other neural and humoral factors such as the direct action of 'permeability nerves', the possibility of 'stretched pores' under extremely high pressure transients, and the influence of a variety of agents which may damage the air-blood barrier[6-10].

In conclusion, the results of the present study are consistent with other recent evidence[11,22,24] and further suggest that the increase in the flow of protein-rich lymph produced by intracranial hypertension is more likely explained by a recruitment of pulmonary microvascular surface area than by an increase in pulmonary microvascular permeability.

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Can blocking the Na/K exchange pump lead to a reduction in intracellular sodium?[1]

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Summary. It has been assumed that a rise in intracellular sodium should follow inhibition of the Na/K exchange pump. However, under certain conditions a reduction in intracellular sodium following pump blockage is possible. Many results postulating 'stimulation' of the Na/K exchange pump by low doses of the cardiac glycosides can be explained in this manner.

It has been assumed that a rise in intracellular sodium should follow the inhibition of the Na/K exchange pump[2-6]. In cardiac muscle, this rise in intracellular sodium is postulated to be an important first step leading to the increase in contractile force produced by cardiac glycosides[2,4]. However there are reports that following the application of low concentrations of the glycosides there is a decrease in intracellular $[Na^+]$ or Na^+ activity, or a rise in intracellular $[K^+]$[7-11]. These results have led to the suggestion that low concentrations of the glycosides can stimulate the Na/K exchange pump[7,9,12]. Another explanation is possible. The low concentrations of the glycosides by partially inhibiting an electrogenic Na/K exchange pump, will change the membrane potential[13-16]. The change in membrane potential will lead to changes in $[Na^+]_i$ and in $[K^+]_i$. A simple model shows the conditions under which low concentrations of glycosides could lower $[Na^+]_i$ owing to this effect.

For simplicity the model will be developed by considering only the Na and K currents and the Na/K pump. The steady state current due to sodium, which is known in cardiac muscle as the inward background current, I_{inb}, is assumed to follow the constant field equation[6,21], and therefore is monotonically rising with an increase in the negativity of the membrane potential (fig. upper panel). The potassium current, I_K, is assumed to depend on membrane potential as shown in the figure, middle panel (inwardly rectifying at negative potentials (< -30 mV) and outwardly rectifying at more positive potentials[17,18]). The Na/K pump is assumed to be potential independent[14], depend linearly on $[Na]_i$, and to have a fixed coupling ratio of 3 Na^+/2 K^+ (fig., lower panel). The pump is therefore electrogenic, with a current I_p. These assumptions are most parsimonious with the available data, although a window

Na/K pump blockage can lead to a reduction in $[Na]_i$

A) Rest	B) Immediately following a 5% pump blockage	C) Later, new steady state*
$V = -90$ mV	$V = -80$ mV	$V = -75$ mV
$I_K = 2$	$I_K = 1.76$	$I_k = 1.71$
$I_p = 1$	$I_p = .95$	$I_p = .855$
$I_{inb} = -3$	$I_{inb} = -2.71$	$I_{inb} = -2.565$
$[Na]_i = 7$ mM	$[Na]_i = 7$ mM	$[Na]_i = 6.3$ mM

*The time necessary to achieve this new steady state is dependent on the cell surface to volume ratio. The steady state achieved is independent of that ratio. I_K, I_{inb} and I_p as given in the figure. The units are arbitrary, and would have to be scaled to the size of the preparation.