J. Physiol. (1974), **242**, *pp.* 173–188
With 12 text-figures
Printed in Great Britain

MEMBRANE POTENTIAL AND RESISTANCE MEASUREMENT IN ACINAR CELLS FROM SALIVARY GLANDS *IN VITRO*: EFFECT OF ACETYLCHOLINE

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(*Received 28 February 1974*)

SUMMARY

1. Cell membrane potential and input resistance measurements were made on segments of submaxillary glands from mice, rabbits or cats placed in a tissue bath, which was perfused with physiological salt solutions.

2. During exposure to a standard Krebs–Henseleit solution, ACh stimulation always evoked a marked decrease in input resistance and time constant. The change in potential evoked by ACh stimulation was either a monophasic hyperpolarization (low resting potential) or a depolarization followed by hyperpolarization (high resting potential).

3. Increasing $[Ca]_o$ from 2·56 to 10 mM resulted in an enhanced input resistance. Under this condition it was sometimes possible to obtain current–voltage relations. The relationship was linear in the range -50 to -10 mV. In the absence of extracellular Ca the resting potential was reduced and ACh mostly evoked hyperpolarizations. In those cases when the resting potential remained high biphasic potentials were still observed.

4. During exposure to Na-free solutions the resting potential was either unchanged or slightly enhanced. ACh never evoked biphasic potentials, but always large hyperpolarizations.

5. In the first period (1 hr) after exposure to a K-free solution ACh normally evoked very large hyperpolarizations, often to more than -100 mV. After several hours of exposure to K-free solution the input resistance gradually increased and ACh evoked a tremendous fall in input resistance and time constant with only a small potential change. Reintroducing control solution, ($[K]_o = 4{\cdot}7$) for a short period at this stage, caused a very marked hyperpolarization (about 30 mV) unaccompanied by a change in input resistance and time constant.

6. Replacing extracellular Cl by SO_4 hyperpolarized the cell membrane. ACh mostly evoked hyperpolarization under this condition, but

occasionally biphasic potentials were observed. Increasing [K] of the sulphate solution depolarized the cell membrane by about 49 mV per tenfold increase in [K]. In the presence of ACh the membrane behaved as a K-selective membrane with a slope of the linear curve relating membrane potential to $[K]_0$ of 59 mV per tenfold increase in $[K]_0$.

7. It is concluded that ACh evokes a marked increase in surface cell membrane permeability of salivary acinar cells. The ACh evoked hyperpolarization is due to an increase in P_K: the depolarization frequently preceding the hyperpolarization is probably mainly related to an increase in P_{Na}. The membrane Na–K pump can act electrogenically at least under conditions of Na loading.

INTRODUCTION

Since Lundberg (1955) described the first attempt to define the electrophysiology of salivary gland cells using glass micro-electrodes, many studies of this kind have been carried out (Petersen, 1972). It is clear now that there were technical problems in many earlier studies of this kind and that therefore the resting membrane potential had been seriously underestimated (Pedersen & Petersen, 1973). The hypothesis that the physiological transmitter substance acetylcholine (ACh) mainly acts by increasing K and possibly Na permeability of the acinar cell membrane (Imai, 1965*b*; Yoshimura & Imai, 1967; Petersen 1970*a*,*b*) has been widely accepted, but never rigorously tested (Schneyer, Young & Schneyer, 1972). It appears now that also the activation of the α-adrenoceptor may be explained in this way (Petersen & Pedersen, 1974).

Previous electrophysiological studies have been carried out using *in vivo* or perfused preparations. The recent progress in liver and pancreas cell membrane electrophysiology (Haylett & Jenkinson, 1972; Dean & Matthews, 1972; Nishiyama & Petersen, 1974) obtained by using superfused slices or segments, encouraged us to use a similar approach for the salivary glands, since in this type of preparation the stability of membrane potential recording is by far superior to that of perfused preparations (Petersen, 1974). The results obtained show that ACh evokes a very marked decrease in input resistance and time constant associated either with a monophasic hyperpolarization or a biphasic (depolarization–hyperpolarization) secretory potential. The hyperpolarization is undoubtedly due to an increase in K permeability and it appears likely that the initial depolarization is due to Na entry.

METHODS

Segments of submaxillary glands from female mice, cats (anaesthetized with chloralose, 80 mg/kg body weight) or rabbits (anaesthetized with urethane, 1·5 g/kg body weight) were used. The segments were about $2 \times 2 \times 5$ mm and always included a totally undamaged free surface area of the gland. It proved unnecessary to decapsulate the surface. The segments were secured to a perspex platform placed in a perspex tissue bath (20 ml.) through which a physiological salt solution warmed to 37° C flowed at about 10 ml./min. The standard solution had the following composition (mM): NaCl 103, KCl 4·7, $CaCl_2$ 2·56, $MgCl_2$ 1·13, $NaHCO_3$ 25, NaH_2PO_4 1·15, D-glucose 2·8, Na pyruvate 4·9, Na glutamate 4·9, Na fumarate 2·7; it was gassed with 95 % O_2, 5 % CO_2. In some experiments a bicarbonate-free solution was used. The solution was then buffered by Tris (5 mM) and the Na concentration augmented to maintain isosmolarity. This solution was gassed with pure oxygen. In experiments where the K concentration was altered corresponding alterations in Na concentration ensured constant osmolarity, similarly alterations in Ca concentration were compensated by altering the Na concentration. The low Cl solution was a bicarbonate-buffered isotonic solution without NaCl but containing Na_2SO_4 and 10 mM-$CaCl_2$. In this case the Na concentration was about 165 mM. The Na-free solutions were Tris-buffered solutions in which all NaCl was replaced by either sucrose or TrisCl. The substrates were added as the acid rather than the Na salt and pH was adjusted to 7·4 with HCl and Tris(base), phosphate was omitted.

The measurement of membrane potential was carried out by using glass microelectrodes filled with 3 M-KCl + 10 mM-K citrate (20–30 MΩ). Input resistance was measured by injecting de- or hyperpolarizing current pulses (100 msec) through the recording micro-electrode using a technique previously described (Nishiyama & Petersen, 1974).

Stimulation of the tissue was normally done by injecting 14 μg ACh directly into the tissue bath (20 ml.) very near to the site of the electrode impalement using a fine needle mounted on a tuberculine syringe. Sometimes small doses of only 0·14 μg were used.

RESULTS

The effect of ACh during exposure to control solution

Mouse submaxillary gland

The mean resting cell membrane potential from superficial cells was −57 mV (n = 107). Figs. 1 and 2 show typical effects of ACh. It is seen that ACh evoked a very marked reduction in input resistance and time constant. The effect on the potential, however, was variable. The most frequent response was a monophasic hyperpolarization (Fig. 1) although a biphasic potential change (depolarization–hyperpolarization) was also seen relatively often (Fig. 2). In some cases a monophasic depolarization was observed. Table 1 summarizes these results. It is clear that the initial depolarization was only seen at relatively high resting potentials similarly to observations under *in vivo* conditions (Kagayama & Nishiyama, 1974).

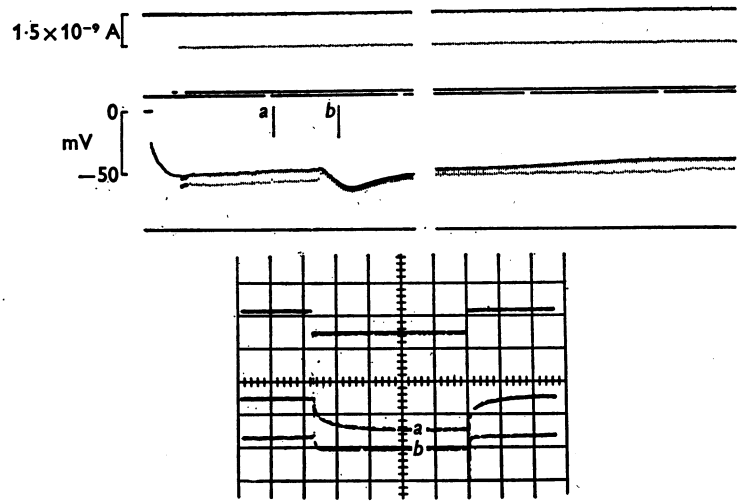

Fig. 1. Mouse submaxillary gland: membrane potential and resistance measurement. Upper part shows the pen recording traces. From top: current monitor, time marker (short pulses every second, long pulses every minute), membrane potential and event marker (at signal 14 μg ACh added to bath). The interruption of the tracings indicates a 4 min interval. Lower part shows an oscilloscope screen picture. Upper trace represents the current signal and *a* and *b* represent the shapes of the current-induced membrane hyperpolarizations at times *a* and *b* in the pen recorder trace. Calibration: horizontal, 20 msec/division; vertical, 10 mV or $2{\cdot}5 \times 10^{-9}$A/division.

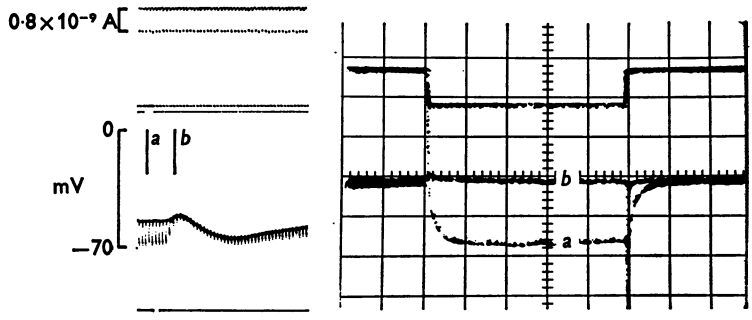

Fig. 2. Mouse submaxillary gland: example of a biphasic potential change following ACh stimulation. Calibration of oscilloscope picture: horizontal, 20 msec/division; vertical, 10^{-9}A or 5 mV/division.

Rabbit and cat submaxillary glands

The mean resting potential from superficial cells in the rabbit submaxillary gland was $-59{\cdot}2 \pm 1{\cdot}1$ mV ($n = 69$) which is close to the *in vivo* value (Kagayama & Nishiyama, 1974). The effect of ACh (14 μg) was essentially similar to that seen in the mouse. The input resistance was

reduced from $5{\cdot}2 \pm 1{\cdot}0$ to $3{\cdot}4 \pm 1{\cdot}0$ MΩ. The effect on the potential could be a pure hyperpolarization, or in a few cases a pure depolarization, but the most frequent response was a biphasic potential change (depolarization–hyperpolarization) (Fig. 3).

The mean resting potential from superficial cells of the cat submaxillary gland was $-38{\cdot}2 \pm 1{\cdot}3$ mV ($n = 51$). The input resistance decreased from $4{\cdot}4 \pm 1{\cdot}2$ to $3{\cdot}4 \pm 1{\cdot}2$ MΩ after application of ACh (14 μg). The effect of ACh on the potential was always to cause hyperpolarization (Fig. 4). The mean value of the ACh-induced hyperpolarization was $16{\cdot}9 \pm 1{\cdot}3$ mV.

TABLE 1. Membrane potential and resistance of mouse submaxillary acinar cells

	Resting potential (mV)	ACh-evoked depolarization (mV)	ACh-evoked hyperpolarization (mV)	*n*	Resting input resistance (MΩ)	Minimum input resistance after ACh (MΩ)	*n*
Total population	$-56{\cdot}9 \pm 0{\cdot}9$	—	—	107	$4{\cdot}3 \pm 1{\cdot}0$	—	22
Biphasic type	$-60{\cdot}4 \pm 1{\cdot}9$	$4{\cdot}9 \pm 0{\cdot}7$	$10{\cdot}0 \pm 1{\cdot}1$	20	$5{\cdot}0 \pm 0{\cdot}7$	$1{\cdot}0 \pm 0{\cdot}2$	6
Hyperpolarizing type	$-50{\cdot}9 \pm 1{\cdot}1$	—	$14{\cdot}8 \pm 0{\cdot}9$	47	$4{\cdot}0 \pm 0{\cdot}7$	$0{\cdot}7 \pm 0{\cdot}2$	12
Depolarizing type	$-66{\cdot}9 \pm 2{\cdot}0$	$9{\cdot}8 \pm 1{\cdot}1$	—	12	$4{\cdot}4 \pm 2{\cdot}5$	$3{\cdot}5 \pm 2{\cdot}9$	4
Cells in which effect of ACh was not tested	$-60{\cdot}3 \pm 1{\cdot}4$	—	—	28	—	—	—

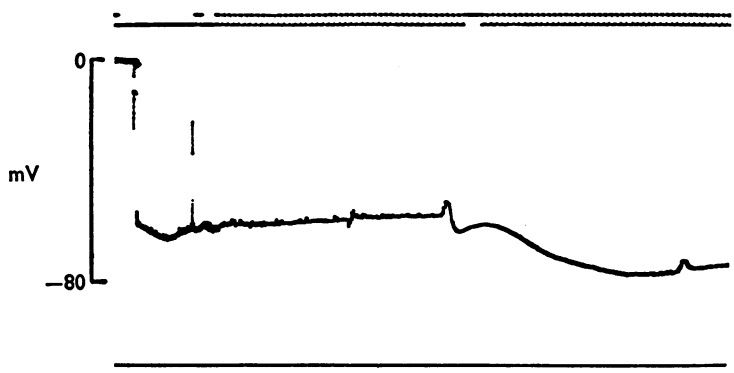

Fig. 3. Rabbit submaxillary gland: effect of ACh on membrane potential.

The effect of varying $[Ca]_o$ *(mouse)*

Omitting Ca from the superfusion solution always reduced the resting membrane potential. 45–180 min after exposure to a Ca-free solution the mean membrane potential was $-49{\cdot}0 \pm 6{\cdot}3$ mV and ACh mostly

evoked pure hyperpolarization (13·7 mV ± 4·8). In a few cases when high resting potentials could be maintained biphasic potentials were observed (Fig. 5).

Increasing $[Ca]_0$ from the normal 2·6 to 10 mM increased the resting potential from −47·3 ± 3·3 to −58·7 ± 2·4 mV. The control value was low because a CO_2/HCO_3-free Tris buffered solution was used. At

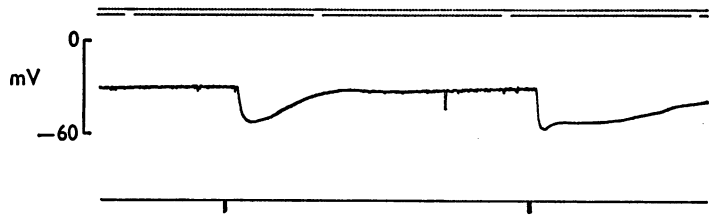

Fig. 4. Cat submaxillary gland: membrane potential recording. At first signal 0·14 μg and at second signal 14 μg ACh was added to the bath.

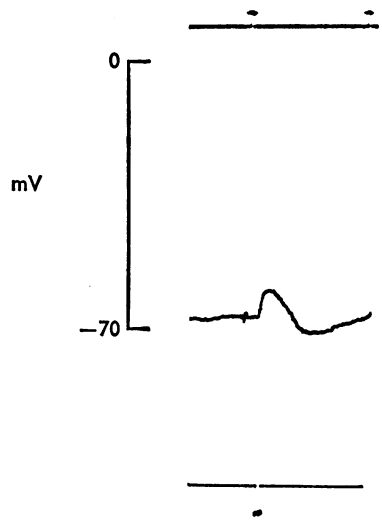

Fig. 5. Mouse submaxillary gland: Ca-free solution (180 min). Biphasic potential evoked by ACh.

$[Ca]_0$ = 10 mM the input resistance was greatly enhanced (13·3 ± 1·8 MΩ) compared to control conditions (4·4 ± 1·0 MΩ). This high input resistance made it possible to study the current–voltage (I/V) relation over a large range of membrane potentials, something which was unfortunately not possible during exposure to control solutions. Fig. 6*a* shows membrane potential displacements caused by rectangular current pulses of varying strength. In Fig. 6*b* is shown a plot of the I/V relation obtained from the

curves of Fig. 6*a*. In the range -10 mV to -50 mV the relation was linear. The effect of ACh during exposure to 10 mM-Ca was similar to that seen during control conditions but the change in input resistance was rather more marked (Fig. 7). ACh evoked a decrease in input resistance from $13{\cdot}3 \pm 1{\cdot}8$ to $1{\cdot}6 \pm 0{\cdot}6$ MΩ. Both pure hyperpolarizations and biphasic potential changes were seen.

The effect of Na-free solutions (mouse)

During exposure to a CO_2/HCO_3-free Tris buffered solution the resting potential was $-48{\cdot}5 \pm 2{\cdot}4$ mV. Replacing all Na by Tris did not have any appreciable effect on the membrane potential ($-48{\cdot}0 \pm 1{\cdot}9$ mV) whereas replacement of NaCl by sucrose slightly increased the resting potential to $-54{\cdot}8 \pm 3{\cdot}8$ mV. In both Na-free solutions ACh evoked marked hyperpolarizations ($16{\cdot}3 \pm 1{\cdot}8$ and $23{\cdot}5 \pm 3{\cdot}5$ mV) (Fig. 8). Biphasic potentials following ACh stimulation were never seen although they were obtained in the control periods of the same experiments.

The effect of K-free solutions (mouse)

The resting membrane potential of the mouse submaxillary acinar cells was clearly dependent on $[K]_o$ (Fig. 12) and removal of extracellular K from the superfusion solution hyperpolarized the cell membrane. In K-free solutions both biphasic potentials and monophasic hyperpolarizing potentials in response to ACh stimulation were observed. In the first period after introducing the K-free solution (up to 1 hr) the most frequently observed effect of ACh was a very large hyperpolarization (Fig. 9). After several hours exposure there was a gradual increase in input resistance to values even higher than those observed in the 10 mM-Ca solution (40 MΩ) and in these cases ACh evoked a tremendous decrease in input resistance with hardly any effect on the potential. In these cases the resting potential was still relatively high (-50 to -70 mV). Reintroducing the control solution ($[K]_o = 4{\cdot}7$ mM) at this stage for a short period caused a very marked hyperpolarization from $-65{\cdot}5 \pm 2{\cdot}0$ to $-98{\cdot}9 \pm 2{\cdot}4$ mV ($P < 0{\cdot}001$). This hyperpolarization was not accompanied by changes in input resistance or time constant (Fig. 10).

The effect of low Cl solutions (mouse)

Replacing extracellular Cl by SO_4 resulted in a marked hyperpolarization of the acinar cell membrane. The mean resting potential during exposure to the sulphate solution was $-76{\cdot}3 \pm 1{\cdot}7$ mV (control level $-56{\cdot}9 \pm 0{\cdot}9$ mV). ACh mostly evoked hyperpolarization in this condition ($12{\cdot}7$ mV $\pm 1{\cdot}7$) (Fig. 11), although biphasic potential changes were occasionally observed.

The effect of varying $[K]_o$

Fig. 12 shows the relationship between membrane potential and $[K]_o$ in the absence and presence of ACh. Only at $[K]_o$ = 4·7 mM and in K-free solutions were clear hyperpolarizations produced by ACh. The slope of the curve relating membrane potential to $[K]_o$ was 49 mV per tenfold increase in $[K]_o$ in the absence of ACh and 59 mV per tenfold increase in $[K]_o$ in the presence of ACh.

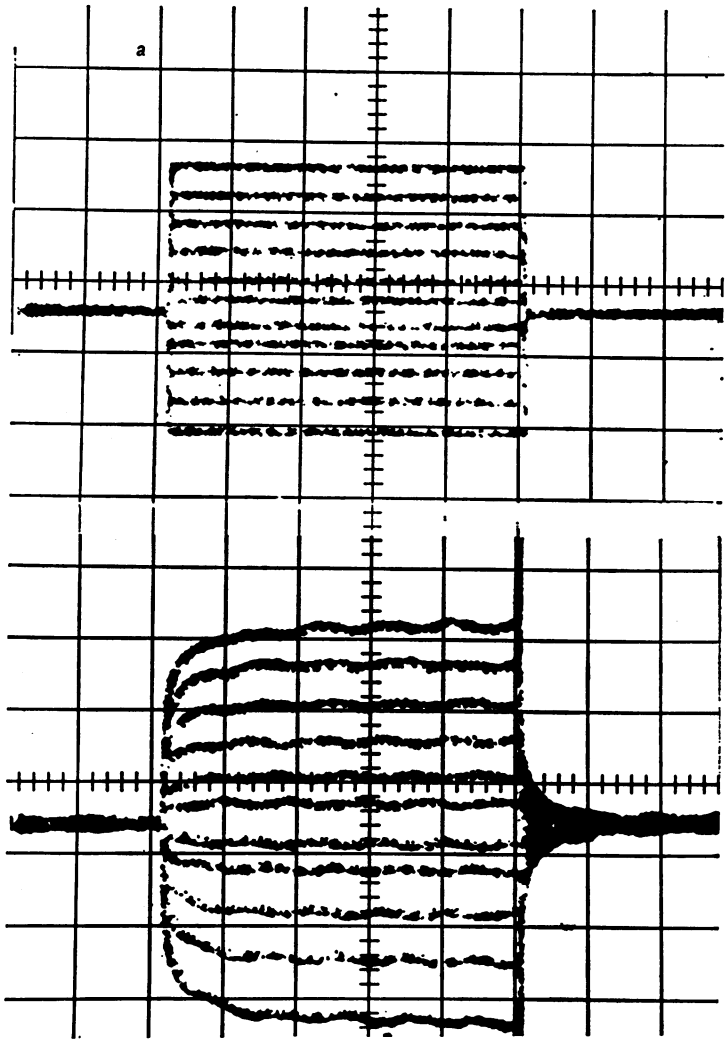

Fig. 6*a*. For legend see facing page.

The effect of omitting Ca

Omitting Ca from the Cl-free SO_4 solution caused a dramatic reduction in resting potential from $-76{\cdot}2 \pm 2{\cdot}2$ to $-36{\cdot}3 \pm 1{\cdot}2$ mV. In the absence of superfusion fluid Ca ACh evoked very large hyperpolarizations (Fig. 11).

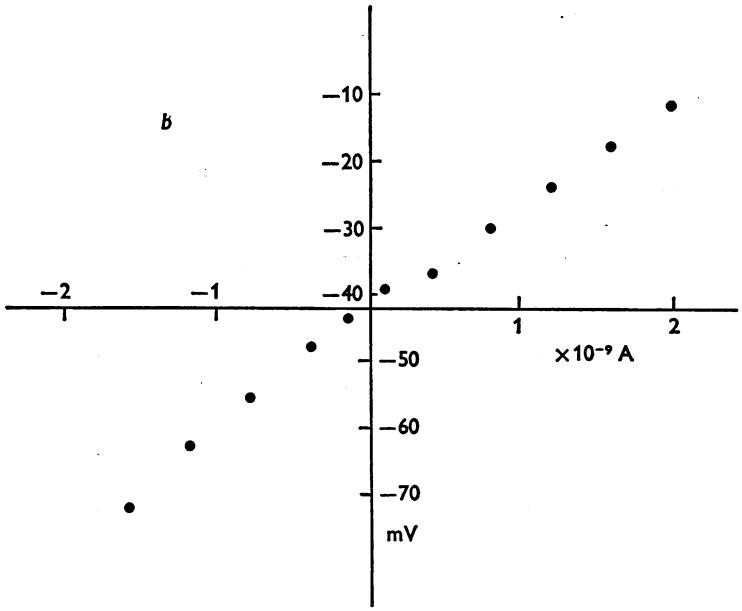

Fig. 6. Mouse submaxillary gland: $[Ca]_0$ = 10 mM. Current–voltage relations. *a*, oscilloscope screen picture. Calibration: horizontal, 20 msec/division; vertical, 10^{-9}A or 10 mV/division. *b*, plot of I/V relation obtained from the steady-state levels of *a*.

DISCUSSION

The present results confirm the findings of Pedersen & Petersen (1973) that the salivary acinar cell membrane potential generally is much higher than previously recognized and that the resting membrane potential is mainly influenced by the potassium equilibrium potential (E_K) (Fig. 12). A most dramatic change in the resting potential was achieved replacing Cl by SO_4. It is possible that this could have been caused by an increase in P_K since the sensitivity to variations in $[K]_0$ was increased during exposure to SO_4 solutions. For liver cells such an explanation has been given (Claret, Claret & Mazet, 1973).

One of the essential findings in the present study is the very marked decrease in input resistance and time constant evoked by ACh (Figs. 1, 2).

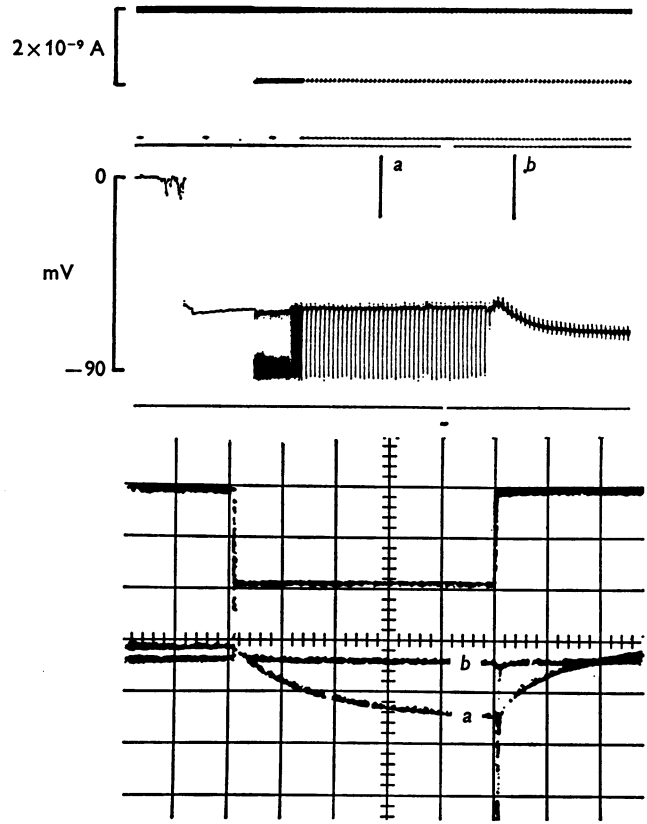

Fig. 7. Mouse submaxillary gland: $[Ca]_o$ = 10 mM. Effect of ACh. Calibration in oscilloscope picture: horizontal, 20 msec/division; vertical, 10^{-9}A or 20 mV/division.

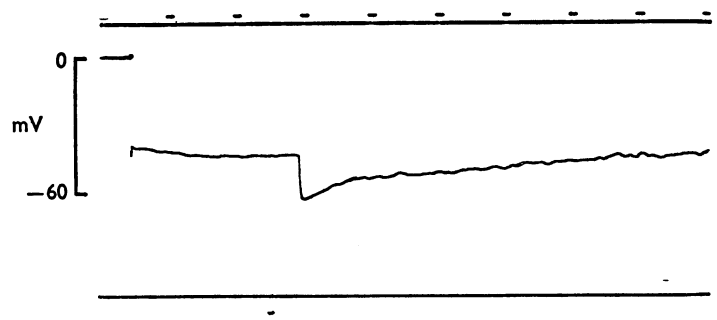

Fig. 8. Mouse submaxillary gland: Na-free Tris solution. Effect of ACh.

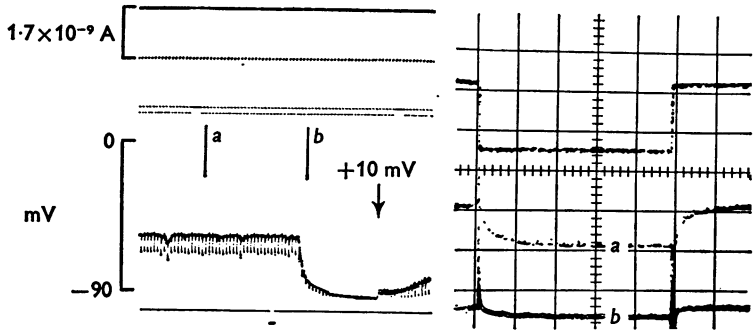

Fig. 9. Mouse submaxillary gland: K-free solution. The hyperpolarization following ACh addition was so large that the pen recorder was saturated. At arrow marked + 10 mV a 10 mV potential was applied between earth and the indifferent bath electrode. Oscilloscope screen calibration: horizontal, 20 msec/division; vertical, 10^{-9}A or 10 mV/division.

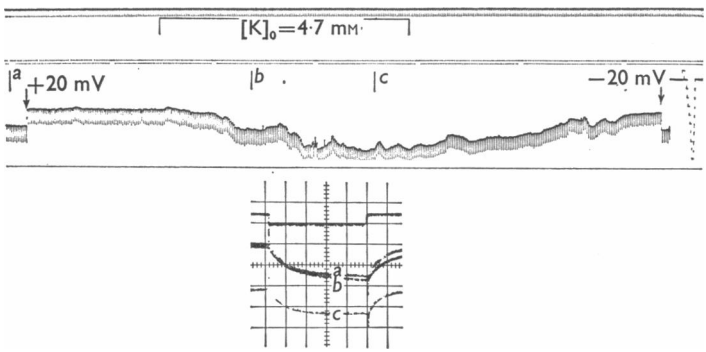

Fig. 10. Mouse submaxillary gland: K-free solution (180 min). Effect of K readmission. At the level of the maximal K-induced hyperpolarization the pen recorder was saturated during the current-induced hyperpolarizations. Strength of injected current pulses was 5×10^{-10} A. Voltage calibration is seen to the right when after retraction of the micro-electrode tip to the bathing solution a 90 mV calibration potential (in steps of 10 mV) was applied. Oscilloscope screen calibration: horizontal, 20 msec/division; vertical, 10^{-9} A or 10 mV/division.

Lundberg (1957) stated that stimulation of the cat sublingual gland changed the cell input resistance from 2·0 to 1·8 MΩ and Imai (1965*a*) reported a change from 18 ± 12 to 14 ± 11 MΩ. These effects were hardly convincing and the present results together with the *in vivo* results of Kagayama & Nishiyama (1974) therefore represent the first unequivocal demonstrations of stimulation-induced reductions of salivary acinar cell input resistance. We have recently reported similar findings for the pancreatic acinar cells (Nishiyama & Petersen, 1974) and concluded that this

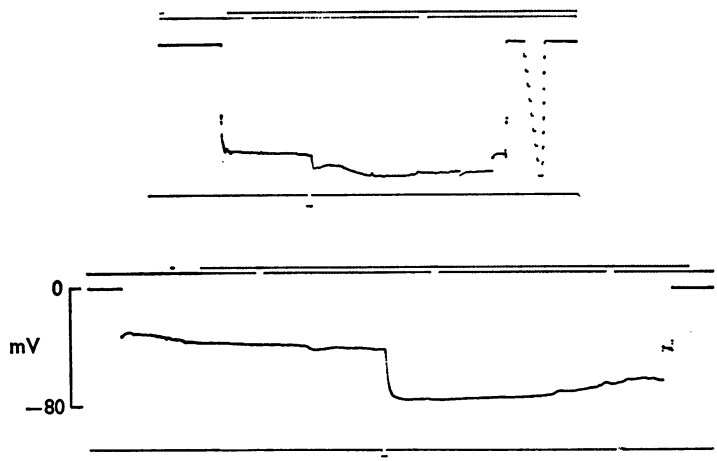

Fig. 11. Mouse submaxillary gland: low Cl sulphate solution. Upper record $[Ca]_o$ = 10 mM, lower record Ca-free (30 min). In upper record calibration signal of 90 mV (in steps of 10 mV).

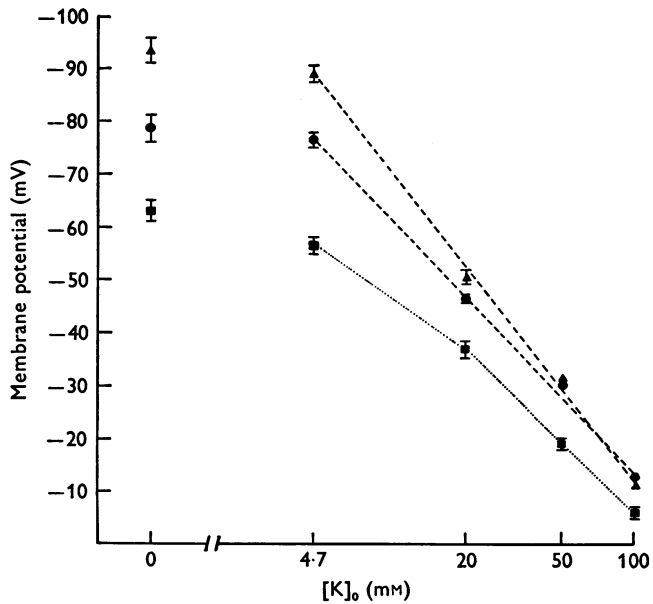

Fig. 12. The dependence of the salivary acinar cell membrane potential on $[K]_o$. Mean values ± S.E. (where graphically possible). ■ control solution, ● low Cl sulphate solution, ▲ low Cl sulphate solution: maximal potential after addition of 14 μg ACh.

decrease in input resistance, in view of the marked reduction of the time constant can only be explained by a reduction of the surface cell membrane resistance. This conclusion is also valid for the salivary glands.

While the potential change induced by ACh was always accompanied by a marked decrease of input resistance and time constant the effect of reintroducing K after a long period of K deprivation was to hyperpolarize the cell membrane markedly without any change in input resistance and time constant (Fig. 10). In the pancreas and the parotid it has already been described that K reintroduction to a K-free solution causes a rapid hyperpolarization which is immediately abolished by strophanthin-G (1 mM) (Petersen, 1973*a*; Pedersen & Petersen, 1973). The present results confirm these observations and furthermore directly show that the hyperpolarization following K-reintroduction is not caused by a membrane permeability change. It thus seems safe to conclude that the membrane Na-K pump in the salivary acinar cells at least under extreme conditions can act electrogenically.

The existence of an electrogenic pump and the existence of biphasic potential changes after ACh stimulation invite a discussion about the possibility that the hyperpolarization following ACh could be secondary to the initial depolarization. The intracellular rise in Na concentration which probably occurs during the initial depolarization (Na influx) could activate the Na–K pump thus hyperpolarizing the membrane. This would be a situation similar to the one described for smooth muscle (Bolton, 1973) and an attractive way of explaining this biphasic potential change following ACh stimulation. Several pieces of evidence, however, disagree with such a contention. (1) The hyperpolarization is accompanied by a marked reduction in input resistance and time constant (Fig. 1). (2) The hyperpolarization is not reduced or abolished during exposure to a K-free solution but clearly enhanced (Fig. 9). (3) The amplitude of the ACh-induced hyperpolarization is severely decreased after augmentation of $[K]_o$ (Fig. 12). (4) During exposure of the tissue to a Na-free solution ACh causes a very marked hyperpolarization (Fig. 8). (5) In the presence of strophanthin-G (1 mM) ACh-induced hyperpolarization is still present (Petersen, 1973*b*). All these results would however be compatible with the hypothesis that the hyperpolarization was due to an increase in K permeability. This conclusion has, as already mentioned in the introduction, been reached many years ago (Imai, 1965*b*; Yoshimura & Imai, 1967; Petersen, 1970*a*, *b*) but the present evidence is much stronger than that previously presented. However, the question about the mechanism of the ACh-induced membrane hyperpolarization is not solved. Is the K permeability change caused by a direct ACh action on the cell membrane or is the hyperpolarization secondary to some other effect of ACh on the

cell, explaining the delay of the hyperpolarization (occurring after the initial depolarization)? We cannot at present answer this question satisfactorily but maybe a clue can be found in some recent results about the importance of Ca for K permeability. The K permeability of the erythrocyte cell membrane is controlled by the intracellular Ca concentration (Romero & Whittam, 1971). It has furthermore recently been suggested that Ca acts as a second messenger in the action of ACh and α-adrenergic substances on salivary gland cells. Selinger, Eimerl, Savion & Schramm (1974) have shown that the Ca ionophore A 23187 can simulate the action of cholinergic or α-adrenergic drugs on parotid K release and Selinger, Batzri, Eimerl & Schramm (1973) have shown that in the absence of extracellular Ca plus addition of EDTA K release evoked by cholinergic or α-adrenergic stimulation is abolished. Petersen & Pedersen (1974) have recently shown that the α-adrenergic membrane hyperpolarization of the parotid acinar cells is shortened in the absence of extracellular Ca and that reintroduction of Ca simulates the action of adrenaline on the cell membrane also in the presence of cholinergic and adrenergic blocking agents. One possible explanation of these findings would be that the mechanism of action of ACh on the acinar cell membrane is to introduce Ca into the cell cytoplasm. In the concept of Selinger *et al.* (1974) the Ca introduced into the cytoplasm comes from outside the cell. In our experiments ACh evoked very large hyperpolarizations in the absence of extracellular Ca (Fig. 11). It is possible that Ca could be released from stores inside the cell as an obligatory event in the ACh-evoked membrane conductance change. Models of this kind have recently been discussed in relation to pancreatic stimulus-secretion coupling (Matthews, 1974). An increase in the efflux of ^{45}Ca from prelabelled cat submaxillary glands following ACh or adrenaline stimulation has previously been demonstrated (Nielsen & Petersen, 1972).

Does ACh membrane receptor interaction cause an increase in Na influx? Imai (1965*b*) originally proposed that ACh mainly acted to increase the membrane K permeability. Petersen (1970*b*) proposed that in addition to the effect on K permeability ACh also increased Na permeability. The experimental basis for this proposal was the finding that the amplitude of the ACh-induced hyperpolarization was greatly enhanced in Na-free solutions. This has since been confirmed by Fritz (1974) and again in the present work. More direct evidence in favour of an increased Na conductance comes from the finding of a conductance change preceding the ACh-induced hyperpolarization, occurring simultaneously with the depolarization seen at high resting potentials. The depolarization could admittedly be explained in other ways. Ca influx or Cl efflux might also be considered. However, in Ca-free solutions biphasic potentials could

be seen in those cases when the resting potential remained high, whereas in Na-free solutions biphasic potentials were never observed. If the depolarization were due to Cl efflux this would imply the existence of a Cl accumulating mechanism in the cell membrane. This cannot be excluded since it has been described in, for example, the squid axon (Keynes, 1963). On the other hand assessment of the intracellular Cl concentration in the dog submaxillary gland, during resting and stimulated conditions showed no difference whereas a marked increase in intracellular Na concentration after stimulation was found in the same experiments (Imai, 1965*b*). On balance the evidence is at present in favour of the hypothesis of a Na influx underlying the initial depolarization. The fact that the initial resistance decrease following ACh stimulation is only accompanied by depolarization at resting potentials above -50 mV or that the equilibrium potential for the initial membrane potential change is about -50 mV suggests that the initial part of the ACh response is not a selective increase in Na permeability but rather a combination of Na and K permeability changes. If this is correct the mechanism of action of ACh on the salivary acinar cell membrane and on the pancreatic acinar cell membrane (Nishiyama & Petersen, 1974) could be considered to be very similar.

This work was supported by a grant from the U.S. National Cystic Fibrosis Research Foundation. We thank G. L. Pedersen for skilfull technical assistance.

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