increased, indicating increased sympathetic neuronal activity in blood vessels. Interestingly, DBH activity in the locus coeruleus area of the brain is decreased[3].

In contrast, serum DBH activity and tyrosine hydroxylase (TH) and DBH activities in blood vessels of SHRs at 14 weeks of age do not differ significantly from those of normotensive Wistar–Kyoto rats[4], whereas TH and DBH activities in adrenal glands are increased[4,5]. These results suggest that the increased sympathetic neuronal activity in young SH rats is decreased during the development and establishment of hypertension in adult SHRs and whereas the adrenal medullary activity in adult SHRs is increased.

The results of Grobecker *et al.* on decreased TH, DBH, and phenylethanolamine N-methyltransferase (PNMT) in the adrenal glands of SHRs at 4 weeks of age are of interest and indicate depressed adrenal medullary activity in contrast to increased sympathetic neuronal activity. I have, however, observed a slight but significant increase of DBH activity in the adrenal glands of SHRs at 3 weeks of age[5].

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Can supernovae make white dwarfs?

LEVENTHAL and McCall[1] have presented a model for a type I supernova light curve, in which the two sections of exponential decay of the light curve are explained by the accelerated decay of $^{56}Ni \rightarrow {}^{56}Co \rightarrow {}^{56}Fe$, occurring in a fully convective white dwarf which is formed by the supernova implosion–explosion. While they did give reasons why a type I supernova would be expected to synthesise ^{56}Ni, they failed to discuss whether it would produce a white dwarf, a result which is strongly, though not entirely, unsupported by recent calculations.

The problem centres on whether the ^{12}C-detonation model of a supernova, thought to apply to stars in some subrange of $4–8M_{\odot}$, can leave a white dwarf, or, indeed, any collapsed remnant. It is already clear (ref. 2 and W. D. Arnett, unpublished) that stars more massive than $\sim 8M_{\odot}$ all undergo core collapse and produce neutron stars or black holes.

Buchler[3] has assumed various arbitrary central densities, higher than those now thought to be attainable, for half ^{12}C, half ^{16}O, $1.4M_{\odot}$ degenerate cores and calculated the results for carbon detonations at these densities. At central density $\rho_c = 8.6\times10^9$ g cm^{-3}, the star was completely disrupted. In a small range around $\rho_c = 1.6\times10^{10}$ g cm^{-3}, his calculation did in fact produce an extended $1M_{\odot}$ Ni–Fe white dwarf, with the remaining $0.4M_{\odot}$ being blown off. At $\rho_c = 2\times10^{10}$ g cm^{-3} a neutron star remnant was produced.

Couch and Arnett[4] have recently carried out further calculations, using the ^{12}C detonation model and including all effects yet thought of, such as a convective Urca shell, which might postpone carbon ignition and allow the high central densities necessary for collapsed remnant formation to be achieved. Their models disrupted completely, except for the case where the C–O core was initially only 10% C by mass. The carbon burned non-explosively and a remnant could possibly be formed unless the central density reached a critical density (between 1×10^{10} and 2×10^{10} gm cm^{-3}), where the oxygen would ignite. They point out however that if He burning had produced such a low C–O ratio, it might become impossible to understand the nucleosynthesis of C and O, which is, presumably, done in higher mass stars, which are able to preserve and eject these nuclei.

Thus the formation of white dwarfs by supernovae remains highly problematical. If it could be shown to occur, Leventhal and McCall's model of type I supernova light curves would gain much credibility. It could also frustrate attempts (ref. 5 and B. M. Tinsley, unpublished) to deduce the mass below which, stars are able to shed enough mass by the planetary nebula mechanism to become stable white dwarfs, by counting white dwarfs in open clusters with high-mass turn off points on the main sequence.

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Carotid body chemoreceptors

THE "New theory for receptor mechanism of carotid body chemoreceptors" proposed by Osborne and Butler[1] requires that the rate of dopamine secretion from the glomus cells be high when blood gas tensions are normal and reduced in hypoxic conditions. The results of new experimental studies[2,3] are incompatible with the 'new theory' because the relationship between secretion of carotid body catecholamines and blood oxygen was found to be opposite to that postulated. In one study[2], the formaldehyde-induced fluorescence of the glomus cells was measured to monitor changes in their catecholamine content. In untreated cats, hypoxia resulted in higher fluorescence intensity of type I cells from innervated carotid bodies than from denervated ones. When cats were treated with a decarboxylase inhibitor, intensity of fluorescence was decreased in innervated carotid bodies compared with denervated organs after hypoxia. Sampson *et al.*[2] concluded that an increase in centrifugal impulse activity in the sinus nerve induced by hypoxia enhanced both release and synthesis of catecholamines in the carotid body. In the second study[3], the content of noradrenaline plus adrenaline in the cat carotid body was measured by the trihydroxy indole method. The content of these amines in the carotid body decreased after hypoxia and increased above the air control level when inspired oxygen concentration was raised to 40%. The decrease in catecholamine content was greatly attenuated when the carotid sinus nerve was cut before hypoxia.

Since the fluorescence technique used measured dopamine as well as noradrenaline and adrenaline, the increase in fluorescence in untreated carotid bodies after hypoxia may be attributed to enhanced synthesis of dopamine, which serves as precursor for noradrenaline and adrenaline. This is the case in the adrenal medulla where neurogenic stimulation activates tyrosine hydroxylase and increases dopamine content while releasing noradrenaline and adrenaline[4]. The results of the two carotid body studies[2,3] are consistent with the idea that stimulation of efferent neural pathways to the carotid body during hypoxia enhances both synthesis and release of catecholamines. The data provide no support for the 'new theory'[1] which requires that hypoxia depress secretion from the glomus cells of the carotid body and postulates that secretion is regulated independently of sinus nerve efferent pathways.