The aorta is a capacitance vessel that enables transformation of the “on-off” blood-flow characteristics of the left ventricle into a less pulsatile flow in more distal vessels. Thus, a smooth nonpulsatile blood-flow pattern is achieved at capillary level. Arterial wall stiffness determines the degree of energy absorbed by the elastic aorta and its recoil in diastole. Aortic pulse pressure (PP) in health is physiologically lower than peripheral PP. Reduction of elevated aortic systolic blood pressure (SBP) protects circulation from pressure-induced damage. Maintaining aortic diastolic blood pressure (DBP) ensures adequate coronary perfusion. A stiffer aorta, which can be observed with aging and in different pathological conditions such as hypertension, obesity, diabetes mellitus, and dyslipidemia, fails in both of these tasks. Numerous studies now show that PP, arterial stiffness, and early wave reflections are strong predictors of cardiovascular (CV) risk.1 The CV complications are usually located above the aortic recoil and mainly affect the heart. The downward consequences of enhanced pulsatility, particularly at the peripheral and even the microvascular levels, are less documented. The study by Mitchell et al2 in the June issue of Hypertension addresses this subject, with particular focus on a healthy elderly population.

Central Versus Peripheral Pulse Pressure: Role of Wave Reflections

In addition to arterial stiffness, the pulse waveform characteristics are influenced by wave reflection. Energy propagated through the circulation usually meets vessel branching points, at which some of the antegrade energy is “reflected” and becomes retrograde. At some point through the aorta and its branches, the incident and reflected waves summate. Where and when this happens depends on the speed of energy transfer along the aortic wall (ie, pulse wave velocity [PWV]), on the degree of arterial luminal diameter mismatch (greater mismatch results in greater reflection), and on aortic length (wave reflection is closely correlated with height). In young, healthy, tall subjects, energy summation takes place low in the abdominal aorta, in early diastole, helping to maintain coronary perfusion. A stiffer aorta with greater PWV, aortic branches with reduced lumen diameter, and shorter height cause the reflections to occur in late systole (rather than in diastole, such as in healthy individuals). Thus, with energy summation occurring closer to the aortic valve and coronary sinuses during systole, coronary perfusion is impaired. The sites of vessel branching and the characteristics of resistance vessels are of critical importance for understanding peripheral PP alteration.

The complex network of small arteries and arterioles represents the resistance vasculature. According to Poiseuille’s law, increased vascular resistance may result from reduced lumen diameter of individual vessels, from larger vessel, or from rarefaction (decreased number of vessels connected in parallel).3 Changes in vascular resistance from larger to smaller arteries occur abruptly over a short length of the path between arteries and veins.3 The very high resistance over a short length causes mean blood pressure (MBP) to fall precipitously over this short length. Thus, high resistance reduces both pulsatile phenomena and steady flow. The amplitude of PP falls at the same time as MBP, resulting in a steady flow through resistance vessels. Arterial pulsations that cannot enter high resistance vessels are reflected and summate with pressure waves approaching the area of high resistance. This summarizes the classical model used in the report of Mitchell et al.2 In practice, changes are not identical along the arterial tree and may differ according to age and the vascular bed in question (eg, brain, heart, kidney). In particular, as observed in healthy elderly people, the widening of pulsatility very close to the brain, heart, or kidney may favor end-organ damage.

Cerebral Versus Cardiac End-Organ Damage

The cerebral circulation is normally autoregulated, that is, within wide limits flow is kept constant during perfusion pressure changes. This process is mediated by the caliber of smaller arteries and arterioles, which constrict when BP rises and dilate when BP falls. In chronic hypertension, autoregulation of cerebral blood flow is adapted to high BP and the lower end of the autoregulatory curve is shifted toward higher pressure. This results in part from large arteries that have resistance properties in the cerebral circulation.4 Their constriction increases vascular resistance and attenuates increases in pressure in the smaller downstream vessels; furthermore, the structural component of resistance vessels is increased. Thus, cerebral circulation is largely protected from increased steady BP. In hypertension, stroke prevention is mainly related to the reduction of MBP. There are some exceptions, because the brain stem benefits to a lesser degree from blood flow autoregulation.4 There are also some condi-
tions, such as Alzheimer disease that are selectively affected by PP.

In coronary circulation, autoregulation is present, but with 2 particularities. First, the perfusion pressure is determined not by MBP but by DBP, because of nearly exclusive perfusion during diastole. Second, in subjects with hypertension, atherosclerosis, or both, the coronary reserve is markedly reduced. Thus, the unique hemodynamic factor related to coronary perfusion is aortic DBP, which in turn depends on vascular resistance and the level of arterial stiffness. In hypertensive subjects, drug treatment reduces vascular resistance, thus preventing stroke, but may have little effect on arterial stiffness, which increases spontaneously and independently with age. Thus, with aging, aortic DBP and coronary perfusion are reduced, whereas SBP and PP are increased. For this reason, central PP is superior to brachial PP in the prediction of myocardial infarction. Accordingly, drug treatment of hypertension prevents myocardial infarction, but less successfully than stroke.

**Pulse Pressure and Kidney End-Organ**

In tissues such as the heart and the brain, precapillary arterioles provide \( \approx 75\% \) of the series resistance of the vascular bed, thus dissipating mean and pulsatile energy before reaching the capillary level. For glomerular capillaries, because efferent arteriolar resistance is normally greater than afferent resistance, the pressure drop across afferent glomerular arterioles in the kidney is relatively small. Mean and pulsatile pressures in the glomerulus are relatively high, approximating 60% of the arterial values. This greater level of pressure ensures a high filtration fraction, but exposes the glomerular capillary to potentially damaging PP.

As with the brain and the heart, the kidney normally autoregulates blood flow across a wide range of perfusion pressures. The combination of myogenic tone in the afferent arteriole and tubulo-glomerular feedback mediates the bulk of this autoregulation, which has traditionally been defined in terms of steady pressure. However, recent studies have shown that myogenic tone in the afferent arterioles is affected by PP. Thus, in models with PP out of proportion with mean pressure, as in hypertension in the elderly, renal hemodynamics would be interesting to evaluate. Additionally, Cortes et al have shown that the glomerulus is selectively exposed to pulsatile forces affecting capillary pressure, glomerular basement membrane, and mesangial matrix. This may contribute to glomerulosclerosis, particularly in 2 experimental models: the remnant kidney and experimental insulin-dependent diabetes mellitus.

**Conclusion**

Interactions between macro- and microcirculation may affect pulsatile forces acting on large and small arteries all the way to microvessels, resulting in increased CV risk, particularly in diabetic subjects. On the other hand, in treated hypertensive subjects, the reversibility of arteriolar structural changes may improve wave reflection and reduce central PP. Investigations such as that of Mitchell et al will contribute to the development of strategies that may help to improve the care of elderly hypertensive subjects in the future.

**References**
