

Volume loss

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During simple haemorrhage, baroreceptors in the carotid artery and aortic arch and volume receptors in the wall of the left atrium initiate a different nerve input to the central nervous system, resulting in the release of both aldosterone and antidiuretic hormone (ADH). Pain can also stimulate ADH release. ADH acts directly on the kidney to cause fluid retention. Decreased pulse pressure stimulates the juxtaglomerular apparatus in the kidney and directly activates the renin-angiotensin system, which in turn increases aldosterone release. Aldosterone causes the renal tubule to reabsorb sodium (and consequently conserve water). ACTH release also augments the aldosterone response. The net effects of ADH and aldosterone result in the natural oliguria observed after surgery and conservation of sodium and water in the extracellular space. The tendency towards water and salt retention is exacerbated by resuscitation with saline-rich fluids. Salt and water retention can result in not only peripheral oedema but also visceral oedema (e.g. in the stomach). Such visceral oedema has been associated with reduced gastric emptying, delayed resumption of food intake and prolonged hospital stay. Careful limitation of intraoperative administration of balanced crystalloids so that there is no net weight gain following elective surgery has been proven to reduce postoperative complications and length of stay. Volume loss

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