thrive. Due to increased pulmonary blood flow, cyanosis might not be apparent. Aortic outflow obstruction may compound already excessive pulmonary blood flow and worsen congestive cardiopathy. Some patients (eg, type IV DILV) may have a preferential favorable stream of systemic venous return to the arterial blood vessel and pulmonary venous return to the aorta. In contrast, an unfavorable stream with a transposition-like blood flow pattern may occur in patients with a right-sided subaortic heart ventricle and straddling right AV valve. Cyanosis and systemic hypoxemia may result.

In the setting of a univentricular heart, a particular degree of stenosis is physiologically desirable to stop pulmonary overcirculation. Severe stricture or atresia may end in profound hypoxemia and cyanosis, however.

### Cardiac catheterization

Cardiac catheterization may provide a close assessment of anatomic and functional features. Objectives include assessment of hemodynamics, systemic and pulmonary venous anatomy, AV and ventricular-arterial connections, ventricular morphology and performance, pulmonary vascular resistance, aorta integrity, and systemic-pulmonary collaterals. Patients with univentricular hearts characteristically have an entire mixture of systemic and pulmonary venous circulations at the ventricular level. If one assumes a pulmonary venous oxygen saturation of 96% and normal systemic blood flow, the arterial oxygen saturation reflects total pulmonary blood flow. As a rule of thumb, values  $\geq 85\%$  and  $<75\%$  signify increased and decreased pulmonary blood flow, respectively.

The presence or absence of hemodynamic and anatomic abnormalities like poor ventricular function, aortic coarctation, arterial blood vessel distortion, increased pulmonary vascular resistance, and abnormal collateral vessels are relevant to therapeutic management plans. Proponents of routine

preoperative cardiac catheterization assert that noninvasive imaging may fail to visualise arterial blood vessel distortion, that cardiac catheterization is that the only valid method to live pulmonary vascular resistance, which abnormal aortopulmonary collateral vessels could also be identified and coil embolization performed if necessary. After initial palliation, patients not fitted to Fontan completion may enjoy repeated catheterizations to reassess pulmonary pressures and magnitude of created shunts, and address complications like shunt stenosis, arteria pulmonalis stenosis, and pulmonary arteriovenous fistulae.

International guidelines for the management of valvular heart condition recommend lifelong anticoagulation with VKAs for all patients with MHVs: recommendations are class I, level of evidence A within the latest American Heart Association/American College of Cardiology guidelines; class I, level of evidence B within the European Society of Cardiology guidelines; and sophistication I, level of evidence B within the American College of Chest Physicians guidelines. VKAs block the carboxylation of 4 coagulation proteins, namely FII (prothrombin), FVII, FIX, and FX. The dosage required for optimal anticoagulation displays individual variations per the diet, drug interactions, disease processes, and gene polymorphisms affecting VKA metabolism. The carboxylation of vitamin K-dependent factors isn't blocked to the identical speed and to the identical extent at the start of therapy, and there's considerable interindividual variation in plasma levels of active, carboxylated factors, at any given INR. Furthermore, changes in dietary antihemorrhagic factor intake, poor compliance, drug interactions, or impaired absorption due to gastrointestinal disturbances all increase the intra- and interpatient variability in efficacy. For of these reasons, the INR is usually unstable, which could be a major determinant of thromboembolic or bleeding events, and reduced survival after MHV implantation, as well. Finally, even when the INR remains within the normal range, thromboembolic complications should occur thanks to the numerous different pathogenetic mechanisms involved in thromboembolism after valve surgery.