Effects of Endothelin in Integrated Venous Function in Health and Disease

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Current Management of Venous Diseases

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This revised and enlarged edition of Cerebral Angiography, which includes new angiographic studies and illustrative drawings, offers detailed guidance on diagnostic imaging, and medical and endovascular surgical management of acute and chronic venous diseases. Dr. Jose Almeida, pioneering expert in the field and host of the annual International Vein Congress, along with other highly regarded practitioners, offers an authoritative understanding of what causes increased venous pressure and solutions for reducing venous hypertension. Detailed, full-color intraoperative illustrations capture key teaching moments, helping you better understand the nature of surgery and improve your ability to perform cutting-edge procedures.

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The book includes an overview of the evidence and demonstrates the clinical utility of novel treatments. Chapters are written by leading experts in the field and reflect state-of-the-art knowledge. The book will be of great use to clinicians, researchers, and students interested in understanding the pathophysiology, pulmonology and critical care medicine. Therefore, the book will be of great value to the physicians and trainees in those specialties and to any person interested in developing in depth knowledge of the management of venous diseases.

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endothelial dysfunction is secondary to the inflammatory and neurohumoral activation associated with the heart failure syndrome, or a primary pathophysiological factor in the development of either form of heart failure. As regards venous function, patients with HF-PSF had a lower venous capacitance than patients with HF-RSF (but similar venous capacitance to the controls). Increased venous capacitance may represent a compensatory response in heart failure that is less marked or absent in HF-PSF compared to HF-RSF. Venous endothelial function was measured with the Aellig dorsal hand vein technique. It was not technically possible to complete an Aellig study in the whole patient cohort, resulting in fewer data being available for analysis. Despite this, both heart failure groups appeared to have impaired venodilatation in response to acetylcholine, compared to controls, although this apparent difference was not statistically significant. While the studies of venous capacitance and endothelial function were not conclusive, they suggest that venous function may be abnormal in HF-PSF. The finding that endothelial and smooth muscle control of arterial tone was impaired in both HF-PSF and HF-RSF may indicate a similar primary pathophysiological process, or indeed a similar response to inflammatory and neurohumoral activation in heart failure. I conclude that the data presented in this thesis supports the hypothesis that HF-PSF is associated with increased arterial stiffness, which in combination with increased LV stiffness is likely to result in impaired ventriculo-vascular coupling. This process is likely to be an important pathophysiological factor in the development of HF-PSF.

Studies of Vascular Function in Patients with Heart Failure and Either Preserved Or Reduced Left Ventricular Systolic Function - Sean Balmain - 2008

Up to 50% of patients with the clinical syndrome of heart failure have preserved left ventricular systolic function (HF-PSF). Invasive studies utilizing cardiac catheterization have demonstrated that patients with HF-PSF have abnormalities of left ventricular (LV) relaxation and filling, or LV diastolic dysfunction. As a result, it has been proposed that LV diastolic dysfunction is the primary pathophysiological process in HF-PSF. However, population-based studies have shown that there is poor correlation between the presence of LV diastolic dysfunction and the presence of heart failure. This controversy has led to a search for alternative pathophysiological processes which could potentially cause HF-PSF. There are some data to suggest that patients with HF-PSF have a combination of LV diastolic dysfunction, or "LV stiffness," and large artery stiffness, when compared with normal subjects and patients with systemic hypertension. This implies that the interaction between the left ventricle and the vasculature is dysmictfunctional and a potential cause of HF-PSF. Although there are limited data on arterial stiffness in HF-PSF, there have been no studies examining other parameters of vascular function in HF-PSF and vascular function has never been formally compared in cohorts of patients with HF-PSF and heart failure due to reduced LV systolic function (HF-RSF). The studies presented in this thesis were designed to further characterize vascular function in HF-PSF and to compare vascular function between patients with HF-PSF, patients with HF-RSF and control subjects. I used non-invasive techniques to assess parameters of arterial function, such as arterial stiffness and arterial endothelial function. I also evaluated parameters of venous function, namely venous capacitance and venous endothelial function. Arterial stiffness, measured by aortic pulse wave velocity (PWV), was significantly elevated in HF-PSF compared to both HF-RSF and control groups, implying that HF-PSF is indeed associated with greater arterial stiffness. In contrast, arterial diastolic waveform analysis failed to show any significant differences in derived parameters of arterial compliance between the three study groups, which may be due to the fact that all three groups were matched for underlying coronary heart disease, reducing the ability of the technique to differentiate between groups. Using Laser Doppler iontophoresis, I demonstrated that HF-PSF and HF-RSF subjects have impaired microvascular responses to both acetylcholine and sodium nitroprusside. This suggests that, rather than being solely a primary disorder of endothelial function, impaired control of vascular tone in HF-PSF reflects significant vascular smooth muscle dysfunction. It is not certain if arterial smooth muscle and/or endothelial dysfunction is secondary to the inflammatory and neurohumoral activation associated with the heart failure syndrome, or a primary pathophysiological factor in the development of either form of heart failure. As regards venous function, patients with HF-PSF had a lower venous capacitance than patients with HF-RSF (but similar venous capacitance to the controls). Increased venous capacitance may represent a compensatory response in heart failure that is less marked or absent in HF-PSF compared to HF-RSF. Venous endothelial function was measured with the Aellig dorsal hand vein technique. It was not technically possible to complete an Aellig study in the whole patient cohort, resulting in fewer data being available for analysis. Despite this, both heart failure groups appeared to have impaired venodilatation in response to acetylcholine, compared to controls, although this apparent difference was not statistically significant. While the studies of venous capacitance and endothelial function were not conclusive, they suggest that venous function may be abnormal in HF-PSF. The finding that endothelial and smooth muscle control of arterial tone was impaired in both HF-PSF and HF-RSF may indicate a similar primary pathophysiological process, or indeed a similar response to inflammatory and neurohumoral activation in heart failure. I conclude that the data presented in this thesis supports the hypothesis that HF-PSF is associated with increased arterial stiffness, which in combination with increased LV stiffness is likely to result in impaired ventriculo-vascular coupling. This process is likely to be an important pathophysiological factor in the development of HF-PSF.

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