

LETTERS TO THE EDITOR

SALIVATION IN NORMAL AND AMYGDALECTOMIZED DOGS

A comment on a paper of Łagowska and Fonberg: Salivary reactions in dogs with dorsomedial amygdalar lesions. *Acta Neurobiol. Exp.* 35: 17-26, 1976.

It was a sad experience to find so many errors and misrepresentations of my views in a paper written by Colleagues who used my method of salivary fistula and a stereotaxic instrument of my design. The discussion in Łagowska and Fonberg's paper (6) is so misleading that a detailed commentary is needed. The matter is made more serious by the fact that this neurophysiological study will be read by many brain specialists who would never bother to read papers devoted primarily to the problems of salivary reflexes.

Let me start with the first striking error. On p. 18 the Authors state: "Before the experiments started, a fistula of the parotid gland was made according to the method of Ganike-Kupalov, as modified by Sołtysik and Zbrożyna." Beside the fact that it is fistula of the Stensen's duct and not of a gland (by definition external fistula is an abnormal connection between a *hollow* organ and the external surface of the body), there are following historical errors in this statement. Ganike and Kupalov (5) invented a recording technique which was used for decades in Pavlov's laboratories to measure salivary secretion until its inadequacy was pointed out by Kozak (4) and Makarychev (7). They had not contributed to the method of preparing salivary fistula which is credited to Glinski (9) who, in Pavlov's laboratory, successfully adapted an older Pavlov's operation of pancreatic duct fistula (8), first for the Wharton's duct and later for the Stensen's duct in dog. Thus, this method is correctly called a method of Pavlov and Glinski. The new method, which Zbrożyna and I (14) described in 1957 and which since then has become a standard procedure of salivary studies in the Nencki Institute, is not a modification of Pavlov and Glinski operation. It is, rather, a modification of unsuccessful operation tried by Schiff and others in XIX century, and consists in creating an

orifice of a cut and shortened duct using its own epithelial lining to form a small island of soft and wet tissue around the orifice.

The next and more serious error involves misrepresentation of my recent study "The effect of satiation upon conditioned *and unconditioned* salivary responses" (11) (the underlined words are omitted in Łagowska and Fonberg's paper). On the page 24 the Authors recall my finding that gradual satiation does not significantly alter the rate of unconditioned salivation. Immediately after this they ascribe to me several other findings and explanations that do not exist either in the quoted paper or in any other ones. Let me quote the four sentences from the Łagowska and Fonberg discussion: "The independence of the hunger drive from salivary reactions has been described by Sołtysik (11), who found that satiated dogs consumed food slower than hungry ones and only because of it salivated less during certain units of time. He suggested that their consummatory mechanisms were impaired. However, four of our non-aphagic dogs showed a decrease of salivation although the speed of their eating seemed not to be changed. Our results showed decrease in salivation of dogs with a normal level of food intake, which indicates that consummation of food and salivation are two different mechanisms. Thus Sołtysik's explanation is not sufficient".

The following statements are false:

1. "Satiated dogs consumed food slower...."

I have never stated that satiated dogs eat more slowly. On the contrary, I have warned the reader that "Unfortunately no registration of chewing movements was available to ascertain if the slight differences in URs could be correlated with changed rate of chewing movements" (p. 62).

2. "...and only because of it salivated less...."

Here two parts of the statement are false. First, the dogs did not salivate less. In one dog (Bryś) there was a small but insignificant decrease of the salivation rate as a function of the amount of ingested food. In two other dogs there was no decrease in salivation rate and even a slight increase of unconditioned secretion was found in dog Yapp. My conclusion was clear: "These results show unequivocally that changing intensity of hunger has rather insignificant effect on the rate of unconditioned salivation, even when it is lower than the rate of CR." (p. 62). And again: "The relatively weak effect (of satiation, S.S.) on the salivary UR and so much stronger suppression of the salivary CR might be explained by the fact that the mechanism of 'attentional shift', as postulated above, would operate on telereceptive CSs but not on the taste US." (p. 62).

And secondly, having not found a decrease in the rate of unconditioned salivation I could not have attributed it to the slower rate of eating.

3. "He suggested that their consummatory mechanisms were impaired"

Never, ever had I proposed anything so vague. I am not even certain what the Authors mean, because they use the word "consummation" interchangeably with "consuming" in obvious disregard for the English meaning and Latin derivation of these terms (cf. *consumere*, *consumptus* = to eat or drink, as opposed to *consummare*, *consummatus* = to complete, to fulfill).

4. "Thus Soltysik's explanation is not sufficient"

There is nothing more natural in science that new data and particularly new explanations render the old views obsolete. But which of my explanations was inadequate in the light of new data presented by Łagowska and Fonberg? That decrease of salivation is caused by slower eating? Or that "consummatory mechanisms are impaired" in sated dogs? These are not my findings or statements as I have discussed above. My real statements were quite different: "It is plausible rather to assume that satiation (or decreased hunger) affects salivation indirectly by modifying the behavior during the presentation of CS." (p. 62). Of course, I did not speculate about the unconditioned salivation because I did not observe any alterations in it. But even unconditioned salivation has conditioned components, at least in the form of glandular readiness (1), so it could be diminished as a result of drastic impairment of conditioned secretion. However, I suspect that Authors may have had in mind my and Konorski's more general hypotheses on the central neuronal mechanism controlling food oriented behavior, with its separate "drive" and "consummatory" subdivisions (2, 3, 10, 12, 13). If so, I would like to confront the Łagowska and Fonberg data with this general hypothesis and find out if there is any reason to reject it or revise it.

The main point in the quoted part of the discussion is that after ablation of the dorsomedial portion of the amygdala some dogs had impaired salivary responses while retaining their normal eating pattern. But let us consider this conclusion in the light of their results. The data in Łagowska and Fonberg's paper are presented as means from 10 dogs (although the scores on Fig. 2, which are in thousands of drops of saliva, obviously cannot be arithmetic means for 10 sec. periods as stated in Methods and in the legend to this figure) and no data for individual dogs (except for a dog Melange) are presented. In contrast to statements in the Discussion about normal eating, the Authors describe the effects of operation in the following words: "Five out of 10 dogs were aphagic or hypophagic..." and further: "In the remaining dogs, in spite of no obvious changes in the amount of food intake, disturbances in various patterns of alimentary behavior were observed. Węgorz (which also became cataleptic, S.S.) and Jesiotr vomited after their meals and refused food during

the experimental sessions. Szprotka also refused food offered as a reinforcement during the experiments. Mean body weight for all animals decreased." (p. 20)

In other words, five dogs were aphagic or hypophagic, and three other dogs were "aphagic" in the experimental situation where the measurements of salivation were made. Even the two remaining dogs that did not refuse to eat lost weight.

How could these results justify the conclusion that "Our results showed decrease in salivation of dogs with a normal level of food intake,..."? (p. 24). How was it possible to average the data from 10 dogs within 10 days after operation when the dogs refused to eat? What does the US column on Fig. 2 represent: salivation in 10 seconds when the dogs were supposed to eat but did not? The Authors made an exception for one dog, Melange, and presented separately the data from the 10 days after operation; and this was the dog which had "The most pronounced aphagia (14 days), followed by several weeks of hypophagia,..." (p. 20). If these words are to be taken seriously, that means that the Authors consider as postoperative unconditioned salivation the salivation in the presence of food and not the salivation elicited by the taste of masticated food. If the dogs were severely impaired in their feeding behavior, the comparisons of unconditioned salivation before and after operation are not really justified because after amygdectomy, it was not unconditioned salivation (at least in 80% of dogs) but "natural conditioned salivary reflex" to the sight of food, that was measured in the 10 sec following the presentation of food. If so, then no discussion of the possible explanation of this discrepancy between motor and salivary components of alimentary consummatory response is needed.

But let us assume, that immediately after operation in some dogs there was no impairment of motor mechanisms of eating and the appetite was normal (as would be proved by normal weight, at least), but still the salivary responses were suppressed. That would indicate that the lesion affected neural mechanism controlling the salivary reflexes selectively. This is not impossible and it would be very interesting indeed. Even if we think of salivation as a component of an integrated act of eating, since there are separate peripheral nerves innervating the salivary glands and masticatory muscles and there are separate nuclei and pathways in the brain stem, there can be no question that a lesion in the CNS could selectively affect one function (secretory) while leaving intact the other (motor). Salivation is a component not only of eating, but also accompanies rejection of inedibles, vomiting, thermoregulatory panting, and possibly other behavioral (e.g., sexual) acts. Therefore, it could have separate representation even in the higher neural structures such as the amygdala.

Would such a selective post-lesion impairment of salivary secretion constitute a basis for revising our model of "alimentary brain"? I do not think so, because the hypothetical structure of drive and consummatory centers does not require that the salivary and motor functions are controlled from the same loci in the brain.

The paper of Łagowska and Fonberg contains very interesting results, and after reading it one would like to know more about the effects of dorsomedial amygdectomy on individual dogs and on different aspects of their feeding behavior. However, the way in which the Authors (mis)treated their own data and the manner in which they discussed other authors' results and views leave the reader in disappointment, or rather, in a state which our Late Teacher might have called anti-consummation.

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