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12. Supported by grants GM 23750 and N01-CP-75876 from the Public Health Service. We thank D. G. Scarpelli and D. S. Longnecker for comments on the manuscript.

5 April 1977; revised 17 May 1977

Hypertension and the Nature of Stress

Friedman and Iwai (*1*) showed that psychic stress can produce some degree of hypertension in rats. This effect was shown in rats that give a strong hypertensive reaction to excessive salt ingestion but not in rats that were not selected for this characteristic. Friedman and Iwai conclude that "there is still insufficient evidence to allow attributing to stress a primary etiological role in essential hypertension."

The failure of Friedman and Iwai to produce hypertension in unselected rats agrees with other studies in which electrical shock or other unpleasant physical stimuli were used as stressors; in these studies it proved difficult to demonstrate marked lasting changes in blood pressure (*2*). Other studies, however, have demonstrated lasting and clear changes in blood pressure after exposure to stress. How do the studies that show such changes differ from those which do not? It appears that the nature of the stressor is important. If the experimental animals are confronted with conspecifics (*3*) or species enemies (*4*) in agonistic encounters, marked and lasting hypertensive reactions can be shown. Such reactions may be highly specific, as suggested by the opposing responses of the iliac artery of a cat confronted with another cat or a dog (*5*). In some species, local vascular responses to agonistic encounters with conspecifics can be extremely strong. Tree shrews, for instance, may die of uremia after repeated exposure to such encounters; the uremia was presumably caused by severe and lasting constriction of the renal arteries (*6*).

Different stressors are probably not equally effective in activating the mechanism underlying hypertension. One dimension along which stressors can differ is in the type and intensity of emotional reactions they produce (*7*). It may be argued that as far as the experimental production of hypertension through psychic stress is concerned, stress imposed by physical means, such as electrical shock, is less effective in eliciting strong appropriate emotional reactions than stress imposed through encounters with species enemies or conspecifics. Thus, in establishing an animal model of hypertension produced by psychic stress it may prove useful to take into account the differential sensitivity of mechanisms underlying hypertension to stressors differing in nature.

MICHAEL PETERS

Department of Psychology,
University of Guelph,
Guelph, Ontario, Canada

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Peters suggests that the nature of the stressor is the crucial variable in the development of stress-induced blood pressure elevations. On the basis of our research, we concur that this is an important factor to consider when assessing the etiological significance of stress in the pathogenesis of hypertension. We had demonstrated that the simple application of electrical shocks or severe food deprivation did not produce hypertension in salt-sensitive rats (*1*). However, placing these rats in a conflict situation in which responses resulted in electrical shocks and food deprivation did elevate the blood pressure. These specially bred rats have exhibited blood pressure sensitivity to a variety of putative hypertensinogenic stimuli. It is now appropriate to list psychological stress, that is, conflict, among them. The elevations in blood pressure we obtained using conflict were much less pronounced and persistent than those obtained using other stimuli (*2*). It is possible that exposing these rats to other types of stress which presumably affect the higher portions of the central nervous system, such as confrontation with conspecifics or species enemies, would result in hypertension as severe as that observed upon exposure to a high-salt diet or renal artery constriction. However, we maintain that until such a demonstration has been reported, there still is insufficient evidence to attribute to stress a primary etiological role in essential hypertension.

RICHARD FRIEDMAN
JUNICHI IWAI

Medical Department,
Brookhaven National Laboratory,
Upton, New York 11973

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11 August 1977