5. Jacobs AD, Gale RP. Recent advances in the biology and treatment of acute lymphoblastic leukemia in adults. N Engl J Med 1984; 311:1219-31.
6. Priest JR, Robison LL, McKenna RW, et al. Philadelphia chromosome positive childhood acute lymphoblastic leukemia. Blood 1980; 56:15-22.
7. Chessels JM, Janossy G, Lawler SD, Secker Walker LM. The Ph1 chromosome in childhood leukaemia. Br J Haematol 1979; 41:25-41.
8. Catovsky D. Ph1-positive acute leukaemia and chronic granulocytic leukaemia: one or two diseases? Br J Haematol 1979; 42:493-8.

SUCCESSFUL TREATMENT OF A MONOAMINE OXIDASE INHIBITOR–TYRAMINE HYPERTENSIVE EMERGENCY WITH INTRAVENOUS LABETOLOL

To the Editor: Hypertensive crisis is the most serious toxic effect of monoamine oxidase inhibitors and is usually related to accumulation of tyramine and other monoamines in aged foods escaping oxidative deamination, with the subsequent release of supranormal amounts of catecholamines.[1] The usual treatment for this type of hypertensive emergency is administration of a short-acting alpha-adrenergic blocking agent such as intravenous phentolamine. We recently used intravenous labetolol to treat a patient with severe, accelerated hypertension secondary to an interaction of tranylcypromine with monoamines present in aged soy sauce.

A 33-year-old woman presented to our emergency department with global headache and stiffness of the neck approximately 20 minutes after eating chicken teriyaki (containing aged soy sauce). She had been taking tranylcypromine sulfate, 40 mg in four divided doses, and desipramine, 60 mg at bedtime, for five months. Physical examination while she was supine showed a heart rate of 55 beats per minute and blood pressure of 230/140 mm Hg. The remainder of the examination, including neurologic evaluation, was unremarkable. The results of urinalysis and the levels of serum electrolytes, blood urea nitrogen, and creatinine were within normal limits. The patient was given labetolol hydrochloride intravenously (20 mg) over five minutes; a gradual lowering of the blood pressure and reversal of the reflex bradycardia occurred. The blood pressure and heart rate stabilized in 30 minutes, at approximately 140/90 mm Hg and 60 to 65 beats per minute, respectively. The patient was discharged after four hours of observation.

Labetolol is an antihypertensive agent competitively inhibiting both alpha- and beta-adrenergic receptors.[2] Intravenous administration has been shown to be effective in various forms of hypertensive crisis,[3] but to our knowledge it has not been used in accelerated hypertension secondary to the interaction of monoamine oxidase inhibitors and ingestion of tyramine. Because the ratio of alpha-blocking to beta-blocking potency for intravenous labetolol is 1:7,[4] there has been concern regarding the use of this drug in states of catecholamine excess, since potent beta-blocking activity may be associated with increased alpha-mediated vasoconstriction. However, our experience demonstrates the safety and efficacy of labetolol in at least this case of accelerated hypertension mediated primarily by norepinephrine.

Jonathan H. Abrams, M.D.
Peter Schulman, M.D.
William B. White, M.D.
University of Connecticut
Farmington, CT 06032 School of Medicine

1. Marley E, Blackwell B. Interactions of monoamine oxidase inhibitors, amines, and foodstuffs. Adv Pharmacol Chemother 1970; 8:185-239.
2. Richards DA. Pharmacologic effects of labetalol in man. Br J Clin Pharmacol 1976; 3: Suppl 3:721-3.
3. Cressman MD, Vidt DG, Gifford RW, Moore WS, Wilson DJ. Intravenous labetalol in the management of severe hypertension and hypertensive emergencies. Am Heart J 1984; 107:980-5.
4. Farmer JB, Kennedy I, Levy GP, Marshall RJ. Pharmacology of AH5158: a drug which blocks both alpha and beta adrenoreceptors. Br J Pharmacol 1972; 45:660-5.

NATHAN PRITIKIN'S HEART

To the Editor: Nathan Pritikin and the principles he publicized have been of interest to physicians for some time. His work has been discussed in many forums, including the *Journal*. Mr. Pritikin died in February 1985 at the age of 69, and we wish to report the clinical and autopsy findings.

In February 1958, the diagnosis of asymptomatic coronary insufficiency was made during a comprehensive medical evaluation. In a Master's two-step test, while Mr. Pritikin's heart rate was 98 beats per minute (56 per cent of the age-predicted maximum), the electrocardiogram showed a 2-mm horizontal ST-segment depression in leads II, aVF, and V5 and a 1.5-mm ST depression in V3. The diagnosis was confirmed by another Master's test in December 1959.

Mr. Pritikin's fasting serum cholesterol level was 280 mg per deciliter in December 1955, and at that time he started to modify his diet. At the time of diagnosis of coronary insufficiency (1958), he formulated and began to follow the Pritikin high-complex-carbohydrate, low-fat, and low-cholesterol diet. A summary of his serum cholesterol levels at various dates follows: 12/55, 280 mg per deciliter; 2/58, 210; 7/58, 162; 9/58, 122; 8/59, 155; 6/60, 120; 12/63, 102; 3/66, 119; 9/68, 118; 1/69, 112; and 11/84, 94.

Mr. Pritikin led a vigorous life, and until late 1984 he ran several kilometers daily. In 1958 Mr. Pritikin had a malignant lymphoma, most closely resembling well-differentiated lymphocytic lymphoma with macroglobulinemia. Intermittent chemotherapy provided control for several years. In 1980 a splenectomy was performed, and in 1984 and 1985 several experimental agents were tried. Anemia became worse in his last few months, partly because of hemolysis and antibody that precluded successful cross-matching. Mr. Pritikin died, after several complications of therapy, in February 1985.

An autopsy revealed lymphoma in partial remission and several findings referable to treatment. A detailed description of the cardiovascular findings follows.

The heart weighed 380 g. The epicardium was smooth, and no scars were visible. The endocardium and all valves were normal, and serial slices of the atrioventricular conduction tissue showed normal morphology. The coronary arteries were soft and pliable. The right, left, circumflex, and anterior descending arteries were opened by parallel slicing at intervals of 3 to 4 mm. They were widely patent throughout. Some yellow, flat streaks were found, but there were no raised plaques and no compromise of the lumens. No clots were present. The left ventricular myocardium was up to 1.5 cm thick. Histologically, scattered large fibers with large, square nuclei were found. No evidence of infarction or scarring was identified grossly or microscopically.

Several systemic arteries showed some yellow, flat streaks. No elevated plaques were present, and no reduction of the lumen was found. No infarcts of any size, or other findings referable to vascular disease, were present in any organ.

In a man 69 years old, the near absence of atherosclerosis and the complete absence of its effects are remarkable.

We are grateful to Mrs. Nathan Pritikin for permission to report this case.

Jeffrey D. Hubbard, M.D.
Albany, NY 12208 Bender Hygienic Laboratory

Stephen Inkeles, M.D., M.P.H.
Ocean View Medical Group

R. James Barnard, Ph.D.
Santa Monica, CA 90405 Pritikin Research Foundation

DOUBLE PRODUCT OF BLOOD PRESSURE AND HEART RATE AND MAXIMAL LIFE SPAN IN MAMMALS

To the Editor: Fries has suggested that a decline in organ function with increasing age, rather than the cumulative effect of competing causes of mortality, limits the maximal human life span.[1] The function of the heart may be especially critical in this regard. The double product (mean blood pressure × heart rate) is an index of cardiac oxygen consumption. If the heart is a key limiting factor in human longevity even in the absence of specific heart disease, the cumulative lifetime double product may have some maximal value for human hearts, with individual values distributed about a species mean, which in turn is distributed with other specific means about a mammalian mean.