Re: “Outcome After Open Repair for Ruptured Abdominal Aortic Aneurysms in Patients with Friendly Versus Hostile Aortoiliac Anatomy”

The work by Dr. van Beek and colleagues represents a very interesting and substantial analysis on the ongoing controversy whether endovascular treatment in ruptured abdominal aortic aneurysms is superior to surgery. However, the presented data did not proof survival benefits for “friendly anatomy” in patients who underwent open surgery contrary to what has been shown in recent publications.2–4

Several studies have shown a lower mortality in endovascular suitable patients who underwent open surgery. Dick et al. reported a mortality ranging between 4% and 16%, Perrot et al. showed a mortality rate of 7%, and our group showed a mortality of 25%. The death rate of this highly selected patient group (including studies showing no survival benefit) is impressive, ranging between 4% and 46%. Technical issues are possible but surgical aortic repair is performed for more than 60 years and should not lead to that range of mortality. Furthermore, this variance of mortality tremendously affects the results and the drawn conclusions, especially when comparing surgery versus endovascular repair.

Interestingly, wherever endovascular treatment was readily available in addition to surgery, no survival benefit was proven in patients with “friendly anatomy” who underwent surgery (van Beek et al. 73 rEVAR vs. 72 OR; Ten Bosch et al. 25 rEVAR vs. 33 OR). In contrast, a lower death rate was evident in endovascular suitable patients treated by surgery, when far fewer endovascular procedures were performed, even so they were not included in the study (Dick et al. 11 rEVAR vs. 196 OAR; Perrot et al. 1 rEVAR vs. 16 OR; Krenzien et al. 5 rEVAR vs. 28 OR).

Obviously, there are unaccounted confounders, affecting the outcome of ruptured aortic aneurysms. Therefore, we believe that more risk stratification of subgroups is required to get reproducible results.

REFERENCES


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Response to ‘Re. Outcomes After Open Repair for Ruptured Abdominal Aortic Aneurysms in Patients with Friendly Versus Hostile Aortoiliac Anatomy’

Drs Krenzien and Fellmer have given an interesting and reasonable explanation for the discrepancies between studies assessing the outcomes of patients with friendly and hostile aortoiliac anatomy. The two studies that reported comparable outcomes harboured a larger group of patients treated with endovascular aneurysm repair (EVAR). Our sensitivity analysis, including patients treated with EVAR, showed a comparable risk of dying in those with friendly and hostile anatomy (adjusted odds ratio 1.090, 95% confidence interval 0.593–2.004), which is somewhat contradictory to the explanation given by Krenzien and Fellmer. Nevertheless, we acknowledge their explanation as a valuable addition to those previously described, such as the method of anatomical classification (prospectively vs. retrospectively) and selection bias by haemodynamic stability.

From the patient’s perspective, these discrepancies and explanations are less relevant as aortoiliac anatomy cannot be treated or altered. The controversy about the optimal treatment modality is more relevant. Therefore, we disagree with Krenzien and Fellmer that more risk stratification of subgroups is needed. We are convinced that more randomized trials assessing the optimal treatment for patients with a ruptured abdominal aortic aneurysm are needed. Based on the recently published trials, EVAR has not been shown to be inferior to open repair. Therefore, possible future directions of studies are comparisons between a treatment strategy that includes both EVAR and open repair versus an ‘EVAR-only’ approach or versus an ‘EVAR-first’/hybrid repair approach.
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Re. ‘Abdominal Hypertension and Decompression: The Effect on Peritoneal Metabolism in an Experimental Porcine Study’

We have read, with great interest, the recently published article by Skoog et al.1 in which the authors have aimed to present metabolic changes in peritoneal tissue, alterations of blood gas/acid base, and hemodynamic parameters during prolonged intra-abdominal CO2 insufflation in a porcine model. Our research interests are linked to this topic and we would therefore like to express our respect for the comprehensively monitored data collected by these authors, which included intra-abdominal pressure, hemodynamics, intestinal blood flow, urine output, and arterial blood gas and metabolite (S-lactate and S-glucose) values. These data may help clinicians to understand the pathophysiological mechanisms of CO2 pneumoperitoneum side effects. In our opinion, repeatedly monitored lactate and glycerol values from the peritoneal cavity, and jejunal and rectal canals could clearly present the dynamics of change of these metabolites during prolonged CO2 insufflation. It is probable that the slight hyperventilation of animals by a volume-controlled Monnal-D-ventilator with a 15-min/L inspiratory rate obscures the hypoxic impact of prolonged CO2 insufflation under 6-h relatively high intra-abdominal pressure (30 mmHg).1,2 We have already shown that intra-abdominal CO2 insufflation profoundly affects blood gases, acid—base balance, and oxygen homeostasis, resulting in metabolic hypoxemia.3 The effect of this strictly depends on respiratory parameters (tidal volume and rate of ventilation) and depth of anesthesia, where spontaneous breathing appears to be more harmful than optimally controlled ventilation (unpublished data; Fig. 1). In our experiments, the control group includes animals without CO2 pneumoperitoneum,

Figure 1. Effect of the intraperitoneal CO2 insufflation on arterial blood lactate values in rabbits with optimal (○) and superficial ventilation (□) in comparison with spontaneously breathing (●) and control animals (●).