best method for analyzing accurately the hemodynamic state of the renal artery in older patients. ^{123}I-OIH has only 2.5% of the equivalent renal radiation dose of ^{131}I-OIH. ^{123}I has beneficial physiologic characteristics inasmuch as it releases no beta rays. The energy and half-life of the gamma rays released by ^{123}I are ideal for use in nuclear medicine.[3] It has been reported that, compared with the dose of ^{131}I-OIH of 18.5MBq, ^{123}I-OIH had a higher dose of 37MBq.[4] Because of the difference in sensitivity of the collimator, renography using ^{123}I-OIH produces a count five to ten times greater than that of ^{131}I-OIH, providing more accurate renographic curves with less analytical variation. Our measurements as determined by Tauxe's and Schlegel's methods were well correlated (Figure 1), showing that renography with ^{123}I-OIH is useful for determining the renal arterial hemodynamic state, even in older patients.

In general, renal function is determined by renal arterial flow and renal excretion, the latter being dependent on glomerular filtration and tubular function. Postulated is the accumulation of Lp(a), with elimination dysfunction as a possible mechanism. However, there is a possibility that Lp(a) is affected by renal artery flow. To explore this possibility, we assessed the relationship between Lp(a) and ERPF in patients with mild renal dysfunction. As shown in Figure 2, there was a negative correlation between Lp(a) and ERPF. Although diminished ERPF is caused by various factors, the most important is renal arteriosclerosis. It is suggested that our patients probably had renal arteriosclerosis because of their age, clinical symptoms, and creatinine clearance. Lp(a) was not correlated with BUN or CRE, which reflect the renal excretion. We, therefore, concluded that Lp(a) is markedly involved in renal arteriosclerosis.

It was generally accepted that blood Lp(a) levels were genetically determined and were not related to sex, age, or dietary habits. This implies that high levels of Lp(a) accelerate arteriosclerosis, including that of the renal artery. Recent reports, however, have shown that Lp(a) is reduced with the administration of drugs such as nicotinates and neomycin, with LDL-apheresis, and with oleic acid diet, indicating that Lp(a) is determined not only by genetic factors but is also affected by environmental factors. It is thus possible that Lp(a) increases with progressive renal arteriosclerosis, or that, conversely, high levels of Lp(a) are the result of renal arteriosclerosis. Although further studies are required to clarify the relationship between increased levels of Lp(a) and renal arteriosclerosis, the results of this study do suggest that a relationship exists between these factors.

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SUPPORTIVE GROUP EXPERIENCE FOR PATIENTS WITH EARLY-STAGE ALZHEIMER'S DISEASE

To the Editor: Dementia is a neurodegenerative syndrome of acquired progressive intellectual impairment that presents one of the greatest challenges to the health care system.[1, 2] Standardizing diagnostic techniques and expanding access to services has been the mobilizing factor of public health concern. Despite the increased focus on diagnosis, research, and clinical drug studies for dementing illnesses, clinicians have paid limited direct attention to either the psychological consequences of the progressively deteriorating level of function in this patient population or its profound impact on their daily activities, role identity, and quality of life.[3]

Individuals with early stage Alzheimer's face solitary challenges peculiar to their level of impairment. Progressive deterioration in role function has an insidious effect on the individual's self-esteem. Individuals often possess relatively intact insight and appreciation of experience, and, as such, are not shielded from the transitional stages of denial, anger, and depression.[4] These individuals frequently feel segregated from their former friends and social activities, leading to feelings of rejection, boredom, and worthlessness. Patients and their caregivers often voice concern over the lack of appropriate resources to assist them in coping with these consuming changes. To date, efforts to understand and treat the psychological impact of neurodegenerative processes have been conspicuously absent.

Historically, older adults have been considered inappropriate candidates for psychotherapeutic interventions.[5] The current psychotherapeutic models, until recently, neglected Alzheimer's disease patients. A search of the literature revealed few references of individual therapy or support groups for early stage Alzheimer's disease patients.[6–8]

Support groups have proven to be a major and invaluable ingredient in the treatment of unimpaired patients and caregivers, as well as for caregivers of individuals with cognitive impairment.[3, 9–12] The reasons for forming an early-stage dementia support group include: (1) to provide a therapeutic intervention for a subset of Alzheimer's patients who, by virtue of the early stage of their losses and relatively intact insight, are particularly vulnerable to states of depression, frustration, and crisis; (2) to identify specific issues unique to this population; (3) to evaluate the influence of a psychotherapeutic group experience on patients' self-esteem, coping skills, and sense of well-being; and (4) to develop practical recommendations from which a model for early-stage Alzheimer's support groups may follow.

This specialized support group could provide a forum for a subset of dementia patients recently diagnosed with early-stage Alzheimer's disease. Participants would be at similar points on the coping-collapse continuum[13] as determined by standard cognitive/mood scales for the subject and caregiver

interview. Common and sensitive group themes might involve individual level of comfort versus discomfort with the diagnosis, how they now perceive themselves, and how others, particularly family members, reacted to their condition; feelings of guilt, anxiety, and hopelessness for a future without executive control; increasing reliance on caregivers for assistance in decision making; and inevitable role reversals occurring with spouses, children, and contemporaries. Life style adjustments would occur for social activities, interpersonal relations, transportation, and employment. Patients and caregivers might make these changes with a sense of urgency and haste as they now carried a diagnosis that was for them unwanted, inescapable, and threatening. These combined factors might serve to explicate the participants' vulnerabilities and bleak future.

Conceivably, early participation by Alzheimer's individuals in a support group might lead to a better understanding and acceptance of the inevitable changes in function, role, and identity. A psychotherapeutic group intervention may have unrecognized potential for alleviating suffering and improving the quality of life for this patient population and their families.

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IMPORTANCE OF AORTIC WAVE REFLECTIONS IN AGE-ASSOCIATED CENTRAL BLOOD PRESSURE CHANGES IN NONHYPERTENSIVE HUMANS

To the Editor: Systolic arterial blood pressure increases gradually with aging,[1,2] which can potentially cause an increase in left ventricular afterload. Previous studies have shown that increased central systolic blood pressure is a risk factor for cardiovascular morbidity in normotensive individuals as well as in hypertensive patients.[3] Several studies have investigated age-related changes in pulse wave velocity, suggesting that the aortic wall becomes stiffer with age.[4] Aortic stiffening causes a widening of the incident wave amplitude because of a reduction in the "Windkessel" effect. Alternatively, aortic stiffening may cause earlier and increased pressure wave reflections by increasing aortic pulse wave velocity. To our knowledge, no previous studies have investigated which component of the central aortic pressure is predominantly influenced by age, resulting in age-associated blood pressure increases. We evaluated the relative contributions of aortic wave reflections to the age-associated changes in central blood pressure and left ventricular remodeling, utilizing the noninvasively determined carotid augmentation index.[2,5]

In 81 nonhypertensive subjects (systolic brachial blood pressure <160 mm Hg and diastolic pressure <95 mm Hg) aged 16 to 90 years, including those who visited the hospital for health screenings and healthy volunteers, we studied the effect of aging on central aortic blood pressure, the augmentation index measured in the conventional carotid pulse wave tracing obtained with an air-filled strain gauge pulse wave transducer (TPW-01B, Toshiba, Tokyo, Japan), and left ventricular mass obtained echocardiographically using Devereux's formula. The accuracy of the pulse wave system has been described previously.[5] Subjects underwent echocardiography to confirm the absence of significant cardiovascular disease. No subjects were taking medications that affect the hemodynamic state. Informed consent was given by all subjects before the study.

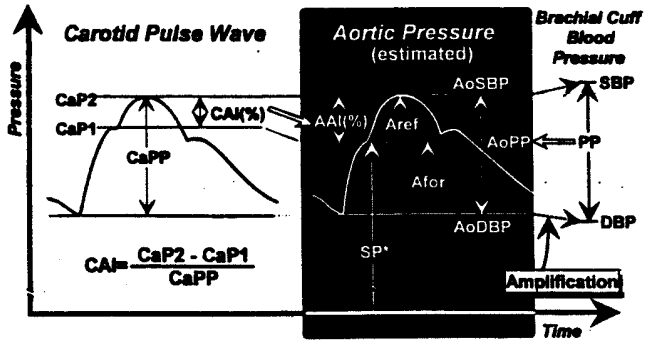

Figure 1. Schematic representation of carotid (CAI) and aortic augmentation indexes (AAI) and components of aortic pressure. Height of the first inflection point of the upstroke or the peak of the incident wave (CaP1) and height of the late systolic peak (CaP2) of the carotid pulse were measured to calculate the CAI. SBP, DBP and PP denote brachial systolic, diastolic and pulse pressures, respectively. Based on published data,[6] central aortic pulse pressure (AoPP) was estimated assuming age-dependent peripheral amplification (Amp%) as: AoPP = PP · 100/(100 + Amp); Amp = (65 − Age) · 8/11. As peripheral and central mean blood pressures (MBP = SBP/3 + 2 · DBP/3) are almost identical,[6] we determined systolic and diastolic aortic pressure estimates (AoSBP and AoDBP) using the following equations: AoSBP = SBP − 2 · (PP − AoPP)/3, AoDBP = DBP + (PP − AoPP)/3. Each component of the aortic pressure was calculated using the AAI in the following manner; A_{ref} = AoPP · AAI/100; A_{for} = AoPP − A_{ref} ($A_{ref} \geqq 0$) or A_{for} = AoPP($A_{ref} < 0$); SP* = AoDBP + A_{for} = AoSBP − A_{ref}($A_{ref} \geqq 0$) or SP* = AoSBP($A_{ref} < 0$).