OBITUARY

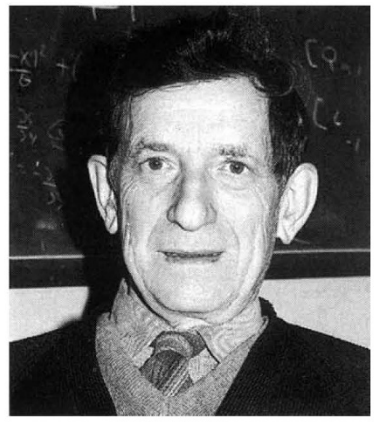
David Bohm (1917–1992)

THE death of David Bohm on 27 October is a sad loss to the community of physicists, and particularly to those concerned with fundamental issues in quantum mechanics.

In straight physics, Bohm's seminal contribution was made in conjunction with his student Yakir Aharanov: the discovery in 1959 of what is now known as the Bohm–Aharanov effect. This predicted the effect of a flux of magnetic induction confined to the interior of a long solenoid on the relative phases of electron waves passing either side of the solenoid, in regions of space where the magnetic induction was zero. This was the first demonstration of how a nontrivial topological feature of the vacuum (in this case identified with the absence of magnetic induction) could produce striking physical effects. Taken together with earlier work on plasma oscillations and the behaviour of electrons in metals, this assured Bohm's reputation as a master of insightful physics in the mainstream tradition.

But in the longer term his exhaustive investigations on unorthodox interpretations of quantum mechanics may be seen as an even more significant contribution. Dissatisfied with the orthodox Copenhagen interpretation, which he had beautifully expounded in his text book *Quantum Theory* in 1951, he published a preliminary investigation of what came to be known as the causal interpretation in 1952. This was a hidden-variables approach, in which the exact location of the particle played the role of the hidden variable and the standard quantum-mechanical predictions were deduced by including an extra term, the 'quantum potential' in the classical Hamilton–Jacobi equation. Effectively, the theory involved waves *and* particles instead of the orthodox waves *or* particles (an idea which actually goes back to de Broglie in the 1920s).

The remarkable feature of Bohm's interpretation was that it apparently circumvented the famous proof given by



von Neumann in 1932 that hidden-variable interpretations of quantum mechanics were mathematically impossible. The situation here was finally clarified in the 1960s by the late John Bell, who showed that von Neumann's proof involved overly strong and physically unmotivated assumptions, which Bohm's theory naturally denied, but the price to be paid for the otherwise appealing hidden-variables approach was a feature of instantaneous nonlocal action mediated by the quantum potential. In a deep sense this violates the spirit of relativity theory, although Bohm was able to show that at the level of statistical observation, all the predictions of relativistic quantum mechanics could be accommodated.

What was the reaction to the causal interpretation? On the one hand Bohm was attacked for being reactionary, of trying to understand quantum mechanics as a sort of glorified statistical mechanics; but on the other hand he was also attacked for his more extreme philosophical views which he had developed in parallel with his causal interpretation. These views distinguished an explicate order, as he called it, of apparently independent entities, from an underlying implicate order involving a view of ultimate reality in which everything was bound up holistically with everything else. His critics regarded this, quite unfairly, as akin to oriental mysticism. However, during the past few years the causal interpretation has been gaining increasing 'respectability' with physicists, not least perhaps because it was enthusiastically championed by Bell, a great admirer of David Bohm.

At the time of his death Bohm was in the process of completing, with his main collaborator Basil Hiley, a definitive presentation of his approach, and this, when hopefully it is published, will be the monument by which Bohm's ideas must ultimately be judged. That I am sure is how he would have wanted it himself. M. L. G. Redhead

coupled metabotropic glutamate receptor[7] acts through a G protein to activate phospholipase C and generate the two second messengers inositol trisphosphate and diacylglycerol (which in turn activates protein kinase C). Herrero *et al.* show that agonists for the receptor mimic the action of phorbol ester in enhancing the 4-aminopyridine-induced release of glutamate, although the intervening stages in the protein kinase C activation are not yet defined.

The presence of a presynaptic autoreceptor (one that responds to the transmitter released by the terminal) that stimulates rather than inhibits release is unusual. Most transmitters are subject to a negative-feedback control by autoreceptors that prevent further release by activating K^+ channels and/or inhibiting Ca^{2+} channels. But such a mechanism acting at the glutamatergic terminal would effectively prevent the terminal from responding to a high-frequency stimulus — the condition necessary for the induction of long-term potentiation (LTP), the most investigated form of synaptic plasticity[8]. On the other hand, a permanently engaged positive-feedback regulator could lead to the glutamatergic synapse running out of control. It is here that the second aspect of the work of Herrero *et al.* becomes relevant. They show that the metabotropic glutamate receptor seems to be coupled to K^+-channel inhibition only in the presence of arachidonic acid. Arachidonic acid can be produced in neurons by phospholipase A_2 activated by Ca^{2+} entering through the NMDA (*N*-methyl-D-aspartate)-selective glutamate receptor[9]. The idea that arachidonic acid acts as a retrograde messenger, whereby it is generated postsynaptically and then diffuses across the synapse to increase glutamate release[1], is based on the integral part played by the NMDA receptor in the establishment of LTP. But until now we have had no convincing explanation of how this messenger function might be executed.

Although Herrero and colleagues are not directly concerned here with synaptic plasticity, their report does provide groundwork on which constructive speculation can be based (see figure). Regardless of whether the ultimate expression of plasticity is presynaptic or postsynaptic, if synaptic plasticity is all-or-none at a given synapse then it is important that once a synapse becomes committed to potentiation, for example by NMDA-receptor activation, the release of glutamate should remain maximal for the period needed to establish plasticity. Arachidonate could assist this process by recoupling the positive-feedback pathway found in the presynaptic terminal. □

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