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Early hemodynamic responses to rapid hemorrhage were studied in anesthetized open chest dogs to test whether Leo Sapirstein's hypothesis is correct that an increase in venous resistance leads to shock after hemorrhage instead of an increase in arterial resistance. Electromagnetic blood flow transducers were used to measure cardiac output in the ascending aorta and venous return in the superior and inferior venae cavae. Systemic arterial, peripheral and central venous pressures were measured with strain gauges, and venous pressures were also measured with water manometers. Rapid removal of 30% of the blood volume caused significant (p Peripheral Vascular Surgery aims to provide an updated and authoritative introduction to peripheral vascular surgery. Most arterial and venous diseases appear to be mainly the result, directly or indirectly, of mural deposition of solid material within a rapidly moving stream of blood. The vascular surgeon must therefore have a working knowledge of blood platelet interactions and probable mechanisms of

thrombosis. He must also understand both the value and the limitations of present methods of blood flow measurement. This book provides an updated description of these complexities and presents essential basic knowledge that will prove valuable to surgeons and researchers working in this field. This text will be useful for post graduate surgeons, as well as for those in need of a reference useful for doctors in related fields such as general medicine and cardio-thoracic and orthopedic surgery. Now in its revised, updated Second Edition, this volume is a thorough, practical guide to the use of Doppler sonography in evaluating peripheral vascular disease. Dr. Polak describes techniques for optimizing image acquisition and provides the clinical and pathophysiologic information necessary for accurate image interpretation. This edition features over 600 new illustrations, including 197 full-color images throughout the book. Chapters cover neck arteries, venous thrombosis, chronic venous thrombosis and venous insufficiency, peripheral arterial disease, and imaging after operative and endovascular interventions. Images are linked to descriptions of pathophysiologic processes so that readers clearly understand the clinical significance of sonographic findings. Purpose of the studyResuscitation guidelines recommend assessing and correcting reversible causes of cardiac arrest (CA) during cardiopulmonary resuscitation (CPR) i.e. by using the 4 Hu2019s and 4 Tu2019s rule. Point-of-care (POC) laboratory analyses could probably supplement clinical information in screening pre-arrest pathologies, i.e. electrolyte disorders, acid-base balance disturbances and abnormal haemoglobin levels.Because arterial or venous blood samples may be

difficult to obtain during CPR, we aimed to study how POC samples from intraosseous (IO) space during resuscitation resemble the pre-arrest arterial values.Materials and methodsWe used an experimental resuscitation model with 23 anesthetised pigs. The Finnish National Animal Experiment Board (ESAVI/1077/04.10.07/2016) approved the study plan. We took baseline blood samples from artery before electrically inducing VFand compared the baseline arterial samples with arterial, venous and IO-samples taken after 5 minutes of CPR, which was started after 7 minutes of untreated CA. We used iSTATu00ae POC device for analyses and Friedman's two-way ANOVA and its post hoc pairwise comparisons with Bonferroni correction for the statistical analyses.ResultsElectrolyte and acid-base values from all sampling sites during resuscitation differ from the pre-arrest values. Figures below display the difference between arterial, IO-, and venous values during CPR compared to the pre-arrest arterial values.ConclusionsVenous, arterial and IO POC samples during CPR resemble poorly the pre-arrest values. POC analysis of IO samples is not inferior to analyses of arterial or venous blood. Structural and functional abnormalities of arteries and veinsmanifest clinically in a broad spectrum of disorders, includinganeurysmal disease, atherosclerosis, vasculitis, venousinsufficiency, microvascular complications, thrombo-embolism andlower limb ulceration. Many of these are common and/orchronic conditions which present for initial assessment by primaryhealth care workers. This new edition is a practical guide to the most commonlypresenting disorders, and provides a structured approach toclinical

assessment, investigations and management. The lastfew years have seen major changes in the use of non-invasive orminimally-invasive techniques, e.g wider use of CT and MRangiography, and increasing use of percutaneous interventions forcarotid, lower limb and reno-vascular disease. The ABC of Arterial and Venous Disease (Second Edition)explains the underlying technology and the applications of newminimally-invasive methods, especially CT and MRI, and provides anup-dated, evidence-based guide to the modern day management ofpatients with common, life-threatening diseases involving differentparts of the circulation. This authoritative, full-colour, illustrated ABC is anideal reference for the primary care, non-specialist practitionerto base effective management and prevention programmes. Ethanol (0.40 g/kg) was administered to 13 healthy men by intravenous (i.v.) infusion at a constant rate for 30 min. The concentrations of ethanol in arterial blood (ABAC), venous blood (VBAC), and end-expired breath (BrAC) were measured at 17 exactly timed intervals. Blood-ethanol was determined by headspace gas chromatography and breath-ethanol was measured with a quantitative infrared analyzer (DataMaster) BrAC was multiplied by 2300 to estimate the concentrations of alcohol in blood. During the infusion of ethanol, ABAC exceeded VBAC by about 10 mg/dL on the average and ABAC was also higher than BrAC × 2300 by about 4 mg/dL on average. When infusion of alcohol ended, ABAC, VBAC, and BrAC were 94.8 ± 2.06 (±SE), 84.7 ± 1.54, and 89.3 ± 2.10 mg/dL, respectively. The concentrations of alcohol in blood (ABAC and VBAC) and breath decreased abruptly after the administration of alcohol

stopped and by 5 min postinfusion, the A-V differences in concentration of ethanol were small or negligible. The mean apparent half-life of the distribution plunge was 7 to 8 min, being about the same for ABAC, VBAC, and BrAC. The disappearance rate of ethanol was 15.5 ± 0.55 mg/dL/h (mean ± SE) for arterial blood, 15.2 ± 0.49 mg/dL/h for venous blood, and 16.3 ± 0.73 mg/230 L/h for breath; no significant differences were noted ($p > 0.05$) We conclude that A-V differences in the concentration of ethanol exist during the loading phase but are rapidly abolished when the administration of ethanol terminates. In the post-absorptive phase of ethanol kinetics, when alcohol has mixed with the total body water, VBAC exceeds ABAC by about 1-2 mg/100 mL on average. Now in its 6th edition, Introduction to Vascular Ultrasonography, by Drs. John Pellerito and Joseph Polak, provides an easily accessible, concise overview of arterial and venous ultrasound. A new co-editor and new contributors have updated this classic with cutting-edge diagnostic procedures as well as new chapters on evaluating organ transplants, screening for vascular disease, correlative imaging, and more. High-quality images, videos, and online access make this an ideal introduction to this complex and rapidly evolving technique. Find information quickly with sections organized by clinical rationale, anatomy, examination technique, findings, and interpretation. Get a thorough review of ultrasound vascular diagnosis, including peripheral veins and arteries, carotid and vertebral arteries, abdominal vessels, and transcranial Doppler. Quickly reference numerous tables for examination protocols, normal values, diagnostic parameters, and

ultrasound findings for selected conditions. Visualize important techniques with hundreds of lavish line drawings and clinical ultrasound examples. Stay current with trending topics through new chapters on evaluation of organ transplants, screening for vascular disease, correlative imaging, and accreditation and the vascular lab. Experience clinical scenarios with vivid clarity through new color ultrasound images. Watch vascular ultrasound videos and access the complete contents online at www.expertconsult.com. Benefit from the fresh perspective and insight of a new co-editor, Dr. Joseph Polak. Improve your understanding of the correlation of imaging results with treatment goals in venous and arterial disease. Learn the principles of vascular ultrasonography from the most trusted reference in the field. Platelets are essential mediators of the physiologic process of hemostasis and pathologic thrombosis. While platelets do not interact with vascular walls under normal conditions, vascular injury or inflammation result in a coordinated series of events including platelet adhesion, aggregation, and promotion of coagulation. In this review, we describe the primary mechanisms involved in these responses in various vascular beds of both macro- and microvessels, and outline key unresolved aspects of these important interactions. There are a number of different techniques used to diagnose vascular insufficiency ranging from expensive hospital based equipment to less expensive devices used in primary care centres. Currently, some of these devices are unsuitable for use on patients with diabetes or DVI and have poor sensitivity for detecting moderate PAD patients.

Additionally, some of the tests, particularly for DVI, require tourniquets or the patient to perform postural changes which some may find difficult. This may extend testing time. The study investigated 2 groups of patients, one with PAD and the other with DVI. The arterial group consisted of 46 controls and 57 patients. PPG probes were placed on the index finger and great toe. The venous group consisted of 24 controls and 25 patients and PPG probes were placed behind the knee and 10 cm above the medial malleolus. Duplex ultrasound was used as the gold standard to assess the arteries and veins in the lower limbs. The aim was to investigate whether signals acquired from patients at rest using Photoplethysmography (PPG) could be used as a screening tool. Pulse wave transit time (PWTT) and shape analysis techniques were used on the pulses from the patients with PAD, while time base and spectral analysis techniques were used on the waveforms of patients with DVI. PWTT and shape analysis techniques achieved sensitivities and specificities of 82% and 84% respectively. Accuracy dropped to 70% for detecting patients with moderate PAD. Spectral analysis techniques gave the best results for detecting patients with DVI achieving sensitivities and specificities of 69% and 80% respectively. In conclusion, reducing the signal acquisition time on patients with PAD did not significantly reduce the sensitivity and specificity. Without any patient movement it was difficult to separate patients with DVI from healthy normals. Background: Vascular complications of diabetes are due in part to impaired angiogenesis. The extent to which these problems are due to disturbed identification of newly formed endothelial cells

(EC) as 'arterial' or 'venous' (i.e. AV specification), recruitment of perivascular cells (PVC), or perturbed growth factor expression, had not been addressed. Methods Results: Using microvascular preparations from type-I diabetes (T1D) and non-diabetic animals transplanted into T1D and non-diabetic hosts, we show that mice T1D have impaired vessel AV specification, displaying ubiquitous expression of arterial marker ephrin-B2, loss of hierarchical organization, and reduced PVC coverage. Blockage of PVC recruitment in normoglycemic mice did not affect blood flow, but recapitulated the vascular immaturity of T1D, implicating PVCs in AV-specification. The latter occurred with downregulated Jagged1 and Notch3, key modulators of EC-PVC interactions. Co-cultures showed that PVCs induce arterial identity by increasing ephrin-B2, while decreasing venous marker eph-B4. This was antagonized by hyperglycemia or by inhibiting Jagged1 in EC. Vessel sprouting and consequent loss of AV identity was also dependent on hepatocyte growth factor (HGF) secreted from PVCs, with HGF levels decreased in T1D. Vessel anastomosis and implant perfusion was delayed in T1D,suggesting a role for HGF in inosculation. Finally, EC exposed to pulsatile stretch (mimicking pulsatile flow) upregulated platelet-derived growth factor-b (PDGFb), which was blunted by hyperglycemia. Importantly, microvessels from mice with T1D displayed normal PVC coverage, hierarchical organization and Jagged1/Notch3 expression after implantation into non-diabetic hosts, indicating no 'metabolic memory'. Conclusion: We show that AV identity is impaired in T1D, due to abnormal PVC recruitment,

dysregulated expression of HGF (by PVC) and PDGFb (by EC). These findings are associated with disordered Jagged1-Notch3 signaling, and lead to ineffective vascular connections with the host, which can be normalized in non-diabetic hosts. Research centering on blood flow in the heart continues to hold an important position, especially since a better understanding of the subject may help reduce the incidence of coronary arterial disease and heart attacks. This book summarizes recent advances in the field; it is the product of fruitful cooperation among international scientists who met in Japan in May, 1990 to discuss the regulation of coronary blood flow. Percutaneous cardiac and endovascular procedures are performed by a variety of interventional physicians and continue to evolve and expand. One of the most important steps in performing these procedures is vascular access and their Achilles heel is vascular access site complications. This volume is intended to help the clinician by providing a practical overview of the techniques and technologies used in top catheterization laboratories to access the arterial and venous beds. Dr. Mazen Abu-Fadel and his contributors, part of the renowned cardiovascular team at the University of Oklahoma Health Sciences Center, carefully walk the reader through the various techniques used to obtain vascular access into most arterial and venous sites. They thoroughly describe current data, techniques, advantages, risks, and benefits of each vascular access site. Covering everything from anatomic landmarks to closures devices, Arterial and Venous Access in the Cardiac Catheterization Lab offers a complete overview of each procedure. In addition, it provides an up-to-date guide to the best medical

technologies and equipment used when performing these procedures. Arterial and Venous Access in the Cardiac Catheterization Lab is an invaluable resource for a wide range of clinical personnel, from attending physicians and trainees to nursing staff and vascular technicians. Written by experienced leaders in the field, it demonstrates how to perform complex, risky procedures while providing patients with expert care. ABC of Arterial and Venous Disease provides a structured, practical approach to clinical assessment, investigation and management of the most commonly presenting arterial and venous disorders. Structural and functional abnormalities of arteries and veins manifest clinically in a broad spectrum of disorders, including cerebrovascular and carotid artery disease, abdominal aortic aneurysms, acute and chronic limb ischaemia, vasculitis and varicose veins. This revised edition incorporates new chapters on coronary artery disease and acute coronary syndrome, visceral artery stenosis and mesenteric ischaemia, and arteriovenous malformations. Many of these common or chronic conditions first present for initial assessment by primary health care professionals. Case vignettes have been added to relevant chapters to aid understanding and decision making. Fully up to date and from an expert editor and contributor team, ABC of Arterial and Venous Disease remains a useful resource for non-specialist doctors such as general practitioners, family physicians and junior doctors in training. It is also a relevant guide for all other primary health care professionals working within the multidisciplinary teams responsible for patients with chronic arterial and venous disorders. The

hemodynamic mechanisms of hypertension are often limited to the study of three dominant parameters: blood pressure, cardiac output and vascular resis tance. Accordingly, the development of hypertension is usually analyzed in terms of a 'struggle' between cardiac output and vascular resistance, resulting in the classical pattern of normal cardiac output and increased vascular resistance, thus indicating a reduction in the caliber of small arteries. However, during the past years, the clinical management of hypertension has largely modified these simple views. While an adequate control of blood pressure may be obtained with antihypertensive drugs, arterial complications may occur, involving mainly the coronary circulation and suggesting that several parts of the cardiovascular system are altered in hypertension. Indeed, disturbances in the arterial and the venous system had already been noticed in animal hypertension. The basic assumption in this book is that the overall cardiovascular system is involved in the mechanisms of the elevated blood pressure in patients with hypertension: not only the heart and small arteries, but also the large arteries and the venous system. For that reason, the following points are emphasized. First, the cardiovascular system in hypertension must be studied not only in terms of steady flow but also by taking into account the pulsatile components of the heart and the arterial systems. Second, arterial and venous compliances are altered in hypertension and probably reflect intrinsic alterations of the vascular wall. Although cardiac output is measured as the flow of blood from the left ventricle into the aorta, the system that controls cardiac output includes many other components besides the heart itself. The heart's rate of

output cannot exceed the rate of venous return to it, and therefore, the factors governing venous return are primarily responsible for control of output from the heart. Venous return is affected by its pressure gradient and resistance to flow throughout the vascular system. The pressure gradient for venous return is a function of several factors including the blood volume flowing through the system, the unstressed vascular volume of the circulatory system, its capacitance, mean systemic pressure, and right atrial pressure. Resistance to venous return is the sum of total vascular resistance from the aortic valve to the right atrium. The sympathetic nervous system and vasoactive circulating hormones affect short-term resistance, whereas local tissue blood flow autoregulatory mechanisms are the dominant determinants of long-term resistance to venous return. The strength of contraction of the heart responds to changes in atrial pressure driven by changes in venous return, with small changes in atrial pressure eliciting large changes in strength of contraction, as described by the Frank-Starling mechanism. In addition, the autonomic nervous system input to the heart alters myocardial pumping ability in response to cardiovascular challenges. The function of the cardiovascular system is strongly affected by the operation of the renal sodium excretion-body fluid volume-arterial pressure negative feedback system that maintains arterial blood pressure at a controlled value over long periods. The intent of this volume is to integrate the basic knowledge of these cardiovascular system components into an understanding of cardiac output regulation. Table of Contents: Introduction / Venous Return / Cardiac Function / Integrated Analysis of Cardiac Output

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