IIa. And when the dissection extends to the distal trunk of the superior mesenteric artery with thrombosed false lumen and occluded true lumen, we may describe it as a type C-III.

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J.Y. Luan, X. Li*
Department of Interventional Radiology and Vascular Surgery, Peking University Third Hospital, Beijing, China

*Corresponding author. X. Li, North Garden Road 49, Haidian District, Beijing 100191, China.
Email-address: 13801209697@139.com (X. Li)

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Response to 'Left Renal Vein Division During Open Surgery of Abdominal Aortic Disease: A Propensity Score-matched Case-Control Study'

Thanks for the comment. Some studies indicate that left renal vein division (LRVD) is a safe procedure during aortic surgery and some demonstrate it's association with post-operative renal insufficiency, especially in pararenal aortic aneurysm repair.[1,2] The purpose of our study is to try to answer the question of whether LRVD leads to some deleterious effects or is only a marker for the complexity of the operative procedure. It's hard to really understand the fate of the left kidney after LRVD because there has been no study evaluating split renal function. However, we do believe that the left renal vein (LRV) should be reconstructed in juxtarenal AAA patients who require suprarenal aortic clamping, in patients lacking collateral tributaries for drainage of the left kidney, or in patients with preoperative chronic renal insufficiency. Beyond this, our study confirms LRVD without reconstruction is safe for infrarenal abdominal aortic disease in Chinese patients, who have a younger average age and better preoperative renal function compared with Western populations.[3]

Marrocco-Trischitta et al.[4] reported the safety of LRV reconstruction. Maybe it's because of different anatomies in the treated population that we encounter the complications of intra- or postoperative bleeding associated with LRV reanastomosis. Therefore, in well-selected patients, we consider LRVD without reconstruction to be safe and can simplify the whole procedure.

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S.J. Xin*
Division of Vascular and Thyroid Surgery, Department of General Surgery, First Hospital of China Medical University, Liaoning, China

*Division of Vascular and Thyroid Surgery, Department of General Surgery, First Hospital of China Medical University, 155 Nanjing North Street, Shenyang, Liaoning 110001, China.
Email-address: sjxin@mail.cmu.edu.cn

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Re. 'Left Renal Vein Division During Open Surgery of Abdominal Aortic Disease: A Propensity Score-matched Case-control Study'

We read with interest the paper by Wang et al.[1] The authors have found that left renal vein division (LRVD) may be a safe maneuver during abdominal aortic surgery as it did not increase the risk of early or a late mortality and morbidity.[1] Standard open repair of juxtarenal abdominal aortic aneurysm (AAA) quite frequently requires a procedure with left renal vein. Approximately 15—20% of treated AAA in our clinic are juxtarenal. However, in some cases, a LRVD and re-anastomosis should be performed. According to our experience, LRVD without reconstruction is only indicated during emergency aortic surgery. Re-anastomosis of the LRV is a relatively simple procedure and we think that except in the previously mentioned situation (emergency) there should be no reason not to reconstruct it. If we cut something, we are obliged to repair it. However, reconstruction is necessary, especially in patients with signs of chronic renal insufficiency and in cases of intraoperative huge renal vein finding, which suggests compensatory enlargement due to venous drainage problems. If reconstruction is not possible it's of huge importance to preserve the gonadal or suprarenal vein. At the same time, these procedures do not affect early and late renal function.[2] We found that LRV temporary transection and re-anastomosis did not have any influence on early and mid-term renal function.

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L. Davidovic, N. Ilić*, M. Markovic, M. Dragas, I. Koncar
Clinic for Vascular and Endovascular Surgery, Clinical Center of Serbia, Belgrade, Serbia
Medical faculty, University of Belgrade, Serbia

I. Banzic
Clinic for Vascular and Endovascular Surgery, Clinical Center of Serbia, Belgrade, Serbia

*Corresponding author. N. Ilić, Koste Todorovića Street 8, 11000 Belgrade, Serbia.
Email-address: fosafosa75@yahoo.com (N. Ilić)

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Re 'How Should We Measure and Report Elasticity of Aortic Tissue?'

We read with interest the article by Khanafer et al.[1] but found that it contains many incorrect or misleading statements, interpretations, and conclusions concerning stress, strain, and elastic modulus measures that are used to analyse the elasticity of aortic tissue. Because of limited space we quote only one example, from the Abstract: "We found that the Almansi-Hamel strain definition exhibited the highest non-linear stress-strain relation and consequently may overestimate the elastic modulus ...". This is misleading because there are simple connections between the elastic moduli associated with different stresses and strains. They all carry the same information.

The strain measures used in Khanafer et al.[1] are members of the family $(\lambda^m - 1)/m$, $m = 1,2,-2,0$. The stresses σ_T, σ_E, and S are related by $\sigma_T = \lambda\sigma_E = \lambda^2 S$. For the engineering strain the elastic moduli associated with these stresses are $d\sigma_T/d\lambda$, $d\sigma_E/d\lambda$ and $dS/d\lambda$, respectively, and they are simply related by

$$\frac{d\sigma_T}{d\lambda} = \lambda\frac{d\sigma_E}{d\lambda} + \sigma_E = \lambda^2\frac{dS}{d\lambda} + 2\sigma_E. \quad (1)$$

Equation (1) scales by a common factor (λ^{-1}, λ or λ^3) for each of the other strain measures. As σ_E is positive, it follows from Equation (1) that the patterns shown in Fig. 5 are immediately obvious. A similar pattern to that in Fig. 5 appears in the first two panels of Fig. 4, which relates to the so-called hypertensive elastic modulus (not defined), although it has been stated that the differences in the elastic moduli are not significant. They are, but those in the third panel of Fig. 4 are not. However, the data therein are not consistent with Equation (1) and cannot be correct (the true stress modulus must be larger than the engineering stress modulus by definition). The "errors" referred to in Table 3 are not errors. The differences are merely manifestations of the connections (Equation (1)).

The stress—strain plots in Fig. 3 are substantially redundant and also misleading as, for example, each of the 12 plots in (a) contains the same information; the changes from engineering stress to the true stress and the second Piola-Kirchhoff stress merely involve scaling of the vertical axis by λ and λ^{-1}, respectively, and the four curves in each panel of Fig. 3 are equivalent because they simply translate from one to the other by a change of the strain, that is by a nonlinear scaling of the horizontal axis. The figures in Fig. 3 are not needed to elaborate the obvious fact that the stress—strain curves and "the elastic modulus" are very dependent on the choice of variables.

Contrary to the assertion of the authors, the choice of stress, strain, and elastic modulus measures is not important for the analysis because there are simple connections between them. In any case, the concept of elastic modulus is not useful in nonlinear mechanics. What is really needed is an explicit nonlinear relation between stress and strain, that is a constitutive equation, that characterizes the material properties of the soft tissue in question. Also, one-dimensional tests yield very limited information about tissue mechanical properties. To assess more fully the elastic properties of, for example, aortic tissues, multi-dimensional