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Title : ROLE OF GLUCOSE-OR POTASSIUM-LACK IN NERVE BLOCK

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Introduction. Nerve blocks are generally performed with isotonic solutions containing sodium chloride but lacking other important physiological solutes such as potassium and glucose. The effect of excess - but not of deficiency - of these substances in local anesthetic solutions is well known^(1,2). This study reports on the ability of mammalian nerve to withstand glucose and potassium deprivation, and the implications for clinical block.

Methods. Vagus nerve of rabbit was incubated at 37.5 - 38°C in 24 mM NaHCO₃ - Ringer's solution at pH 7.4, with 5% CO₂ in oxygen, while resting on stimulating and recording electrodes. The time course of changes in C-fiber action potential was recorded photographically by raising the electrode array out of the solution and stimulating supra-maximally with 0.3 msec pulses at 1 Hz for 4 sec every 5 or 10 min. At the end of an experiment the nerve, which comprises only one fasciculus, was desheathed and the residual K⁺ and Na⁺ content of the core determined by flame photometry. The experimental modifications of the solution consisted of omitting or including 4 mM potassium chloride, and of omitting or including 5 mM glucose (5 different nerves with each of the modifications). Sodium chloride content was adjusted to preserve isotonicity.

Results. When glucose was absent from the medium conduction always failed; but it failed more rapidly ($p < 0.01$) when external potassium was present (Table 1, row B) than when it was absent (row A), even though in the latter case there was a much greater loss of potassium from the core. Inexcitable nerve in glucose-free bicarbonate - Ringer required the addition of 2 mM glucose to the medium for partial recovery, and 5 - 6.2 mM for complete recovery ($n = 2$).

When 5 mM glucose was present in the medium conduction remained normal for at least 2 hours, regardless of whether there was potassium in the bath or not; however, when potassium was present, the core K⁺ content was unchanged (row D), whereas when external potassium was absent (row C), the core K⁺ content decreased significantly ($p < 0.05$). Na⁺ changes were not significant, except as shown.

Discussion. In this system the nerve deprived of circulation in vitro approximated a condition produced in vivo with extravascular vasoconstrictor. Maintenance of normal neural excitability with glucose (Table 1, rows C and D) did not of itself ensure normal conservation of potassium by the fasciculus. Loss of potassium occurred when the K⁺ diffusion gradient was large (row C). This suggests that in the presence of a large K⁺ diffusion gradient the plasma membrane pumps in the core were unable to retrieve all the potassium that leaked outward through the membrane. At least part of the potassium that leaked out into interstitial fluid went on to leak through the perineurium (rows A and C). The magnitude of the loss due to the large K⁺ diffusion gradient can be estimated from the differences in the

K⁺ losses in rows A and B. In both A and B glucose was lacking and pumps were incapacitated, but in row B there was a normal K⁺ diffusion gradient to the exterior and the loss was due to non-pumping, whereas in row A there was in addition an abnormally large K⁺ diffusion gradient. The excess loss of K⁺ in A as compared with B was therefore diffusional. Yet in row C, where there was no lack of glucose, the K⁺ loss was almost as large as in row A. Assuming approximately similar diffusional losses occur in rows A and C, one is led to suspect that, notwithstanding the availability of glucose, row C also manifests some K⁺ loss due to non-pumping. A likely site for this is the perineurium since, at the outer surface of the perineurium in the conditions of row C, no external potassium was available for pumping.

Conclusions. The findings imply that (1) The perineurium's role in protecting the neural supply of potassium may not be entirely passive. (2) Lack of glucose in a local anesthetic solution may itself contribute to conduction block, especially when large volumes containing a vasoconstrictor have been used. (3) Lack of potassium in the solution, on the contrary, may tend to delay the onset of block. The results may call for reassessment of the optimal solute composition of local anesthetic solutions.

TABLE 1. EFFECTS OF EXTERNAL GLUCOSE AND/OR K⁺ LACK ON C-FIBER EXCITABILITY

Extraneural Glucose K ⁺ (mmol/kg)		Time to Non-conduction (min)	Core Na ⁺ (mmol/kg)	Core K ⁺ (mmol/kg)	Row
0	0	110 ± 10	117 ± 5	47 ± 5**	A
0	4	78 ± 9**	134 ± 13**	57 ± 6*	B
5	0	∞	110 ± 6	49 ± 5*	C
5	4	∞	117 ± 8	62 ± 3	D
unincubated control			110 ± 9	63 ± 4	

± S.D., $n \geq 5$, * $p < 0.05$, ** $p < 0.01$ (analysis of variance & unpaired t test)

∞: C-fiber potential normal for >120 min

References.

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