Effect of stimuli on the secretion of the parotid gland / by P. Butler Stoney.

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5.

EFFECT OF STIMULI ON THE SECRETION OF THE PAROTID GLAND.

By P. Butler Stoney, *late Resident Physician in St Bartholomew's Hospital.*

A healthy young woman having been admitted to St Bartholomew's Hospital for a parotid fistula of long standing, I took advantage of the opportunity to make some experiments on the effect of stimuli of different kinds on the secretion of the parotid gland. The fistula was occasioned by a cut across the cheek received in childhood. The orifice of the duct was about two inches in front of the right ear. It was sensitive, but not particularly tender, and a fine probe could be readily passed up for some distance. There was no communication whatever with the mouth, so that the whole of the saliva secreted was discharged by the fistula and ran down the cheek. The power of various stimuli to increase secretion was estimated by the rapidity with which a drop formed at the fistular opening before and after their application. The method of procedure was as follows :—The duct was emptied by passing a soft napkin gently but firmly along its course from the gland to the orifice which was then completely dried, and the time which elapsed before a drop formed sufficiently large to run down the cheek carefully noted. The duct was then emptied and the fistula wiped a second time, the stimulus applied, and the time required for the formation of a drop again observed. The effect of mastication alone was first tested by giving the patient a glass stopper to chew. The experiments numbered I. II. and III. were all made at one time with a short interval between. The time required for the formation of a drop was in—

Expt.	I.	II.	III.	IV.
Natural secretion without a stimulus	3′	3′15″	4′	
While chewing	1′15″	1′	50″	1′20″
After chewing	2′40″	5′	6′	5′

The effect of taste alone was tried by placing sugar or tartaric acid first on the tip and next on the base of the tongue.

Expt.	I.	II.	III.	IV.
A little sugar placed on the tip of the tongue	3′	5′	12′	
Sugar on the base of tongue	4′15″	5′	12′	4′
Tartaric acid on the tip	10″	20″	30″	1′10″
Tartaric acid on the base	10″	30″	60″	30″

The effect of mastication and taste together was observed by allowing the patient to chew a piece of meat.

	Expt. I.	II.	III.	IV.
No stimulus	2′15″	2′45″	5′	
While chewing	15″	30″	30″	15″

The effect of mental stimuli was first tested by desiring the patient to think of something nice. This had no effect on the secretion whatever, and food was then placed before her.

	Expt. I.	II.	III.	IV.
Food placed before patient	1′13″	4′	6′	12′

These experiments show (1) that mastication alone stimulates the flow of saliva from the parotid to a considerable extent. (2) That the effects of taste vary with the sapid substance, sugar having no effect, while tartaric acid acts most powerfully. (3) That sapid substances act equally when applied to the tip and base of the tongue. (4) That the effect of mastication and taste together is much greater than that of mastication alone. (5) That mental stimuli had a considerable effect in one experiment, but in others none at all. These results accord in some respects with those obtained by Schiff in his experiments on dogs, though differing from them in others. This physiologist observed that mastication alone had little or no stimulating action on the parotid secretion in dogs, and Dr Brunton informs me that he has found this to be the case also in rabbits, while in the experiments above described the action was very distinct. The effect of the application of sugar and tartaric acid to the tongue of dogs was the same as that observed by me. The slight effect of purely mental stimuli in this case is remarkable, as the parotid is stated by Kühne to be readily affected by them, but this may have been due in great measure to the character of the patient, who seemed to be dull and unimaginative. An experiment was also made for the purpose of determining the time required for the absorption of drugs and their excretion by the saliva. For this purpose iodide of potassium was administered, and the saliva constantly tested till it appeared. The time which elapsed between its administration by the mouth, and its appearance in the saliva from the parotid duct, was found in one experiment to be 29 minutes 30 seconds.

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(5A)

ACTION OF DIGITALIS ON THE BLOOD-VESSELS.

By T. Lauder Brunton, M.D., D. Sc., and Adolph Bernhard Meyer, M.D.

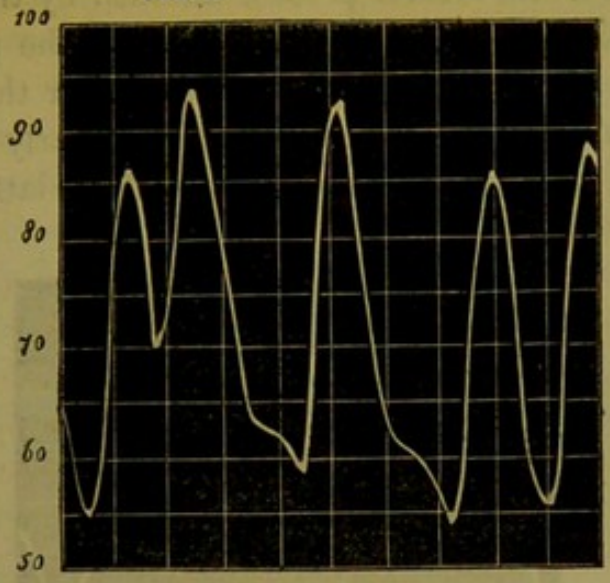
Independently of each other, and in different ways, we both arrived at the conclusion that digitalin causes contraction of the small blood-vessels[1]. Wishing to support our views by still more conclusive proofs, we took advantage of the opportunities afforded to us in the physiological laboratory of the Berlin University to perform together, in February, 1868, some experiments on the subject. We are perfectly aware of their incompleteness, but circumstances having prevented us from continuing them, and the departure of one of us for a distant land rendering it improbable that we shall be able to resume them together, we now publish their results.

We believed that by a comparison of the form of the curves indicating the blood-pressure before and after the injection of digitalin into the circulation, we should be able to determine exactly whether it caused contraction of the arterioles or not. The kymographion we employed was that of Ludwig, as modified by Traube, and the experiments were conducted on dogs in the following manner. The animal being narcotized by hydrochlorate of morphia, a canula was inserted into the crural artery, and a curve (Fig. 1) showing the normal blood-pressure was described. Digitalin, suspended in a small quantity of distilled water, was then injected into the carotid artery, and pressure-curves again described. Injection into the artery was employed because Blake[2] found that digitalin produced a much greater effect on the blood-pressure when introduced into the circulation in this way than if injected into a vein. A comparison of the tracings thus obtained, after the injection, with that of the normal pressure and pulse (Fig. 1), showed a slowing of the pulse, accompanied by an increase in the mean

[1] T. Lauder Brunton *On Digitalis: with some Observations on the Urine*, London, 1868, p. 52, and A. Bernhard Meyer, *Zur Lehre von den Herzgiften in Untersuchungen aus dem physiologischen Laboratorium der Züricher Hochschule.* herausgegeben von Professor Fick. Wien, 1869, p. 71.

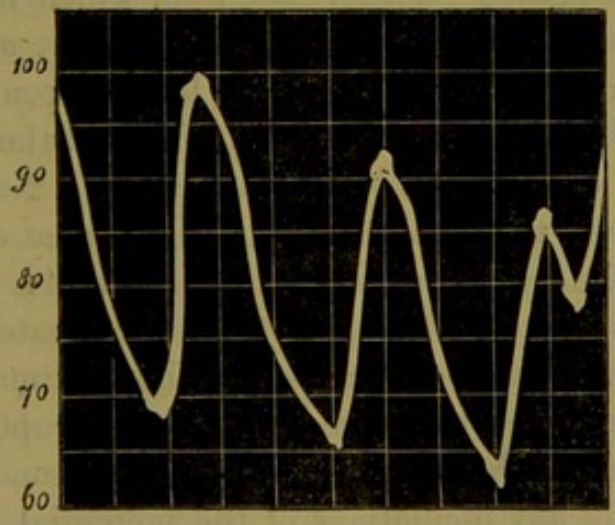
[2] *Ed. Med. Journ.* 1839.

FIG. 1.



blood-pressure, while the height of the wave occasioned by each cardiac pulsation remained much the same (Fig. 2). The pressure continued gradually to rise although the pulse not only became slower and slower, but the oscillations of the mercurial column at each pulsation diminished in extent (Fig. 3). This rise could be due either to the heart propelling a greater quantity of blood into the aorta at each pul-

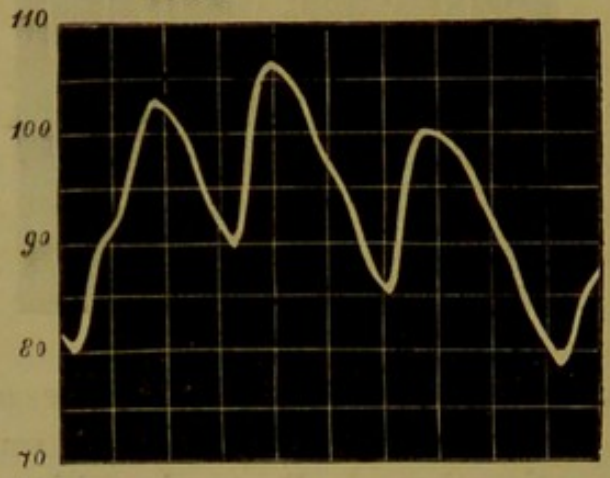
FIG. 2.



sation, or to the arteries, contracting so as to hinder it from escaping from the arterial into the venous system. The diminished height of the pulse-wave seems sufficient of itself to negative the former idea, and to show that the increased pressure can only be due to contraction of the arterioles, but

we think that a still clearer proof is afforded by the form of the wave. The time occupied in the ascent of the pressure-wave (indicated by the horizontal distance between the lowest and highest parts of the ascending limb) is nearly the same in Figs. 1 and 3, but the descending limb of the latter sinks very

FIG. 3.



gradually indeed, while in the former it falls almost as quickly as it rises. What then is the explanation of this phenomenon? During the diastole of the heart, the sigmoid valves when healthy, as they were in this case, completely close the cardiac end of the aorta. The whole arterial system may then be compared to an elongated elastic vessel, from which fluid is issuing by a narrow opening. The greater the pressure of fluid in the vessel the more rapidly will it escape by the opening, the more quickly will the pressure consequently fall, and the more abrupt will be the descent of the pressure-curve. Now the mean blood-pressure in the normal tracing is somewhat over 70 millimetres[1], and the maximum height of the wave 44, while in that taken when the action of the digitalin was greatest, the mean pressure is somewhat over 90 mm., and the maximum 104. The fall of pressure ought, therefore, to be more abrupt, but instead of this it is more gradual. This alteration cannot, we think, be explained by any oscillations of the mercurial column independently of the blood-pressure, and can only be due to contraction of the arterioles retarding the flow of blood from the arterial into the venous system during the cardiac diastole.

[1] The true heights are of course nearly double these, but for convenient comparison with the tracings we have taken the numbers as they stand in the figures.

In a recent paper, Boehm[1] considers that the rise in blood-pressure produced by digitalis, is chiefly due to the increased action of the heart, and that the condition of the arterioles has little or nothing to do with it. He seems, however, to interpret tracings of the blood-pressure in the arteries of mammals in the same way as those obtained from the excised heart of the frog, and apparently forgets that while in the latter the form of the diastolic as well as of the systolic curve depends on the heart alone, in the former the heart can have but little or no influence on the pressure in the arterial system during the diastole, since all communication between them is prevented by the closure of the sigmoid valves. The curves which he gives confirm our views, for they show the same gradual fall in the pulse-wave, after the injection of digitalis, that ours do, and being traced with Fick's spring-kymographion, are free from any fallacies due to oscillations of the mercurial column. The continued high pressure he observed during prolonged stoppage of the heart, and which he attributes to continuous cardiac systole, we would ascribe to contraction of the vessels so far as it is not due to changes in the respiration. If the arterioles were not contracted the pressure would fall, as *e.g.* in the experiments of Ludwig and Hafiz[2].

We next attempted to ascertain whether the slowing of the pulse is due to a direct specific influence of the drug on the roots of the vagus as supposed by one of us[3], or to the stimulation of these roots by the increased pressure of blood in the cranium produced by the contraction of the arterioles, as supposed by the other[4]. In order to do this we diminished the blood-pressure by the inhalation of nitrite of amyl after it had become high, and the pulse slow from the injection of digitalin. If the slowing of the pulse were due to a specific action of the digitalin on the vagus roots, it ought to continue although the pressure falls, but if due to stimulation of these roots by the high blood-pressure, it should disappear whenever the pressure is reduced. Our experiments showed that whenever the pressure fell after the inhalation of the nitrite of amyl the pulse became

[1] *Pflüger's Archiv*, v. 190.
[2] Ludwig's *Arbeiten*, 1870.
[3] Brunton, *Op. cit.*
[4] Meyer, *Op. cit.*

quick. It might thus appear that the slowing is due in part at least to the high pressure, and not altogether to a direct influence of the digitalin on the vagus; but this must be decided by farther experiment.

Lastly, we tried to discover whether digitalis causes contraction of the vessels by acting directly on their walls or on the vasomotor centre. This we sought to do by observing whether the injection of digitalin into the circulation caused any alteration in the calibre of the vessels of the rabbit's ear after the sympathetic nerve of the same side as well as both vagi had been divided in the neck. The vagi were divided in order to prevent the digitalin from slowing the heart, and thus disturbing the circulation, and the sympathetic to prevent any influence being transmitted to the vessels of the ear from the vasomotor centre. The results of these experiments were not constant, and we are unable to draw any definite conclusions from them; but the fact that the vessels of the ears were occasionally seen to empty themselves more quickly after the injection of digitalin than before, seems to us to indicate an action upon the walls of the vessels themselves.

The conclusions to which we have arrived are shortly, 1st, that digitalin causes contraction of the arterioles. This is proved by the small height of the pulse-wave, and by its descent becoming more gradual after the injection notwithstanding the increased blood-pressure. 2nd, that the slowing of the pulse is probably due in part to the increased blood-pressure which results from the contraction of the arterioles.

We gladly take this opportunity of expressing our obligations to Professor Rosenthal for the assistance and advice which he so constantly and kindly afforded us, and to Herr Merck of Darmstadt, to whose kindness we owe the digitalin we employed.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