We have repeated the experiments using a fluorescent spectrofluorometer which permits use of narrow spectral bands and methods of proved specificity for norepinephrine[5] and serotonin[6]. The administration of 0·1 mgm. of reserpine per kgm. subcutaneously to female guinea pigs weighing about 300 gm. at no time caused a rise in brain or heart norepinephrine. In 20 min., when Sheppard and Zimmerman had found a 74 per cent rise in brain norepinephrine, we found that serotonin and norepinephrine showed no change from normal, or perhaps a small decline. After 4 hr. the brain amine-levels were 60–70 per cent of normal (Table 1).

Table 1. GUINEA PIG BRAIN AND HEART AMINES AFTER ADMINISTRATION OF 0·1 MGM./KGM. OF RESERPINE SUBCUTANEOUSLY

		γ/gm.* ± S.D.	Percentage of normal
Brain serotonin	Control	0·28 ± 0·04	100
	Reserpine, 20 min.	0·28 ± 0·02	100
	Reserpine, 4 hr.	0·19 ± 0·04	68
Brain norepinephrine	Control	0·42 ± 0·04	100
	Reserpine, 20 min.	0·40 ± 0·02	95
	Reserpine, 4 hr.	0·25 ± 0·07	60
Heart norepinephrine	Control	2·71 ± 0·34	100
	Reserpine, 20 min.	2·46 ± 0·38	91
	Reserpine, 4 hr.	0·63 ± 0·30	23

* Each value represents the average from 6 animals.

Heart norepinephrine fell to 90 per cent of normal in 20 min. and was only 20 per cent of normal in 4 hr. (Table 1).

These results show that the fall in brain and heart amines after the administration of small doses of reserpine to female adult guinea pigs is qualitatively similar to that observed in other animal species when using larger doses of reserpine.

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THE preceding communication contains data refuting the rise in catecholamines which we had observed 20 min. after the subcutaneous injection of reserpine (0·1 mgm./kgm.). In repeating the experiment in order to check the questioned specificity of the analytical method, we found that we were unable to reproduce our original findings. Instead, the brain catecholamine-levels expressed as percentage of controls were found to be 105, 100, 64, 26, 37 and 31 per cent after ¼, 2, 4, 6, 8 and 72 hr. The shape of the curve was very much like that observed for serotonin and the 4-hr. values agreed with those reported by Orlans and Brodie. We are, at present, unable to detect the factors in the original experiments which gave us the values we reported, but we intend to pursue this matter further. In spite of this discrepancy, the serotonin values remain undisputed and the main conclusion remains valid, namely, that there exists no correlation between the levels of brain amines and the intensity of observable effects.

It is unfortunate that Orlans and Brodie, while correctly questioning the method of analysis for catecholamines, misinterpreted and mis-stated what was written in our original letter. Suffice it to say that our communication was not a criticism of the many skilled workers who had demonstrated the ability of reserpine to lower tissue amine-levels. Contained in it, however, was a criticism of the attempts by some workers to attribute the presumed central effects of large doses of reserpine (1–5 mgm./kgm.) to changes in the levels of brain serotonin or norepinephrine. The actions of reserpine described by Brodie *et al.*[1] as "loss of righting reflex, enhanced salivation and active closure of the eyelids" would be considered as undesirable toxic side-effects if found to occur clinically. To imply that such effects describe in equal fashion such terms as tranquillizing, sedative and central action of reserpine does little to clarify a terminology which is already too vague for scientific usage. It is important to define terms more clearly and to signify what responses one is trying to correlate with changes in tissue amine-levels. It remains to be determined which, if any, of the effects of reserpine may be attributed to changes in serotonin or norepinephrine-levels alone. Previous work with reserpine labelled with carbon-14 or tritium[2] had demonstrated that the alkaloid was present in the brain during the entire period of visible action of the drug, but that here too no correlation could be made between the intensity of response and drug-level. It is important to recognize that the application of other techniques as used in the study of behaviour may bring to light some actions of the drug which are not readily observed, and for which a correlation can be found with changes in brain amine and/or reserpine-levels. In our opinion, no experiment has been reported which conclusively eliminates reserpine, *per se*, as the active agent and establishes serotonin and/or norepinephrine in its place. It remains for future work to establish the true mechanism of action of reserpine and similar alkaloids.

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HÆMATOLOGY

β-Globulin Polymorphism in Chimpanzees

INTRASPECIFIC variation in the electrophoretic mobility of serum iron-binding β-globulins ('transferrins') have been described in man[1,2], cattle[3–5], sheep[6,7], goats[8] and certain monkeys[4,9]. In all but monkeys these variations have been established as inherited.

We have recently examined the sera of 25 chimpanzees, *Pan satyrus*, and observed seven β-globulin phenotypes. Prior to our examination, these animals had been used for the assay of poliomyelitis vaccine. Fifteen were affected with spontaneously acquired tuberculosis. Serum was held 2–20 weeks at − 15° C. before use. Electrophoresis on borate-