Cardiovascular responses to shock

Cardiovascular effects of IPPV and PEEP

Shock is defined as inadequate tissue perfusion resulting in tissue damage. It is usually further classified according to the physiological cause into four categories. Hypovolemic shock is due to a decrease in the circulating blood volume, most commonly due to haemorrhage, but gastrointestinal or non-sensible losses may also cause this state. Low resistance shock is a result of the a dramatic decrease in total peripheral resistance, this may be seen in cord injuries, anaphylaxis and most commonly excessive inflammatory response in sepsis. Cardiogenic shock is due to decreased cardiac output from an intrinsically cardiac cause, normally due to a large MI, myocarditis, or valvar disease. Obstructive shock is due to a decrease in cardiac output due to obstruction from an extrinsic to the heart, such as cardiac tamponade, tension pneumothorax or PE.

Cardiovascular changes during pregnancy include: Upward and leftward cardiac displacement, aortocaval compression after 20/40 with associated maternal hypotension and decreased uteroplacental flow (hence left lateral positioning). Placental blood flow of 625 mL/min, 30-50% increase in circulating volume (mediated by aldosterone, oestrogen and progesterone) ~12% increase in HR, 25% increase in stroke volume, 30-40% increase in cardiac output, 20-40% decrease in TPR due to the placental circulation (low resistance running in parallel with other systemic organs), 5-10 mmHg decrease in diastolic pressure at 12-20/40, increase in renal plasma flow and GFR (75% and 30% respectively), During labor: 300-500 mL autotransfusion with uterine contractions and ~25% increase in cardiac output.

Valsalva Manoeuvre was first described by the Italian Anatomist Antonio Valsalva in the early 18th century. It refers to the cardiovascular responses to forced expiration against a closed glottis for a duration of ten seconds or more. There are four main phases described in the diagram below.

Phase I Increased intrathoracic pressure causes a brief increase in preload as the pulmonary vasculature empties to the LA/LV. This causes an increase in CO and therefore blood pressure and a fast baroreceptor driven decrease in heart rate.

Phase II The increased intrathoracic pressure causes reduced venous return which leads to decreased preload and CO. As blood pressure now falls, the baroreceptor response is an increase in HR and TPR. The increase in TPR means that the diastolic pressure will be increased more than the systolic and as a result the pulse pressure narrows.

Phase III The intrathoracic pressure now drops, transiently decreasing preload and CO as blood pools in the hyperventilated lung. There is a transient decrease in blood pressure and circumferential and longitudinal stretch receptors located in the carotid sinus and aortic arch. The lungs are pushed out towards the mediastinal vessels and the aorta body. This causes a compensatory increase in heart rate and TPR. However, this is only a temporary response and the blood pressure will fall again.

Phase IV As CO returns to normal, the residual increase in TPR (and probable venous constriction) means that there is an overshoot and blood pressure spikes (with associated fast baroreceptor decrease in HR) before stabilising.

In heart failure most mechanisms are blunted and the pressure on the aorta leads to an increase in blood pressure and a characteristic ‘square wave’ pattern of blood pressure changes.