lesioned ($\bar{X}=67.6\%$ of load excreted, s.d. $\pm 23.3\%$) and normal ($\bar{X}=78.4\%$, s.d. $\pm 4.0\%$) rats' urine volume. Raphé-lesioned rats were thus not noticeably deficient in retaining a water load following water deprivation.

To test further the possibility of exaggerated drinking due to polyuria, the water intake of four additional lesioned rats and four normal animals was observed after kidney removal. Following the first 24 h of elevated drinking (days 4–7 after surgery), lesioned rats were anaesthetized with ether and their kidneys removed bilaterally. Normal rats were treated in the same way. All nephrectomized animals were returned to home cages to eat and drink freely for 24 h. While both groups drank reliably less ($P<0.01$) than before surgery, lesioned rats continued to drink significantly more ($P<0.025$) than controls (6.8 ml./100 g body weight compared with 2.2 ml./100 g body weight, respectively). Neither group ate an appreciable amount of food (1–2 g at most). Lesioned rats were thus still hyperdipsic relative to normal rats when urine excretion was no longer possible. Linked with the lack of differences in volume of urine excreted following water loading, these findings imply that the hyperdipsia which follows disruption of serotoninergic neurones by raphé lesions is a consequence of altered central thirst mechanisms. Examination of histological materials revealed 1.0–1.5 mm diameter lesions centred in both dorsal and median raphé nuclei. In the same animals, forebrain serotonin and 5-HIAA were lowered to 26.7% and 24.2%, respectively, of control levels 32 days after the lesion ($P<0.001$ compared with control levels in both cases).

At present, the neural mechanisms responsible for this hyperdipsia, as well as its transient nature, are not known. Since similar brain lesions lower serotonin but not noradrenaline[3], it seems unlikely that this drinking is related to changes in this catecholamine. No data are available regarding the effects of raphe lesions on levels of brain acetylcholine, an amine strongly implicated as a neurochemical mediator of thirst[4]. It also remains to be determined whether alterations in serotonin are directly or indirectly associated with the observed hyperdipsia.

Apart from providing preliminary evidence for involvement of brain serotonin in water intake, this report demonstrates the efficacy of depleting serotonin by raphé lesions as a tool for behavioural study. In the past, almost all biochemical and psychological research examining long term effects of serotonin depletion has used the tryptophan-hydroxylase inhibitor, *p*-chlorophenylalanine[5]. Although it is effective in reducing brain serotonin stores, this drug also reduces peripheral serotonin[5] as well as brain noradrenaline[6]. Raphé lesions circumvent these drawbacks.

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Note added in proof. While this article was in the press, a paper by Lorens, Sorensen and Yunger appeared (*J. Comp. Physiol. Psychol.*, 77, 48; 1971) corroborating the present report of transient hyperdipsia following dorsal and median raphé lesions.

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Errata

THE title of the article by Jack Schubert and Edward B. Sanders (*Nature New Biology*, **233**, 199; 1971) should have been "α,β-Unsaturated Carbonyl Sugars as the Cytotoxic Radiolysis Products of Irradiated Carbohydrates". Fig. 6 was inadvertently omitted from the article, and is reproduced below.

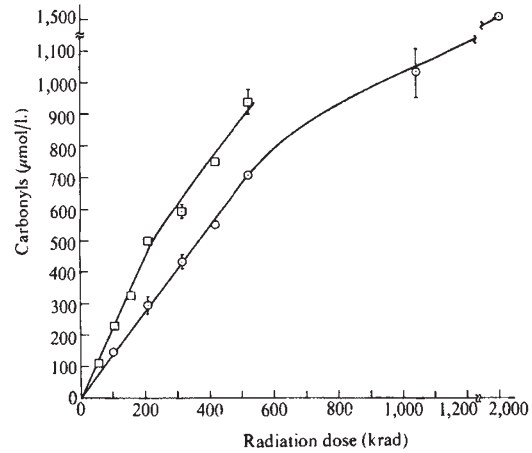

Fig. 6 The carbonyl yield in gamma irradiated unbuffered sucrose (0.058 M) at 25° C as a function of radiation dose. The average deviations are indicated for each point unless they were less than the width of a given point. The molar absorptivity of the 2,4-DNP anions of the carbonyls was assumed to be 18,000. □, Solutions saturated with N_2O; ○, solutions O_2-free.

IN Table 1 of the article "Amino-acid Sequence of Human Placental Lactogen" by Louis M. Sherwood, Stuart Handwerger, William D. McLaurin and Michael Lanner (*Nature New Biology*, **233**, 59; 1971), Arg was inadvertently omitted from position 132 in the HPL sequence.

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