STUDIES ON THE SECRETORY POTENTIAL OF ACINAL CELL OF DOG'S SUBMAXILLARY GLAND AND THE IONIC DEPENDENCY OF IT

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Electrophysiological studies by LUNDBURG⁶⁻¹⁰⁾ have thrown light on the study of the salivary gland of which the secretory mechanism is not known. He found that the membrane potentials of acinal cells of cat's submaxillary gland and sublingual gland are hyperpolarized by stimulation of the chorda tympani, and called this hyperpolarized potential the secretory potential. The origin of these potential changes is not clarified in detail. LUNDBURG has discussed the various possibilities and concluded that the hyperpolarization of the intracellular potential is due to an active transport of chloride ion from the extracellular fluid into the cell.

It has been previously reported by the present authors^{4,5)} that the intracellular resting potential of the acinal cell of dog's submaxillary gland was -42 mV on an average of 150 measurements, and the potential is hyperpolarized by stimulation of the chorda nerve to -57 mV on an average. The site of this potential generation was confirmed to be located in an acinal cell of the gland with the Turnble blue method. In an attempt to examine the hypothesis of LUNDBURG, a perfusion experiment on dog's submaxillary gland was performed, and the intracellular potential was measured with the microelectrode method. The ionic balance study of the nonperfused gland and the analysis of ionic concentrations of the gland tissue were also carried out.

METHOD

The dogs were anesthetized with sodium thiopental (30-50 mg/Kg intravenously). The submaxillary gland was dissected, leaving the nerves, duct, and blood vessels connected to the head of the animal. A cannule was inserted into the duct to collect the saliva flowing out of it.

Perfusion. All branches from the carotid artery and juglar vein were ligated except those leading to the submaxillary gland. One of the carotid branches, usually the lingular artery, was connected to a cannule of the perfusion apparatus, which had been

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devised by INOUE³⁾, and reported elsewhere^{4,15)}. A hydrostatic pressure of 120 cmH₂O was used to flow the perfusate which was warmed by passing it through a 38°C-bath. The perfusate was circulated through the submaxillary gland after clamping the carotid

FIG. 1. Schematic representation of perfusion experiment with microglass-electrode.

TABLE 1. Composition of the perfusate.

Normal-Ringer		NO ₃ -Ringer	
NaCl	140.0 (mM)	NaNO ₃	140.0 (mM)
KCl	4.0	KNO ₃	4.0
NaHCO ₃	10.0	NaHCO ₃	10.0
CaCl ₂	2.0	Ca(NO ₃) ₂	2.0
MgSO ₄	1.3	MgSO ₄	1.3
Li-Ringer		SO ₄ -Ringer	
LiCl	140.0 (mM)	Na ₂ SO ₄	90.0 (mM)
KCl	4.0	K ₂ SO ₄	2.0
CaCl ₂	2.0	NaHCO ₃	10.0
MgSO ₄	1.3	MgSO ₄	2.0
K-isotonic Solution		K-free-Ringer	
KCl	150.0 (mM)	NaCl	144.0 (mM)
		NaHCO ₃	10.0
		CaCl ₂	2.0
		MgSO ₄	1.3

Each solutions are added 25g of PVP per litter.

artery (FIG. 1). The perfusate was composed of an isotonic saline solution as indicated in TABLE 1, 2.5% of polyvinyle pyrrolidone (PVP) which maintained the colloid-osmotic pressure, and red blood cells which were suspended in 35 Vol% to supply enough oxygen to the gland.

Micro-glass-electrode. Conventional glass capillary microelectrodes as described by LING and GERARD were used for recording the intracellular potential. They were filled with 3M-KCl and had a resistance of from 10 to 30 M Ω . Each microelectrode tip was checked with a microscope ($\times 500$) before use. The gland was placed on a plastic spoon which had been covered with a reference electrode (Ag-AgCl), and was fixed by pressing a plastic ring on the surface of the gland (see FIG. 1). For recording the intracellular potential, a cathode follower type preamplifier (Nihon-Koden MZ-3A), dc-main amplifier (Nihon-Koden ADH-2) and a pen-recorder (Nihon-Koden W-1) were used. The secretion of saliva was provoked by stimulating the chorda tympani with an electric stimulator (Nihon-Koden MSE-3) which generated a tetanic current (usually 2V, 20 cps, pulse duration 5 msec).

Ionic balance study. Similarly to BURGEN's experiment¹¹, the venous blood from the gland was collected every 30-60 sec. separately during the resting, secreting and restoring state. The ionic concentrations of the plasma and the flowing rate of blood were measured. The ionic concentration and flowing rate of saliva were also measured. The ionic balances of the gland were examined, respectively during the resting, secreting and restoring state, and the ionic balance in the gland tissue was calculated from the measurements of the flowing rate and the concentration of the saliva and blood. Sodium and potassium were measured with the flame-photometer (Lange), chloride by the titrating method of Sharles and Sharles and inorganic phosphate by the Fiske-Subbaraw method.

Ionic concentration of gland tissue. With the dog nephrectomized¹¹, the submaxillary glands on both sides were removed one by one at the resting state and then the secreting state, 30 minutes after an intravenous injection of inulin (0.2 g/Kg of body weight). The secreting state was maintained for five minutes by stimulating the chorda tympani (2 V, 20 cps). After obtaining the wet weight, dry weight of the excised gland, inulin concentrations of the homogenate and plasma were measured to calculate the inulin space of the gland^{11,12,13}. The ionic concentrations in the plasma and gland homogenate were also measured to calculate the ionic content in gland cell.

RESULTS

Intracellular potential of the acinal cell of the perfused gland. When the submaxillary gland was perfused with the perfusate of normal ionic content (arterial blood or TABLE 1, normal Ringer), the intracellular potential of the acinal cell was -40 ± 8 mV at the resting state, on an average of 200 measurements, and was hyperpolarized to -54.5 ± 11 mV on an average at the secreting state provoked by electric stimulation of the chorda tympani. The secreting rate of saliva was 0.02-0.1 ml/min/g. gland. The latency of this hyperpolarization was 0.4 sec. and the rate of its rise was 13-25 mV/sec. The hyperpolarization reached its maximum after 1-2 secs., and then it was maintained at an almost constant level during stimulation. Thus this characteristic feature of the hyperpolarization seems to correspond to the first type of secretory potential described by LUNDBERG with cats' submaxillary gland (see FIG. 2 left side).

FIG. 2. Effect of increased K^+ concentration in the perfusate on the intracellular potential, salivary flow (empty column) and the K^+ -concentration of saliva (shaded column). Horizontal line of the potential record indicates the level of resting potential, while the hyperpolarized curve indicates the secretory potential. K^+ concentration in the perfusate being raised over 13 mM, the secretory potential was completely abolished, while the secretion of saliva remained, though its rate was reduced.

By raising the potassium concentration in the perfusate, by the addition of various amounts of isotonic KCl solution to the arterial blood to be perfused, the intracellular potential was influenced considerably.

When the K concentration in the perfusate was normal, i.e. 4 mM/l, the resting potential was -40 mV and the secreting potential was -54 mV, while they were decreased to -35 mV and -37 mV respectively, on an average, by raising the potassium concentration to 11 mM (FIG. 2 and 5). When the potassium concentration was 13 mM, the resting potential was -34 mV on an average, while no secretory potential appeared by stimulation, i.e. the intracellular potential remained unchanged by stimulation. The more the potassium concentration of perfusate was raised the more the resting potential was decreased, and it was -20 mV at the potassium concentration of 96 mM. Even in such a case of high potassium perfusate, the secretion of saliva still remained, though its rate decreased, as is seen in FIG. 2. The hyperpolarization of the intracellular potential was, however, completely abolished at a higher concentration of potassium than 11 mM in the perfusate. The resting potential, secretory potential and the secreting rate of saliva were able to be restored promptly by returning the potassium concentration to a normal value in the perfusate, even after it was raised to 43 mM.

FIG. 3. A record of intracellular secretory potential of submaxillary gland of which Cl^- in the perfusate was replaced completely with SO_4^- Ringer solution. The secretory potential remained almost normal 15 minutes after perfusion. The resting potential of about -40 mV was hyperpolarized to -50 mV by chorda stimulation and a salivary secretion of $0.01\text{ml/min./g.gland}$ was obtained.

FIG. 4. Relationships between intracellular potential as well as flow rate of saliva and chloride concentration in the perfusate (replaced by nitrate). Open circles indicate the resting potential and solid circles the secreting potential. Columns show the maximum flow rate of saliva.

When the gland was perfused with the potassium free solution, the normal secretory potential was still observed at 15 min. after the start of perfusion, while the rate of salivary secretion was reduced markedly at 5-10 min., and disappeared at 15 min. At that time, the resting and the secreting potential didn't differ so much from those of the control, while the secretory potential disappeared at 20 min. As potassium was detected in 1 mM or more in the perfusate from the vein, and the normal potassium concentration of dogs' red cells is not high, this K in the perfusate may be explained as mainly derived from the gland cell. By losing potassium, probably the excitability of the gland cell was decreased as is generally observed with nerve and muscle cells, and thus the salivary secretion and secretory potential disappeared. By returning the potassium concentration of the perfusate to the normal value, the salivary secretion and the secretory potential were restored gradually and attained the normal state after about 20 min.

On the other hand, the reduction of the sodium concentration to half of the normal plasma value by replacing part of the plasma with Li-Ringer solution (see TABLE. 1) did not cause any appreciable change in the salivary secretion nor in the secretory potential even after 30 min. while a complete replacement with Li-ion abolished it promptly. Thus, the secretion and secretory potential are more sensitive to changes in the potassium than in the sodium in the perfusate. The complete replacement of chloride ion with sulphate ion could not affect the secretory potential until after 15 min., while the salivary secretion was reduced appreciably (FIG. 3). The complete replacement of Cl^- with NO_3^- also abolished the salivary secretion and secretory potential immediately, while its half replacement did not affect them appreciably. In FIG. 4, the resting as well as secreting potential is plotted against the Cl concentration in the perfusate. The secreting potential seems to be affected somewhat by the replacement of Cl^- with NO_3^- . But the potential change is not so sensitive when compared with the influence of the K concentration on the secretion potential as is seen in FIG. 6. The secretion of saliva was reduced by a decrease of chloride concentration in the perfusate. But its dependency on the chloride concentration does not coincide with the curve presented by LUNDBERG⁹.

These results suggest that the chloride ion in the perfusate is not indispensable for the generation of hyperpolarization in the acinal cell membrane by electrical stimulation, and that the secretion does not necessarily accompany the secretory potential, while they are usually concomitant after stimulation.

Ionic balance study of gland tissue. Similar to the experiment by BURGESS¹⁰, the ionic balance of the gland was studied by measuring the ionic concentrations and the flowing rate of arterial, venous blood and of saliva by using the submaxillary gland of the dog.

The balance of all ions in the resting state was almost zero. The flowing

rate of arterial and venous blood through the gland was about 0.2 ml/min./g. gland, and the ionic concentration of the venous plasma was almost equal to that of the arterial plasma. The K-concentration of the plasma was 4 mM/l, Na 130 mM/l, Cl 110 mM/l and inorganic P was 1.85 mM/l.

When the chorda tympani was stimulated electrically, however, the ionic balance of the gland tissue was greatly affected. When the rate of salivary flow was about 0.2 ml/min./g.gland, the K-concentration of saliva was 30 mM/l, Na 110 mM/l, Cl 90 mM/l and inorganic P 0.6 mM/l. The rate of blood flow

FIG. 5. Ionic movement of the gland tissue from or to serum and to saliva during and after chorda stimulation. Blank column shows a gain (positive side) or a loss (negative side) of an ion in the gland tissue from or to serum at the rate of $\mu\text{Eq/g.gland/min}$. Shaded column shows that to saliva. Small circle along the curve shows the time course of the ionic balance in the gland tissue in $\mu\text{Eq/g.gland}$.

increased to 2.0 ml/min./g.gland on a maximum, and the ionic concentration of the venous plasma changed greatly compared with that of the arterial plasma, e. g. K was 9.3, Na 124, Cl 110 and inorganic P 4.5 in mM/l.

The difference of the flowing rate between arterial and venous blood being assumed to equal the rate of salivary flow, the balance of the various kinds of ions in the gland tissue in the resting state as well as secreting state was calculated as follows:

Let V_a , V_v and V_s represent the rate of arterial, and venous blood flow through the gland and the rate of salivary flow, respectively: then the ionic balance of the gland tissue is given by the following equation,

$$(\text{net loss or gain of an ion of gland tissue}) = C_a \times V_a(1 - H_a) - C_v \times V_v(1 - H_v)$$

and,

$$(\text{the rate of loss of that in saliva}) = -V_s \times C_s$$

where C_a , C_v , C_s are the ionic concentration of the arterial and venous plasma, and saliva, respectively, H_a and H_v are the hematocrits of the arterial and venous blood, respectively.

FIG. 5 represents the balances of ions of the gland tissue in the resting state, the secreting state and the state of restoration after secretion. The histogram which stands on the horizontal base line (positive side) represents the input of ions into the gland tissue, while that under the base line (negative side) is the output, and the balance between the two is indicated by a small circle along the curve of balance which represents the time course of the ionic balance in the gland tissue. By algebraically summing up the positive and negative gain of ions in the gland tissue during the stimulation and also during the period of restoration, the ionic balance in the tissue is obtained as indicated by the following table:

TABLE 2. Gain (+) or loss (−) of ions of gland in $\mu\text{Eq/g. gland.}$

a) during stimulation for 1 min.

ion	blood	saliva	balance	sum
K ⁺	−6.0	−7.8	−13.8	−2.3
Na ⁺	+39.0	−27.5	+11.5	
Cl [−]	+28.5	−22.5	+6.5	+6.5

b) for 15 min. after stimulation.

ion	blood	saliva	balance	sum
K ⁺	+13.7	—	+13.7	+2.7
Na ⁺	−11.0	—	−11.0	
Cl [−]	−7.5	—	−7.5	−7.5

It is shown in FIG. 5 that the potassium was transferred the gland tissue into the blood as well as saliva during the secreting state. This fact is described as "potassium transient" by BURGEN¹¹. After the cessation of stimulation, i. e. during the state of restoration, the potassium was taken into the gland tissue from the blood, and the zero balance of potassium was attained 15 minutes after the end of stimulation. As is seen in the table the total balance of K in the gland during stimulation for 1 minute is $-13.8\mu\text{Eq/g}$, while it is $+13.7\mu\text{Eq/g}$ during the restoration period after 15 minutes. Thus, the restoration is completed in 15 minutes. Concerning the sodium and chloride, a large amount of respective ion was taken from the blood and was transferred into the saliva during the secreting state. And some of it was, however, retained somewhat in the gland, while it was driven out to the blood during the restoring state. The zero balance of sodium as well as chloride was achieved almost completely after 15 minutes in the restoring state, as is indicated in the table.

The balance of inorganic phosphate in the gland presented a similar loss to that described above. These movements of ions seem to be coupled with the metabolic processes in the gland. The movement of sodium and chloride seems to be caused by the mechanism of salivary secretion, while the potassium transient may be caused by the increase of potassium permeability of the gland cell membrane in the secreting state.

Ionic concentration of the gland. In attempt to reconfirm the results obtained in the balance study of the gland, the ionic concentration of the gland tissue was analysed in the resting as well as the secreting state. Five dogs were used for the experiment. Na^+ contained in the resting submaxillary gland of the dog was $34 \pm 2.8 \text{ mM/Kg}$, K^+ $65.4 \pm 1.7 \text{ mM/Kg}$ and Cl^- $60.8 \pm 3.6 \text{ mM/Kg}$ tissue weight. The water content of the resting gland was $78.1 \pm 1.4\%$, and the inulin space was 23% of tissue weight. As the concentration in plasma water was 145 mM for Na^+ , 4 for K^+ and 114 for Cl^- , the following intracellular concentration was obtained: Na^+ 3, K^+ 117 and Cl^- 62 mM/Kg H_2O . In this experiment, care was taken not to stimulate the chorda tympani prior to excising the gland. The ionic content of the secreting submaxillary gland of the dog was measured with a gland which was excised after 5 minutes stimulation of the chorda tympani with the current of 2V, 20 cps. The contents of the ions in the gland tissue were as follows, Na^+ 51.6 ± 4.5 , K^+ 48.0 ± 8.7 and Cl^- $58.6 \pm 4.5 \text{ mM/Kg}$ tissue weight. As the water content of the gland was $76.8 \pm 0.2\%$, and the inulin space was 23%, the following average intracellular concentration was obtained: Na^+ 34, K^+ 89 and Cl^- 60 mM/Kg H_2O . Thus, the gland gained $31\mu\text{Eq/g}$ cell water of Na^+ , while it lost $28\mu\text{Eq/g}$ cell water of K^+ during the 5 min. stimulation.

These results almost coincide with that of the ionic balance study, indicating that the gland tissue gained $11.5\mu\text{Eq/g}$ tissue of Na^+ , while it lost 13.8

$\mu\text{Eq/g.}$ gland of K^+ during the 1 minute stimulation. The fact that $6.5\mu\text{Eq/g.}$ gland of Cl^- is gained in the balance study during the 1 minute stimulation, while the balance was almost zero in the gland cell for the 5 minutes stimulation, may be explained by the fact that the tissue fluid was retained in the gland tissue during stimulation.

DISCUSSION

According to LUNDBERG, the hyperpolarization potential of the acinal cell⁶⁻¹⁰ during the stimulation, i.e. the secretory potential originates the active transport of Cl^- ion and initiates the secretion of salt and water through the acinus cell. In the present authors' study, however, it was verified by the perfusion experiments that the salivary secretion is not always concomitant

FIG. 6. Relationship between intracellular potential and potassium concentration in perfusate. Open circles indicate the resting state, and solid circles the secreting state. The height of the vertical line denotes the standard error of the mean, and the figures in parenthesis indicate the number of observation.

with the secretory potential, and the secretion can be detected when the hyperpolarization is abolished by an increased K^+ concentration in the perfusate, while it remains almost normal in the perfusion experiment with SO_4^- -Ringer, when the secretion decreases appreciably. On the other hand it was indicated that the intracellular potential, as well as the secretory potential, was very sensitive to change in the potassium concentration of the perfusate (see FIG. 6). From the experiment of the ionic balance in the gland tissue, it was suggested that the permeability of the cell membrane for potassium increased during the secreting state, and the secretory potential may be explained as due to the equilibrium potential of potassium during the excited state of acinus cells. By adopting the GOLDMANS constant field hypothesis of the equilibrium potential, the membrane potential of the acinus cell, E , may be expressed as follows:

$$E = -\frac{RT}{F} \ln \frac{(K)_o + q(Na)_o + r(Cl)_i}{(K)_i + q(Na)_i + r(Cl)_o}$$

$$q = \frac{P_{Na}}{P_K} \quad r = \frac{P_{Cl}}{P_K}$$

FIG. 7. Graphical calculation of P_{Na}/P_K and P_{Cl}/P_K in the resting state as well as the secreting state. Small circles show the resting state, and triangles show the secreting state.

where $q = P_{Na}/P_K$ and $r = P_{Cl}/P_K$, and P is the permeability coefficient of the respective ion, and suffixes o and i mean "outside the cell and its inside", respectively. This equilibrium equation was applied to the intracellular potential of the submaxillary gland at various states, i.e. the gland perfused with Ringer of which the potassium content was changed from 4 to 13 mM/l. The above equation can be converted into the following general form $Y = qx + r$, where, Y and X can be determined by the above-mentioned experiments with perfusates of different ionic compositions. By means of the method of least square, the parameters q and r were determined as follows (see FIG. 7).

At the resting state, $q = 0.130$; $r = 0.071$

At the secreting state, $q = 0.038$; $r = 0.020$

These data suggest that the rate of increase of potassium permeability to that of Na^+ and Cl^- at the secreting state is about 3.5 times as high as those at the resting state. Thus, it is deduced that the secretory potential may be explained as being due to an increase of K^+ permeability of the cell membrane by which the potential approaches the K^+ equilibrium potential.

When the gland was perfused with the potassium free solution to reduce its intracellular concentration, the secretion of the gland disappeared rapidly, while the secretory potential remained until about 20 minutes after the commencement of perfusion. When the gland was perfused with a high potassium solution, the secretion was decreased, but it remained even with a solution of 96 mM K , when the concentration in the saliva was raised, while the secretory potential disappeared even with the perfusate of 13 mM K . Thus, it is inferred that the secretion of saliva is more sensitive to a loss of intracellular potassium than the secretory potential is, while the salivary secretion is more resistant to the increase of potassium concentration of the perfusate than the secretory potential which is initiated by an increase of potassium permeability of the cell membrane during stimulation.

In any case, the secretory potential is by no means the cause of salivary secretion, while the initiation of the two are sometimes concomitant. As was demonstrated by the electron micrographs of FUJITA et al.²³⁾, the pinocytosis and the basal infoldings along the outer cell membrane of acinus cell increased during the secretion. Thus, the cell drinking mechanism is suggested as being the factor that initiates the salivary secretion. On the other hand, the initiation of the secretory potential seems to be explained by an increased permeability of the cell membrane to potassium ion which makes the intracellular potential approach the equilibrium potential of potassium.

In this connection, it should be mentioned that LUNDBERG experimented with cat's sublingual gland, while the present experiments were performed with dog's submaxillary gland. As the ionic composition of saliva from cat's sublingual gland (mainly composed of Na , K , Cl) is quite different from that of submaxillary saliva of the dog, the above discussion on salivary secretion

may not be applicable to cat's sublingual gland. In the case of dog's submaxillary gland, however, the ionic composition (Na, K, Cl, HCO_3) of precursor saliva taken from the terminal portion of the gland is close to that of serum, as was reported by TOYOKI¹⁴⁾. Therefore the secretion of ions in the terminal portion of gland cannot be explained under the posturation of Cl-pump. If the active transport of individual ions in saliva were to appear in its secretion, many pumps of various ions which act to transport ions so as to produce a tissue fluid like excreta should be postulated. Posturation of such a complicated pump of active transport seems rather ridiculous. Thus, the cell drinking through the acinus cell seems to be the most possible mechanism of secretion of the precursor saliva.

SUMMARY

The perfusion experiments were performed with dog's submaxillary gland and the mechanism of salivary secretion and the initiation of the secretory potential were studied by means of the microelectrode technique. Results obtained are as follows:

1. The intracellular potentials were very sensitive to changes of K^+ concentration in the perfusate. When K^+ was raised over 13 mM/l, the secretory potential completely disappeared, while the secretion was still observed. Even when Cl^- in the perfusate was replaced completely by sulphate, the secretory potential remained practically normal 15 minutes after starting the perfusion, but the rate of salivary flow decreased.

Therefore, the secretory potential is not the causal factor of secretion, and it seems to originate from some other factor than the Cl^- pump along the cell membrane of the gland acinus.

2. From the balance study, it was concluded that the gland lost K^+ with the rate of about $14\mu\text{Eq/g.gland/min.}$ into the blood and saliva during the secretion, while it was restored into the gland from the blood after cessation of stimulation and the restoration was completed in 15 min., Na^+ and Cl^- were retained in the gland tissue en route from the blood to saliva by stimulation, while they were completely driven out and then into the blood during the restoration of 15 min. By analysis of the ionic concentration in the gland tissue, a similar ionic movement was observed. The K^+ in the gland decreased during the salivary secretion, whereas the Na^+ concentration increased.

3. It is clarified by the aid of GOLDMAN's equation that the rate of potassium permeability to that of the other ions increased more markedly in the secreting state than in the resting state. Thus, the equilibrium potential which is manifested by the increase of K permeability during the stimulation of the gland seems to account for the hyperpolarization of the intracellular potential of the gland acinus. The secretion of saliva is not necessarily concomitant

with the hyperpolarization of the cell membrane potential, and should be explained by some other factor than the secretory potential. A possible mechanism of salivary secretion was suggested in the discussion.

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