

# Impact of liver resection on portal venous pressure and renal function



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## BACKGROUND:

- Liver dysfunction as well as increase in portal pressure per se (“hepatorenal reflex”) can lead to hepatorenal syndrome.
- Pathophysiology behind hepatorenal syndrome is mesenteric vasodilatation (“splanchnic pooling”) with a reduction of effective circulating plasma volume, countered by an increase of vasopressin levels and activation of sympathetic nervous system and renin-angiotensin-aldosteron system (RAAS) to stabilize systemic blood pressure despite effective hypovolemia.
- Acute kidney injury as indicated by oliguria and increased serum-creatinine is regularly observed after liver resection.
- Liver resection may therefore be a potent model to evaluate the impact of portal hypertension on the kidney despite stable liver function

## PATIENTS & METHODS:

- 24 patients (mean age 66.2 years) undergoing elective liver resection surgery because of malignant tumor were assessed and grouped according to resection size: (1) hemihepatectomy, n=14 versus (2) segmentectomy, n=10
- Hepatic venous pressure gradient (HVPG) was measured before and after resection by cannulation of a hepatic vein under fluoroscopic guidance.
- Liver function was assessed by indocyanine green plasma disappearance rate (ICG-PDR).

## AIM:

In this prospective investigation we determined the impact of liver resection on portal venous pressure by measuring the HVPG, on concentrations of vasoactive peptides and on renal function.

Figure 1. Hepatic venous pressure gradient before and after resection

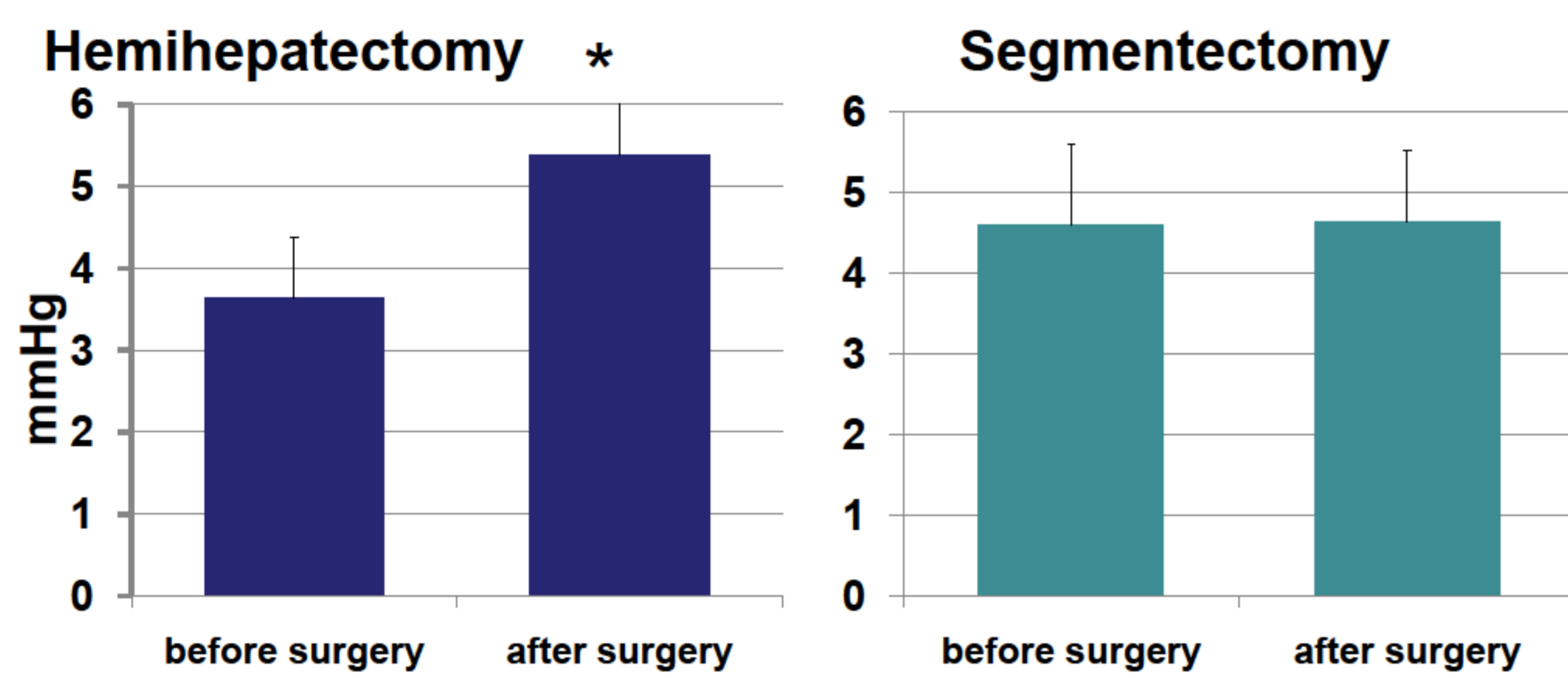


Figure 2. RAAS before, during and after surgery, and postoperative days 1 and 5

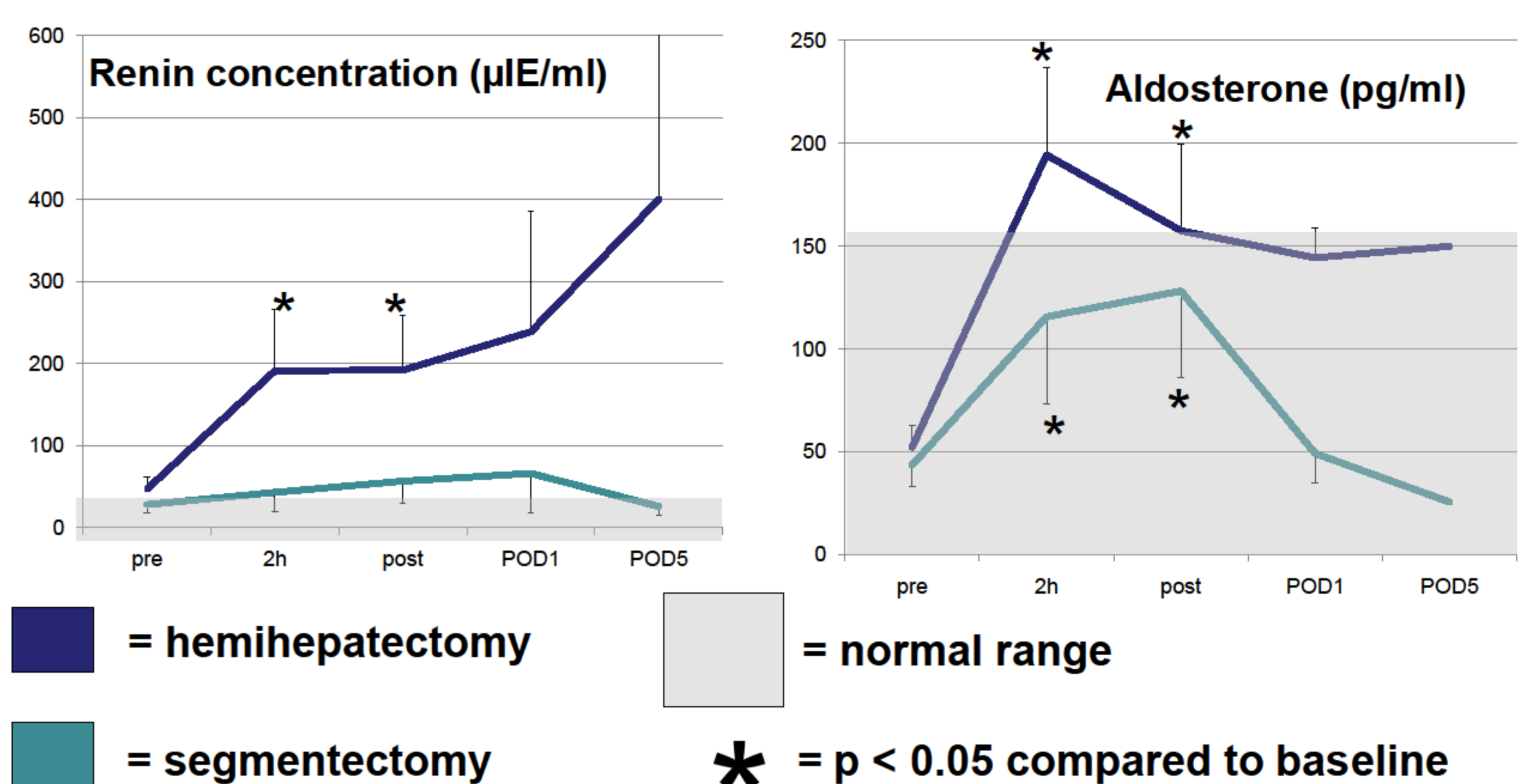


Figure 3. Indicators of effective circulating plasma volume before, during and after surgery, and postoperative days 1 and 5

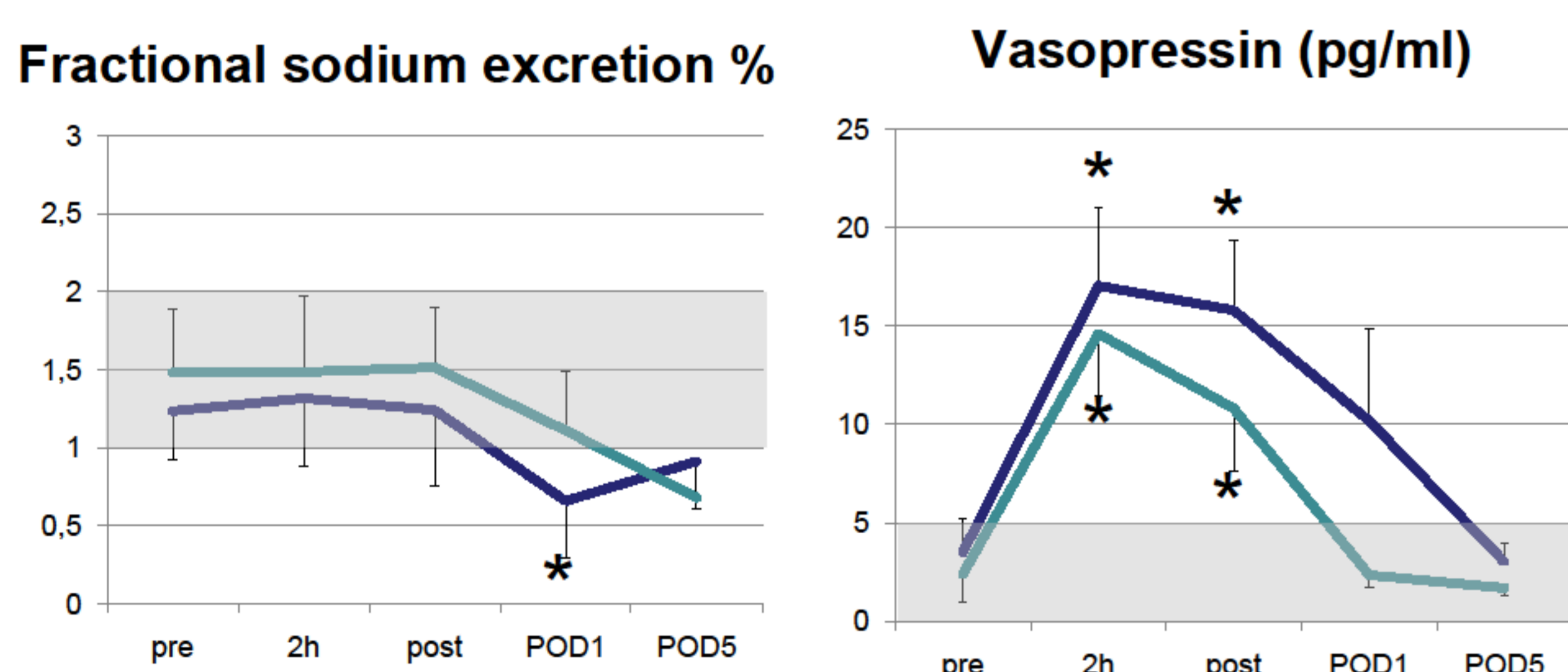
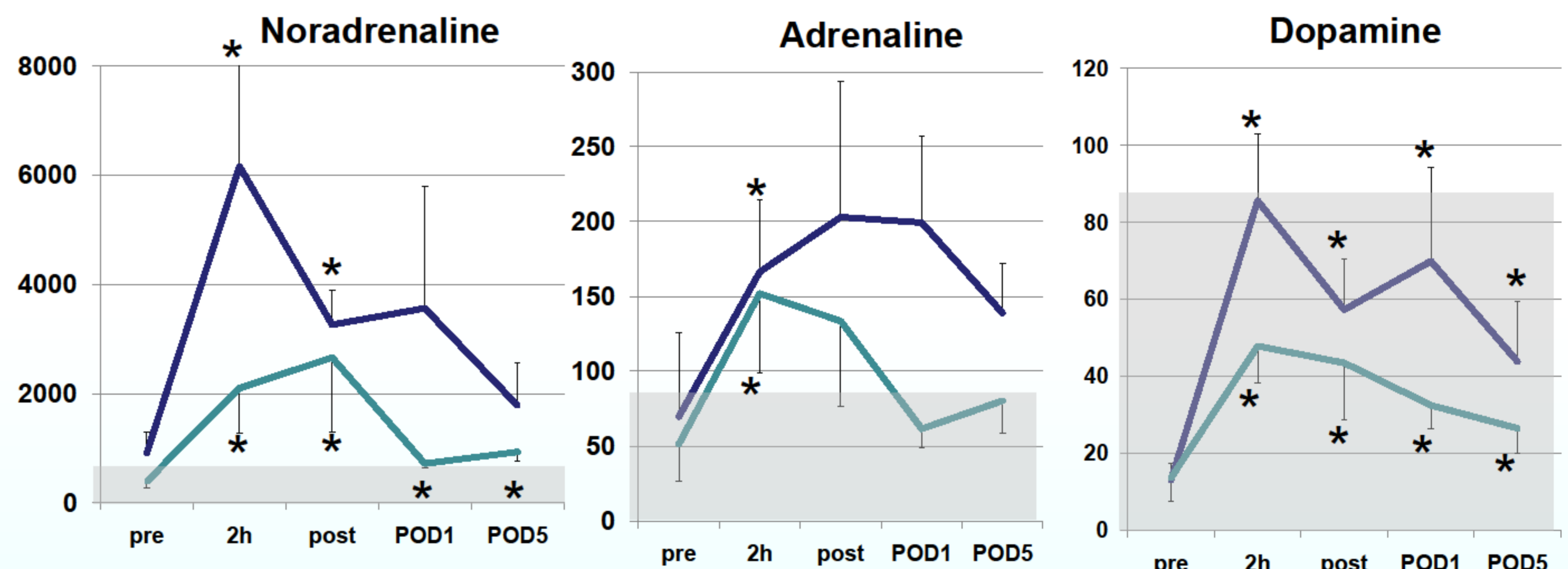


Figure 4. catecholamines (ng/l) before, during and after surgery, and postoperative days 1 and 5



## RESULTS:

- HVPG increased in group 1 from 3.6 to 5.4 mmHg (p<0.05) and remained stable in group 2 (4.6 to 4.6 mmHg, p=ns).
- ICG-PDR decreased in group 1 by day 1 (p<0.05) and remained stable in group 2.
- Renin, aldosterone, ADH, adrenaline, noradrenaline and dopamine increased significantly (p<0.05) in group 1 during operation. Group 2 showed a significant rise only in ADH and dopamine.
- Acute kidney injury occurred in 36% of patients in group 1 and no patient in group 2, respectively

## CONCLUSIONS:

- Liver resection increases the HPVg depending on resection size, presumably by reduction of hepatic vascular reserve.
- Regulatory mechanisms such as RAAS activation and ADH release activate immediately during operation, suggesting an effect independent of liver function.
- Acute kidney injury following liver resection in hemodynamically stable patients may be attributed to an increase in portalvenous pressure (hepatorenal syndrome).

