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E L S E V I E R

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NEW ANTITHROMBOTIC STRATEGIES IN ARTERIAL THROMBOSIS

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Over the last decade, a series of clinical trials has documented the efficacy and safety of new antithrombotic strategies. The most clinically relevant data can be summarized as follows:

- 1) In primary prevention trials, oral anticoagulants (but not aspirin) lower the number of fatal myocardial infarctions in high-risk individuals. The risk of bleeding associated with this strategy deserves further evaluation;
- 2) A novel thienopyridine derivative, clopidogrel, is better than aspirin in the prevention of recurrence of ischemic events in patients with PAD;
- 3) The additive effect of ticlopidine and aspirin in the prevention of coronary stent thrombosis has been demonstrated. This supports the possibility of combinations of antithrombotic agents in high-risk individuals;
- 4) In combination with aspirin, several Gp IIb/IIIa antagonists infused intravenously, are more effective than aspirin alone in unstable angina and in MI, and are more effective than aspirin in preventing restenosis in patients undergoing PTCA. This further supports the possibility of improved efficacy of combinations of antithrombotic agents in high-risk settings;
- 5) The ability of a dietary supplementation n-3 polyunsaturated fatty acids to prevent recurrence of myocardial infarction, and to lower the risk of sudden death, has been established (GISSI-prevenzione Study);
- 6) Folic acid and vitamin B₁₂ normalize moderately elevated levels of homocysteine, a risk factor for arterial and venous thrombosis. This further supports the role of nutrition in the risk of arterial thrombosis;
- 7) In view of the results of the European Stroke Prevention Trial 2 (ESPS-2), the clinical efficacy of dipyridamole has to be reassessed;
- 8) Direct thrombin inhibitors (desirudin, bivalirudin) are at least as effective as heparin in patients with unstable angina, deep vein thrombosis, and PTCA;
- 9) Statins affect central prothrombotic mechanisms (*in vivo* platelet activation NO formation, tissue factor expression). This supports the concept of statins as antithrombotic agents.

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THE INTIMA MEDIA THICKNESS

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The progression from arterial wall injury to the clinical phase of cardiovascular disease, crosses one or more pre-clinical steps (not symptomatic), detectable by non-invasive diagnostic techniques. B-Mode ultrasound (US) of superficial arteries (carotid and femoral), is one of the most reliable techniques to approach this problem, providing excellent information on the pre-clinical phase of the disease. B-Mode US is totally non-invasive, and can be used, with multiple scans, to evaluate the effectiveness of treatments also in asymptomatic subjects. US studies, focused on the intima media thickness (IMT) of extracranial carotid arteries, have contributed to the establishment of a clear correlation among atherosclerosis risk factors (hypercholesterolemia, hypertension, diabetes, smoking, etc.) and pre-clinical disease. Carotid IMT has been also proposed as a significant predictor of the clinical acute phase of the coronary and cerebral pathology. An observational study, carried out in 963 patients affected by lipid disorder, demonstrated that also the IMT obtained in the normal clinical practice, without the aid of sophisticated measurement techniques, can discriminate patients with and without coronary heart disease and with peripheral artery disease, but not cerebrovascular disease. The carotid IMT, obtained in the normal clinical practice, was associated with age, gender, blood pressure, TC, LDL-C, TG, blood glucose and, inversely, with HDL-C and also linearly related with the number of vascular risk factors. These findings establish the highly predictive value of IMT in the screening of individuals at high risk of vascular disease.

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NON-INVASIVE EVALUATION OF THE ELASTIC PROPERTIES OF THE ARTERIAL WALL

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Compliance is the term used in clinical physiology to describe the elastic behaviour of the arterial wall and is represented by the ratio of volume change per unit of pressure change. Decreased compliance involves increased stiffness, which is the inverse of compliance.

Several different techniques have been used to determine the elastic properties of arteries in humans. They include magnetic resonance imaging, Doppler ultrasound examination, echo-tracking, M-mode echography, and B-mode echography. Several parameters have been proposed as indices of the elastic properties of arteries, including arterial strain, elastic modulus, stiffness. Stiffness has the theoretical and experimental advantage of being influenced less by changes in blood pressure.

We performed ultrasound imaging of the abdominal aorta of 67 hypercholesterolemic children in order to measure arterial diameters and derive calculations of aortic strain, stiffness and elastic modulus. An influence of hypercholesterolemia on age-related modification in the elastic properties of the aorta was demonstrated in our children (Metabolism 1999, 48:55-59). Arterial stiffness may be a useful indicator of early vascular changes occurring in the arterial wall during atherogenesis.

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CARDIAC AND VASCULAR CHANGES IN ESSENTIAL HYPERTENSION.

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Atherosclerosis and hypertension, one of its major risk factors, develop over many years before they are clinically expressed as a cardiovascular complication. Noninvasive exploratory techniques of human arteries make their early detection possible, before they become symptomatic. Three main cardiovascular markers of atheromata can be documented in hypertensive individuals: arterial wall thickening, arterial wall rigidity and left ventricular hypertrophy (LVH). Arterial wall thickening, quantifiable by echography, may take two forms: *diffuse* preintrusive thickening and *focal* intrusive thickening (plaque). Both diffuse and focal intimal-medial thickness of the common carotid and common femoral arteries can be measured automatically by computer-assisted echography. Diffuse intimal-medial thickening does not necessarily mean atherosclerosis and may be compatible with vascular hypertrophy. Diffuse arterial rigidity, due to the sclerotic component of atherosclerosis, is detectable by the measurement of pulse-wave velocity. Diffuse vascular sclerosis might contribute to left ventricular hypertrophy by increasing pulsatile workload, and accelerate the degenerative processes in arterial walls submitted to higher pulsatile cyclic stress. For what concerns cardiac changes, in hypertensive patients, usually LVH and geometric remodelling are found. More precisely concentric LVH, obtained by echocardiographic measures of left ventricular mass and relative wall thickness, is related to a greater pressure load. All these changes and their correlations are not yet completely explained; probably the efforts for studying the early phases of hypertensive disease and the role played by the Renin Angiotensin System could fill this lack in the next future.