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The mechanism of potassium dispersal in brain tissue

By A. R. Gardner-Medwin, Josephine L. Gibson and D. J. Willshaw*. *Department of Physiology, University College London, London WC1E 6BT*

When a local build-up of extracellular potassium occurs in neural tissue, depolarization of nerve and glial cells may cause currents to flow through the cells in such a manner as will assist the dispersal of potassium (the spatial buffer mechanism: Kuffler, Nicholls & Orkand, 1966). The fact that such regions commonly develop an extracellular negativity with respect to the rest of the tissue (see Somjen, 1979, for review) supports this idea, but it has never been clear whether the contribution

* Present address: National Institute for Medical Research, London NW7 1AA.

made to K^+ dispersal is significant compared with extracellular diffusion. In the present study the extracellular negativity is examined quantitatively both in isolated pieces of frog brain *in vitro* and in the neocortex and cerebellum of anaesthetized rats. The results suggest that the spatial buffer mechanism is in some circumstances the principal means for K^+ dispersal.

A closed chamber on the brain surface (0·5–3 mm diameter) is perfused with artificial cerebrospinal fluid so that the surface K^+ concentration can be changed rapidly. The resulting changes of potential are measured with bridge electrodes between the interior of the chamber and the bath or pool of fluid (of fixed composition) outside the chamber. Raising the K^+ concentration makes the chamber negative, by approximately 0·5 mV/mM for small concentration changes. The initial rise is more rapid in the frog (2–3 sec for 50 % rise) than in the rat (20–40 sec). The potential changes in the *in vitro* preparation are reduced to 10 % by formalin fixation but are little affected by ouabain (10^{-4} M). The size and time course of the effects of changes from base-line levels (2·5–3 mM) to values over the range 0–20 mM can be fitted by simultaneous numerical solutions of the diffusion and cable equations implicit in the spatial buffer hypothesis. The data requires parameters such that over distances greater than a certain space constant (*ca.* 200 μm (rat) or 30 μm (frog)) about 80 % of the K^+ moving through the tissue is moving through cells rather than through extracellular space. This is a similar conclusion to that previously reached (in experiments on rats) for a flux of K^+ caused by current through the tissue rather than by a concentration gradient (Gardner-Medwin, 1977, 1978; Gardner-Medwin & Nicholson, 1978). Whether or not the underlying mechanism is essential for normal neural functioning, it probably helps to reduce the build-up of K^+ round active neural tissue until such time as it is restored by active transport to its cells of origin.

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