TRANSMURAL ELECTRIC POTENTIALS AND THEIR RESPONSE TO SUGARS IN THE INTESTINES OF ACUTE CHOLERA PATIENTS AND NORMAL SUBJECTS

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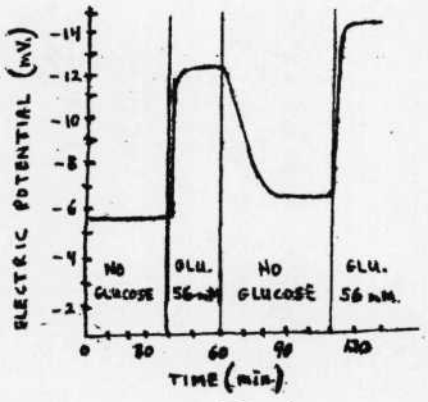
In an effort to study the intestinal pathophysiology of cholera, we have devised a technique for the measurement of transmural electric potential in the intact human intestine. A polyvinyl tube swallowed by the subject and completely filled with electrolyte solution serves as the luminal electrode. The site of the neutral or reference electrode is the lightly abraded skin of the forearm, which has been demonstrated to be equipotential with intestinal serosa.

In normal subjects, transmural potentials in the stomach and colon are in the order of -25 to -60 mV., lumen negative with respect to the reference electrode. Baseline potentials in the small bowel are much closer to zero, usually ranging from +2 to -6 mV.

With the instillation of certain sugars into the small bowel lumen, there is a prompt and sustained increase in luminal negativity. Figure 1 demonstrates this effect as elicited by the infusion of glucose into the normal ileum.

Figure 1

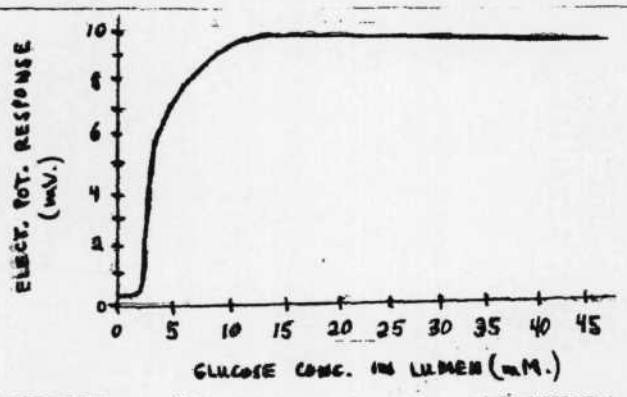
PATTERN OF ELECTRIC POTENTIAL RESPONSE TO GLUCOSE



Moreover, there seems to be a direct relationship between the concentration of glucose in the intestinal lumen and the magnitude of the increase in potential difference. This relationship, as demonstrated in three separate experiments in the distal ileum, is shown graphically in Figure 2.

Figure 2

ELECTRIC POTENTIAL RESPONSE TO GLUCOSE

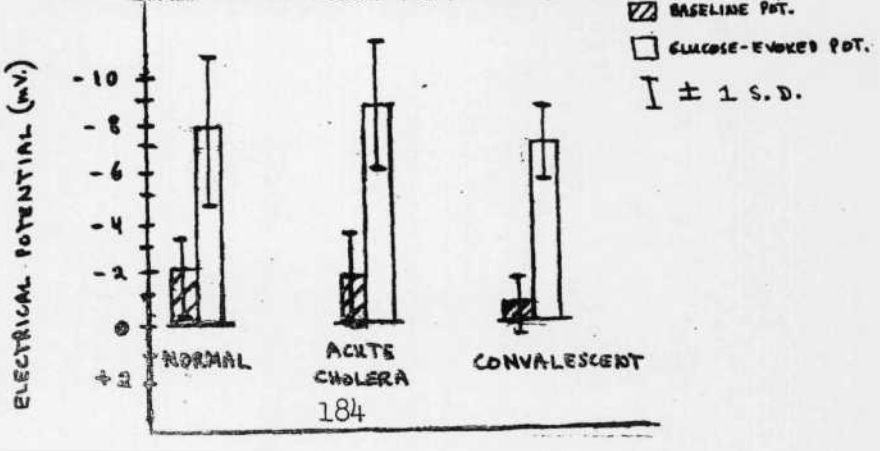


Similar electrical effects are seen with galactose, which is also actively transported though not metabolized by intestinal epithelium; but these effects have not been observed with any of the non-actively transported sugars we have tested: fructose, mannose, mannitol, and xylose.

In applying our technique to the study of cholera, we have measured baseline and glucose-evoked potentials in the small bowels of acute cholera patients, and we have demonstrated no abnormality. Figure 3 shows the baseline and glucose-evoked potentials in 6 normal subjects, in 7 patients with acute cholera, and in the same 7 patients restudied in convalescence. There are no differences among these three groups in their baseline potentials nor in the magnitude of their response to glucose.

Figure 3

BASELINE AND GLUCOSE-EVOKED POTENTIALS
IN CHOLERA PATIENTS AND NORMAL SUBJECTS



Numerous other animal and *in vitro* studies have demonstrated that transmural electric potential in the intestine is primarily attributable to active sodium transport from the lumen. We have concluded, therefore, that whatever may be the pathogenetic mechanism of intestinal fluid loss in cholera, there is no impairment of that portion of active sodium transport which is electrogenic and which is stimulated by actively transported sugars.

SYMPOSIUM ON CHOLERA

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