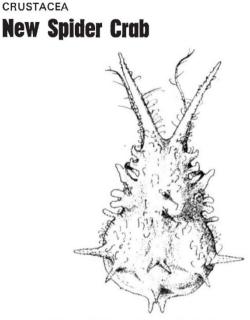
body was male. All of these visual mutants have been found to be autonomous, that is, the mutant eye always functions as mutant tissue, independent of the amount of normal tissue present elsewhere. Thus, at least in this series of mutants, the primary causes of the abnormality in the mutant eyes are within the eye and not elsewhere in the nervous system or brain.

Hotta and Benzer (*Proc. US Nat. Acad. Sci.*, **67**, 1156; 1970) selected mutants of *Drosophila*, carrying the required X-linked recessive mutation plus marker genes, and they crossed these flies with females of a strain that often loses the X chromosome spontaneously during mitosis. About 10 per cent of the female progeny were found to be mosaics. By counter current distribution, the mutant males were selected to be negatively phototactic and gynandromorphs were selected which had one eye of the normal genotype (that is, positive phototaxis) and the other eye of the mutant type (deficient visual function).

Several of the gynandromorphs were tested for their phototactic response. All the composite flies expressed the visual deficit of the mutant, and parabiosis with a normal half of a fly did not remove the deficit. The response to a short flash of light was measured in these gynandromorphs with the aid of electroretinograms (ERG). The ERG can be tested separately in each eye of a mutant fly. ERGs obtained from the female eye, in which the normal allele is present, were the same as for a normal fly. The ERG recorded from the male eye, in which the recessive mutation was uncovered, was defective in the same way as in a non-mosaic fly. As before, the normal half in parabiosis with the mutant half did not compensate for the defect in the mutant. For flies with heads which had normal and mutant portions, the same result was obtained in every Thus, these mutations are clearly autonomous case and res de within, or close to, the eve. In six eves in which the line of mosaic cells occurred within the eye, there seemed to be a high degree of autonomy between those segments of the eye that were normal and those that were deficient. Whatever the genetic defect in these flies, it is clearly expressed only in the mutant parts.

Similar results have been reported by Ikeda and Kaplan (Proc. US Nat. Acad. Sci., 67, 1480; 1970), who have studied the behavioural phenotype caused by a sex-linked, single gene mutation in gynandromorphs of Drosophila. The gene, hyperkinetic, controls a leg-shaking action during the administration of ether anaesthesia. In their studies the male tissue of the gynandromorph was hemizygous for hyperkinetic whereas the female tissue was heterozygous. The characteristic neural activity caused by the hyperkinetic gene can be recorded only in the presumptive male region of the thoracic ganglion. Motor regions of the male side were found to function independently of their counterparts on the female side of the fly and they were unaffected by the genotype of other parts of the body. Thus, expression of the hyperkinetic gene is genetically autonomous, and each side of the ganglion is independent of the other. There is also a high correlation between the external genotype and the genotype of underlying neural tissue of the thorax, but the correlation between external and internal genotype is not absolute.

Although these studies are incomplete, a start has been made in the application of the techniques of bacterial genetics to the problems of understanding the role of the brain and nervous system in higher organisms. It will be interesting to observe the results of future studies using mutants with a lesion which can be mapped at a site distant from the sensory organ.



Among the benthic invertebrates dredged up some time ago off Rottnest Island, Western Australia, was this strange looking animal which D. J. G. Griffin now describes as a new species of spider crab, *Chlorinoides occidentalis (Records of the Australian Museum*, **28**, 65; 1970). The illustration shows the carapace and rostrum of the male holotype which is 17.1 mm long.

## TRYPANOSOMIASIS No Room for Complacency

## from a Correspondent

HUMAN trypanosomiasis has been, statistically speaking, a scarce disease in Africa for some years, giving the general impression that it is fully under control. But this is not so; there are probably a million cases of sleeping sickness in the Congo (Kinshasa) at the present time. This gave added interest to the research reported at the annual seminar of the Trypanosomiasis Panel of the Overseas Development Administration, held in London on November 12.

Although contributions covered the whole range of current research on trypanosomes and their tsetse fly vectors, two new developments are particularly worth noting. The first, reported in a series of papers by Dr B. A. Newton, Mr J. K. Burnett and Dr G. A. M. Cross (Molteno Institute, Cambridge), concerns the differing, but constant, DNA from different strains of the same species of trypanosome. Particular attention has been paid to the DNA of the *T. brucei* group, which contains the species causing sleeping sickness, for it may provide information on differential resistance, or sensitivity, to trypanocide drugs.

The approach being considered is that of comparing the metabolism of normal and mutant bloodstream and culture forms, and observing the sequence of metabolic changes during transformation from bloodstream to culture forms. It has been found that different