GRAVITATION

Waves from Black Holes

by our Cosmology Correspondent

ACCORDING to the conventional understanding of relativity, collapsing black holes should produce gravitational radiation in the form of pulses, like those detected by Weber. But under some circumstances it seems that a black hole can produce long trains of gravitational waves, and it may be that the bursts of radiation detected by Weber arise from this process. The important feature of a disturbance which will produce such trains of radiation is that it perturbs a black hole from its preferred state of spherical symmetry (W. H. Press, *Astrophys. J. Lett.*, **170**, L105; 1971). It is no surprise that such a perturbed black hole will radiate gravitational waves as it restores sphericity; but it is surprising to learn that this radiation may be emitted in a long train of nearly sinusoidal waves, rather than in a single intense burst.

Following calculations of perturbations of integer-spin zero rest mass fields by R. H. Price, Press has built on earlier work of Regge and Wheeler (*Phys. Rev.*, **108**, 1063; 1957) and of Zerilli (*Phys. Rev.*, **D2**, 2141; 1970) which describes the dynamics of the process for the odd parity and even parity cases. It seems that a crude physical picture of the situation can be gained by imagining the black hole to vibrate around spherical symmetry in a quasi-normal mode which is slowly damped by gravitational radiation.

But what excites these oscillations? Somehow, the black hole must be struck, to set it ringing like a bell. There is reason to believe that a test particle falling into a black hole can excite vibrational modes (Davis *et al.*, *Phys. Rev. Lett.*, **27**, 1466; 1971); in order to produce the long wave trains predicted by Press disturbances with a high spherical harmonic index (multipolarity $\gg 1$) must be excited. This raises some difficulties with the excitation mechanism, but in real systems quite complex vibrations might be caused when the hole is accreting from, say, a surrounding ring of matter.

The most intriguing feature of this work is the discovery that black holes might be able to interact with the rest of the universe in a quite complex way. If wave trains of gravitational radiation are discovered, or if the Weber pulses turn out to have detailed structure of the kind postulated by Press, there is, in principle at least, a tool available for studying the properties of black holes such as their rotation which will surely affect the radiation. And because high multipole vibration can produce relatively short-wavelength radiation even from massive objects, this mechanism sets no upper limit on the mass of a black hole which might produce waves suitable for detection by Weber's apparatus. This could explain the apparently excessive number of gravitational wave events reported by Weber in recent years, which has seemed too large to account for simply in terms of the collapse of supernova remnants to form black holes.

LIPID MEMBRANES

Carriers and Pores

from our Membrane Correspondent

BY incorporating various antibiotics, bilayer lipid membranes (BLM) can be made to mimic some of the ion permeability properties of natural membranes and this model system has been extensively studied in recent years. There are essentially two possible mechanisms by which the antibiotics can transport ions across these membranes—either by acting as mobile carriers which diffuse back and forth across the membrane or by forming pores which offer selective pathways for ion movement. Which of these two mechanisms is the one which is operative has been the subject of some controversy but the majority view seems to be that macrocyclic antibiotics of relatively small size, such as nonactin and valinomycin, act as carriers, whereas more complex antibiotics such as gramicidin A and alamethicin form pores. This distinction has recently been put on a firmer footing by the work of Krasne, Eisenman and Szabo (*Science*, **174**, 412 ; 1971).

Krasne *et al.* have examined the effect of nonactin, valinomycin and gramicidin on BLM above and below the thermal transition temperature of the lipid constituting the membranes. This temperature is that at which, on cooling, the state of the long hydrocarbon chains of the lipid molecules changes from a relatively fluid state to a fairly rigid array similar to that in solid hydrocarbons. Krasne *et al.* found that the permeability effects (as judged by membrane conductance) of nonactin and valinomycin were abolished by cooling the membrane through its transition temperature whereas the effect of gramicidin was only slightly reduced. These antibiotic molecules are too large to fit into the interstices between the lipid hydrocarbon chains and so in order for them to act as mobile carriers it is necessary for the membrane to be in its fluid state.

Doing without Tyrosine Residues

IN next Wednesday's *Nature New Biology* (January 19), Hayashi and Stamatayonnopoulos present an examination of the properties of two abnormal human haemoglobins, which correlate satisfyingly with Perutz's model for the oxygenation mechanism. The two mutant forms, haemoglobins Rainer and Bethesda, are of particular interest in being the only known variants with substitutions for the important tyrosine residues next to the termini of the β-chains. Perutz has shown that in normal haemoglobin, in the deoxygenated state, these side phenolic groups lie in pockets, making hydrogen bonds with backbone carbonyls. On oxygenation, the tyrosine side chains are displaced.

Just like normal haemoglobin deprived of these tyrosines by carboxypeptidase treatment, haemoglobins Rainer and Bethesda show anomalously high oxygen affinity and only vestigial haem–haem interactions, as well as a reduced Bohr effect. The natural cofactor, diphosphoglycerate, is also shown to have little effect on the oxygenation equilibrium of the variants. Moreover, in haemoglobin Bethesda, which (unlike haemoglobin Rainer) possesses the normal complement of sulphydryl groups, the chemical reactivity of the latter is quite different from those of normal haemoglobin; not only do they react unusually rapidly in the oxygenated form, but, by contrast with normal haemoglobin, the sulphydryl groups of which become very unreactive on deoxygenation, those of haemoglobin Bethesda actually react much more rapidly after deoxygenation.

All these observations are readily explicable in terms of Perutz's scheme. There seems to be no question of any inability to enter the characteristic deoxyhaemoglobin conformation. Indeed, Greer and Perutz have shown that deoxyhaemoglobin Rainer, as well as carboxypeptidase treated normal deoxyhaemoglobin, are both in that state in the crystal. Rather it seems that the free energy difference between the deoxyhaemoglobin and oxyhaemoglobin conformations is greatly lowered, so that in the deoxygenated state a measurable part of the population has the oxyhaemoglobin conformation. This is a consequence of the absence of the stabilizing contribution of the tyrosines in the β-chain pockets.

Evidently the same tyrosines are also needed to create the cofactor binding site. In the absence of the vital tyrosines, a salt bridge, involving the adjoining histidine, is not made, and this interaction was postulated by Perutz to account for a large part of the Bohr effect. Furthermore, the salt bridge in deoxyhaemoglobin physically occludes a sulphydryl group in each β-chain, so that the much higher thiol reactivity in haemoglobin Bethesda is also accounted for.