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AN ATTEMPT AT MODELLING OF THE CENTRAL ALIMENTARY SYSTEM IN HIGHER ANIMALS

IV. EXPERIMENTS ON CLASSICAL CONDITIONING

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In the preceding papers of this series (Konorski and Gawroński 1970, Gawroński and Konorski 1970ab) a concept of organization of the central alimentary system was proposed, and its model reproduced my means of the artificial nerve-net. In the present paper we shall present the results of the basic experiments on classical food conditioned reflexes (FCRs) performed on this model.

METHODS AND EXPERIMENTAL PROCEDURE

The diagram of the organization of the alimentary system is presented in Fig. 1. It may be seen from this figure that it is composed of two subordinate systems denoted as the feeding system and the hunger-satiation system respectively.

The feeding system consists of two reciprocally related subcenters denoted as F subcenter and ∼F subcenter; the former is activated by the food unconditioned stimulus (FUS) represented by the taste of food in the mouth (T), the latter, by the ∼FUS represented by absence of food in the mouth (∼T). Beside this each of these subcenters may be activated by the food conditioned stimulus (FCS) and no-food conditioned stimulus (∼FCS) respectively. Activation of the F subcenter produces the salivary response.

The hunger-satiation system consists of the satiation center (Sat) stimulated by the nutritive substances in the blood and hunger center composed of on-hunger subcenter (H) and off-hunger subcenter (∼H). Both subcenters are reciprocally related and both are under the inhibitory influence of the satiation center. The H subcenter is activated by a hunger conditioned stimulus represented by the experimental situation (ΣHCS).

In our experimental set-up the FUS is represented by a constant quantum of food of the optimal taste ("pellet"). The ∼FUS is also considered constant and is in operation throughout the experimental session, except the moments of the action of the FUS. On the other hand, the FCSs may be variable and possess various degrees of strength measured on a voltmeter. The degrees used are: 11, 13, 16, 19. CS 11 is usually subliminal or nearly so; CS 13 represents a weak CS, CS 16, a moderate one, and CS 19, a strong one. The strength of FCS 19 is almost equal to the strength of the US.

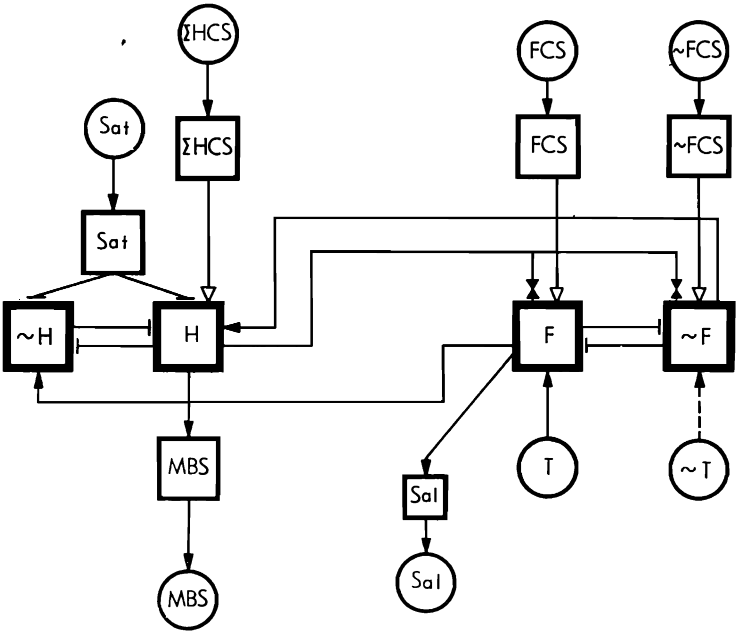

Fig. 1. Model of the alimentary system. Squares, centers; circles, receptors or effectors. H, ~H, on- and off-hunger subcenters; Sat, satiation center; F, ~F, food and no-food subcenters; T, ~T, on and off-taste receptors; ΣHCS, hunger CS; MBS, motor behavioral system; FCS, ~FCS, food and no-food CSs; Sal, salivation. Black triangles, excitatory unconditioned connections; white triangles, excitatory conditioned connections; double triangles, facilitatory connections; stopped lines, inhibitory connections; dashed line denotes adaptive connection.

The levels of satiation were also arbitrarily chosen and five grades were selected denoted on the scale of a voltmeter as 0, 5, 10, 13 and 15. Sat 0 means a complete lack of satiation, that is, maximal humoral hunger. Sat 15 means that satiation is relatively high (that is, the humoral hunger is relatively low), but not as high as to completely inhibit the hunger center. The three other degrees of humoral hunger are intermediate.

The HCS is considered constant because it is represented by the experimental situation (ΣHCS). Accordingly, it is in operation throughout the experimental session.

The interconnections between the feeding system and hunger-satiation system are also shown in Fig. 1. Briefly, the F subcenter has excitatory connections with the ~H subcenter, and the ~F subcenter has excitatory connections with the H subcenter. The H subcenter has facilitatory connections with both F and ~F subcenters. These subcenters cannot be activated without this facilitatory influence.

As noted in the preceding papers of this series, we have a direct access to all four subcenters and can measure their excitation. Excitation of the F subcenter (which may be considered a measure of salivation in arbitrary units) represents the magnitude of the FCR or the FUR; excitation of the H subcenter represents the magnitude of hunger; excitation of the ~F subcenter denotes the strength of the perception of "no food in the mouth", and, finally, excitation of the ~H subcenter denotes the intensity of the satisfaction (pleasure) produced by the FUS or FCS. All these data are recorded in impulses per unit of time fired by the given subcenter. It should be remembered that each subcenter represents a set of units of indentical functional significance.

In our first experiment we shall try to replicate a typical Pavlovian experiment on FCRs, in which besides the measurements of salivary responses (expressed by F) also the functional states of the subcenters ~F, H and ~H will be recorded. Thereafter, the results of experiments with slightly modified weights of particular connections will be presented.

A TYPICAL EXPERIMENT WITH CLASSICAL CONDITIONING (EXPERIMENT I)

A typical experiment with classical CRs, when all the connections between the corresponding subcenters were appropriately balanced, is represented in Table I and in Fig. 2 and 3.

The data obtained in intertrial intervals are shown in the first columns of numbers of each tablet in Table I and in Fig. 2*a*. It may be seen that F values and ~H values are zero, because the F subcenter is silent and does not fire impulses to the ~H subcenter. In consequence, the ~F subcenter stimulated by the ~T receptors and reciprocally related to the F subcenter is maximally excited. For the same reason the H subcenter, being reciprocally related to the ~H subcenter, is also maximally excited, since the HCR is supposed to be established to the experimental situation. The excitation of the H subcenter fully depends on the humoral hunger and the slope of the regression of H values on Sat values reflects the increasing inhibitory influence of the satiation center upon the H subcenter. As regards ~F values, they are influenced by the facilitatory action of the H subcenter upon ~F subcenter and, therefore, they also diminish with the increase of satiation although the slope of the regression is not so steep. This is because of the strong stabilizing influence of ~T receptors upon the ~F subcenter.

Quite opposite is the effect of food in the mouth (FUS) represented in the last but one columns of each tablet of Table I and in Fig. 2*b*. Here the ~F subcenter is silent because ~T receptors are not stimulated. In consequence, the F subcenter is maximally excited for the given satiation level, being free from inhibitory influence of the ~F subcenter. This excitation leads to a strong activation of the ~H subcenter through F–~H connections. This in turn produces strong inhibition of the H subcenter.

Table I

Experiment I

F

S \ Sat	0	CS 13	CS 16	CS 19	us	Sum
0	0	24	25	29	42	120
5	0	23	28	31	42	124
10	0	15	24	28	35	102
13	0	0	15	23	29	67
15	0	0	12	16	22	50
Sum	0	62	104	127	170	463

~F

S \ Sat	0	CS 13	CS 16	CS 19	us	Sum
0	78	67	50	29	0	224
5	78	70	49	31	0	228
10	71	57	46	28	0	202
13	45	45	30	11	0	131
15	14	14	14	0	0	42
Sum	286	253	189	99	0	827

H

S \ Sat	0	CS 13	CS 16	CS 19	us	Sum
0	1020	520	227	59	42	1868
5	922	570	185	62	42	1781
10	462	274	160	56	17	969
13	130	190	60	23	14	417
15	14	14	12	11	11	62
Sum	2548	1568	644	211	126	5097

~H

S \ Sat	0	CS 13	CS 16	CS 19	us	Sum
0	0	38	75	145	313	571
5	0	20	70	95	225	410
10	0	13	24	45	120	202
13	0	0	15	23	75	113
15	0	0	12	10	45	67
Sum	0	71	196	318	778	1363

S0 denotes intertrial interval.

Although the H subcenter is strongly inhibited by the ~H subcenter, its excitation does not reach the zero value. If it did, the F subcenter would be momentarily silenced (because of the lack of facilitatory influence of the H subcenter), the ~H subcenter would cease to be excited and the H subcenter would regain its strong activation.

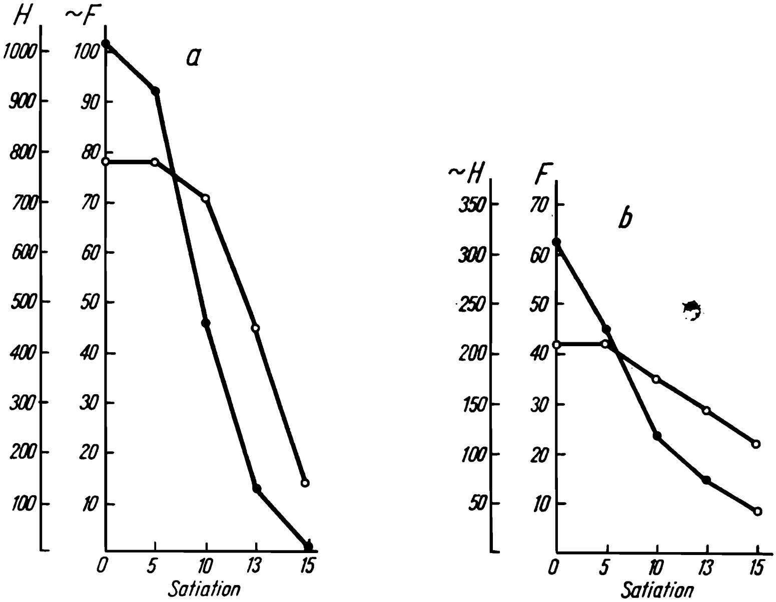

Fig. 2. Experiment I. *a*, H values (black circles) and ~F values (white circles) during intertrial intervals at various levels of satiation; *b*, ~H values (black circles) and F values (white circles) in response to FUS at various levels of satiation.

As seen from Table I, during the operation of the FUS all three active variables — F, ~H and H — decrease monotonously with the increase of satiation. The steepest decrease concerns the ~H values (Fig. 2*b*). This is due to the fact that with the increase of satiation the ~H subcenter is directly inhibited by the satiation center and in addition it is less strongly stimulated by the diminishing activation of the F subcenter. On the other hand, the decrease of the F values with increasing satiation is insignificant, because it directly depends on the decrease of activation of the H subcenter which is under the influence of two opposite factors: the increase of satiation directly attenuates the activation of the H subcenter, but also releases it more and more from the inhibitory influence of the ~H subcenter. The stabilizing role of the F– ~H–H–F loop will be substantiated by the next experiment.

We turn now to the analysis of the data obtained in response to the

FCSs of various strength under various levels of humoral hunger (Table I, columns second, third, and forth of each tablet, and Fig. 3).

As we look at first at the F values representing food conditioned responses (upper left tablet and upper left graph of Fig. 3) we may observe the following rules.

1. The Pavlovian law of strength of the CSs is fully obeyed: the F response to the weak CS (13) is smaller than that to the moderate one (16), and the response to the moderate CS is smaller than that to the strong one (19). This is true for al the levels of humoral hunger.

2. The law of the strength of the CSs is more conspicuous under the moderate humoral hunger than under the strong one. In fact, when satiation is zero the F responses are large and almost equal (the so called equalization phase at the high level), whereas under stronger satiation they are smaller and their differences are more manifest.

3. Decrease of humoral hunger much more strongly affects the weaker stimuli than the stronger ones. If we take, for instance, CS 13 its effect drops with the increase of satiation from 24 to 0, whereas for CS 19 this effect drops from 29 to 16.

4. The magnitude of the CR to the strong CS does not attain the value of the UR. This is explained by the fact that whereas during the operation of the FUS the ∼F subcenter is silent, during the operation of the CS this subcenter is active and it partially suppresses the effect of the CS. This is not true for all dogs and in Experiment III we shall show the instance when these two values are equal.

If we turn now to the analysis of the H and ∼H values observed during the operation of CSs, we may observe that both these values decrease dramatically with the increase of satiation (lower tablets of Table I and Fig. 3, lower graphs), because of the inhibitory effect of satiation center. On the other hand, the dependence of H and ∼H values on the strength of the CS is the reverse to each other: the ∼H values increase with augmentation of the strength of the CSs, while the H values decrease (Fig. 3, lower graphs). These relations are due to the fact that the stronger the CS, the stronger the activation of the F subcenter, hence the stronger the activation of the ∼H subcenter, hence the stronger its inhibitory influence upon the H center. On the contrary, when the CS is subliminal or does not operate at all, activation of the H subcenter is maximal, whereas the activation of the ∼H subcenter drops to zero.

To end this analysis we turn to the discussion of the ∼F subcenter (upper right tablet of Table I and Fig. 3, upper right graph). It should be reminded here that because the reciprocal relations between the F and ∼F subcenters are not "stiff" but "soft" (Gawroński and Konorski

1970b), nearly always we observe the compromises between them, so that they are both activated with the relative dominance of one of them. The zero value of ~F may be due either to the lack of activation of the ~F

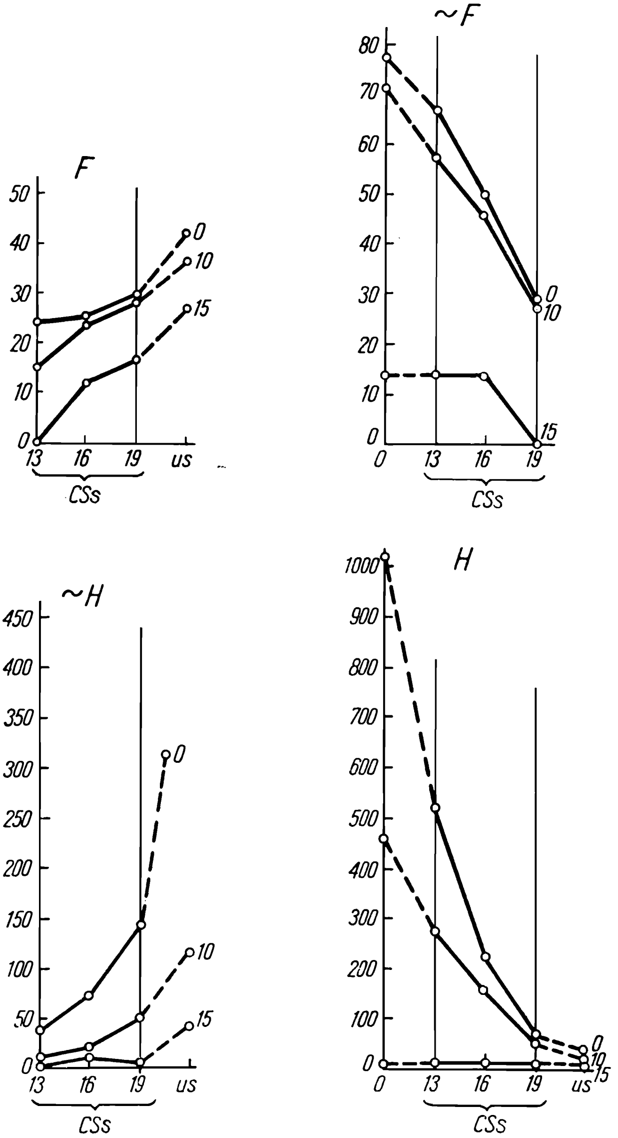

Fig. 3. Experiment I. Dependence of F, ~F, ~H and H values on the strength of CSs at Sat 0, 10 and 15. Abscissae, strength of CSs and US (0, intertrial interval). Ordinates, values of responses. In each graph: upper line, Sat 0, middle line, Sat 10, lower line, Sat 15.

subcenter because of the silence of the ~T receptors when food is in the mouth, or to inhibition of the ~F subcenter by overwhelming activity of the F subcenter.

In the present experiment complete inhibition of the ~F subcenter occurred only once (at CS 19 and Sat 15). In all other cases both F and ~F subcenters were activated with the clear dominance of the ~F subcenter. As we shall see in Experiment III this dominance of the ~F subcenter is not a general rule, because it depends on the weight of the ~T– ~F connections.

As it may be seen from Fig. 3, all the relations between our dependent and independent variables, whether increasing or decreasing, were monotonous. Taking the high complexity of the interrelations between the subcenters involved this is not a general rule, and in our later experiments we shall see many "anomalies" of these relations. In the present experiment the only slight "anomaly" was observed in the values of F, ~F and H obtained with CS 19 (Table I, fourth columns). They were slightly higher at Sat 5, than at Sat 0. This anomaly remained at CS 16 but it concerned only the F responses. These irregularities, which cannot be easily explained, give a hint that if we obtain similar irregularities in "true" CR experiments on normal dogs, this is not necessarily due to some accidental extraneous influences, but to the complex interrelations between the centers involved in conditioning (cf. Pavlovian "paradoxical" and "ultraparadoxical" phases).

The present experiment is only one of several experiments performed by us on Dewan, in which the weights of connections between various subcenters were given slightly differing values. Nevertheless, in spite of the fact that in each of these experiments the values for each dependent variable were different, the general rules presented in Experiment I were strikingly preserved. This indicates that the system has a strong internal stability which resists small modifications in the parameters. We are able, however, to overthrow this stability when a considerable change is introduced, even in only one connection. This fact will be shown in the two following experiments.

Inspecting all the data obtained in Dewan in Experiment I one is struck by their similarity with those, obtained in CR experiments on normal dogs. Here we shall list some of them.

1. During the intertrial intervals the level of hunger is usually very high, which is manifested by the general motor excitement of animals. This level of hunger falls down immediately at the onset of the FCS, when the animal calms down and looks intently at the feeder. Only if the CR training lasts for a very long time, does the situation change because the intertrial intervals become the inhibitory HCSs (cf. Konorski 1967

Chapter VI). This may eventually lead to the deterioration of FCRs because, according to our model, when the strength of the ΣHCS drops to zero, the facilitatory effect of the H subcenter upon the F subcenter disappears. The reluctance or refusal to take food during the CR session is one of the symptoms of this state. Of course, modelling such a state on Dewan would not present any difficulty.

2. The dependence of the magnitude of FCRs on the degree of satiation, and the particular sensitivity to satiety of reflexes to weak and not to strong CSs is another well documented fact (cf. Maiorov 1954, Chapter XVII).

3. The dependence of the magnitude of CRs on the strength of CSs is again one of the generally observed facts. This principle is best manifested when the hunger is moderate, whereas with high hunger we have to do with the "equalization phase at the high level" (Pavlov 1940, Maiorov 1954).

4. In many dogs the conditioned response to a strong CS is smaller than that to the US. In some animals, however, the FCR to a strong CS is equal to that to the US. The model of such dog will be presented in Experiment III.

The seeming difference between Dewan and the normal dogs is that in the latter ones the FUR hardly depends on the levels of satiation, at least within their large range. As a matter of fact, in Dewan, too, the UR was the same under Sat 0 and Sat 5, and under Sat 10 it was only slightly reduced. The reduction was significant only at Sat 13 and Sat 15. The difference between Dewan and normal dogs in this respect, if it really exists, may be due to the mediation in the FUR of the lower order salivary center (in the medulla), apparently independent of the intensity of hunger. Of course, we could easily build in this center in Dewan.

WEAKENING OF INHIBITORY CONNECTION BETWEEN ~H AND H (EXPERIMENT II)

The ideal food for CR experiments should fulfill two conditions: it should be tasty, that is, should evoke a strong activation of the ~H subcenter, and should give a subject a full satisfaction, that is, should inhibit the H subcenter. The slimy tasty food obviously fulfills these requirements.

On the other hand, if a dog receives bones or crackers, which require a hard work of grinding before they are transformed into a bolus, the situation is different: the consummatory act of mastication is performed under the influence of a strong hunger, otherwise the animal could not swallow the presented morsel. In such a situation the animal cannot indulge in a passive "satisfying state of affairs", as it is when the food can be immediately consumed. There is a pleasure because the food is tasty, but the hunger is not inhibited. We may assume that such a situation is modelled by weakening the inhibitory bond linking the ~H subcenter with the H subcenter. This modification was made in Dewan and the results of this experiment (Experiment II) are presented in Table II, and Fig. 4.

Table II
Experiment II

F

S Sat	0	CS			us	Sum
		13	16	19		
0	0	29	48	68	91	236
5	0	27	43	62	77	209
10	0	16	20	27	40	103
13	0	0	20	22	29	71
15	0	0	9	9	18	36
Sum	0	72	140	188	255	655

~F

S Sat	0	CS			us	Sum
		13	16	19		
0	81	68	49	40	0	238
5	80	67	48	38	0	233
10	68	54	40	18	0	180
13	28	27	21	21	0	97
15	9	9	9	8	0	35
Sum	266	225	167	125	0	783

H

S Sat	0	CS			us	Sum
		13	16	19		
0	968	434	143	100	93	1738
5	874	410	135	88	73	1580
10	346	209	28	20	20	623
13	42	40	21	14	15	132
15	9	9	9	9	9	45
Sum	2239	1102	336	231	210	4118

~H

S Sat	0	CS			us	Sum
		13	16	19		
0	0	116	536	780	910	2342
5	0	85	430	641	770	1926
10	0	17	39	81	240	377
13	0	0	21	21	118	160
15	0	0	9	9	52	70
Sum	0	218	1035	1532	2090	4875

S0 denotes intertrial interval.

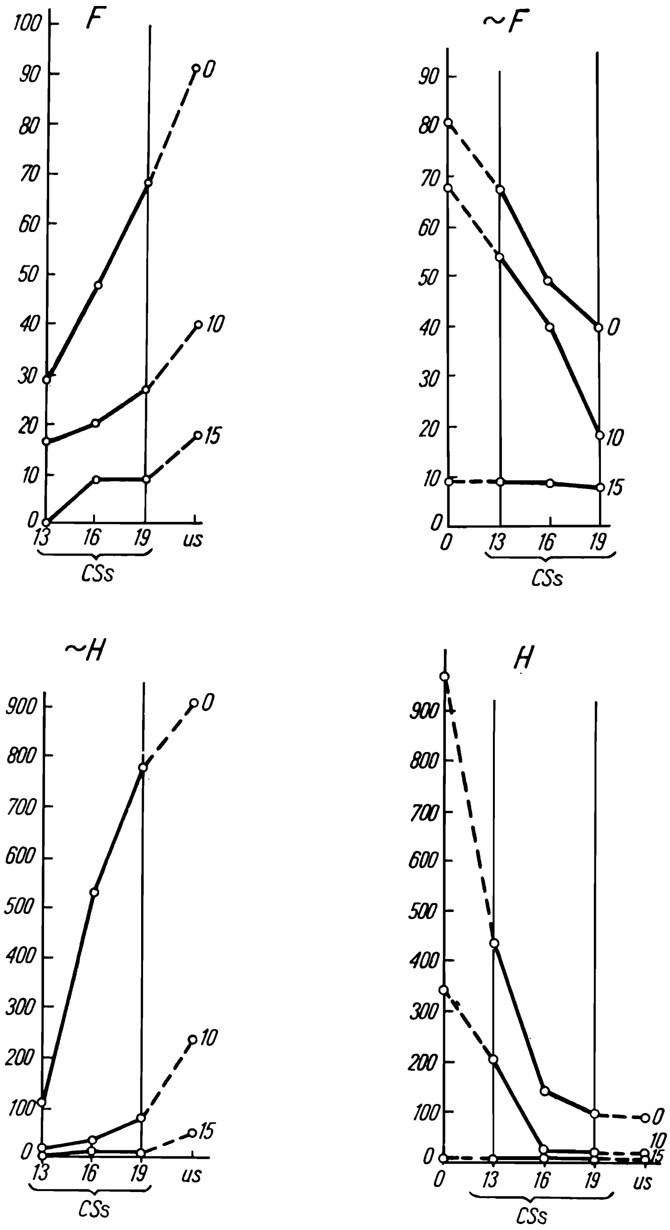

Fig. 4. Experiment II. Explanations as in Fig. 3. Note that the scale in the lower left graph (~H values) is twice smaller in Fig. 4 than in Fig. 3.

A cursory comparison of Table II and Table I (or Fig. 3 and 4) reveals that this modification produced far-reaching changes in the whole CR activity. For the sake of clarity the comparison of the data obtained in Experiments I and II are shown in Fig. 5 and 6.

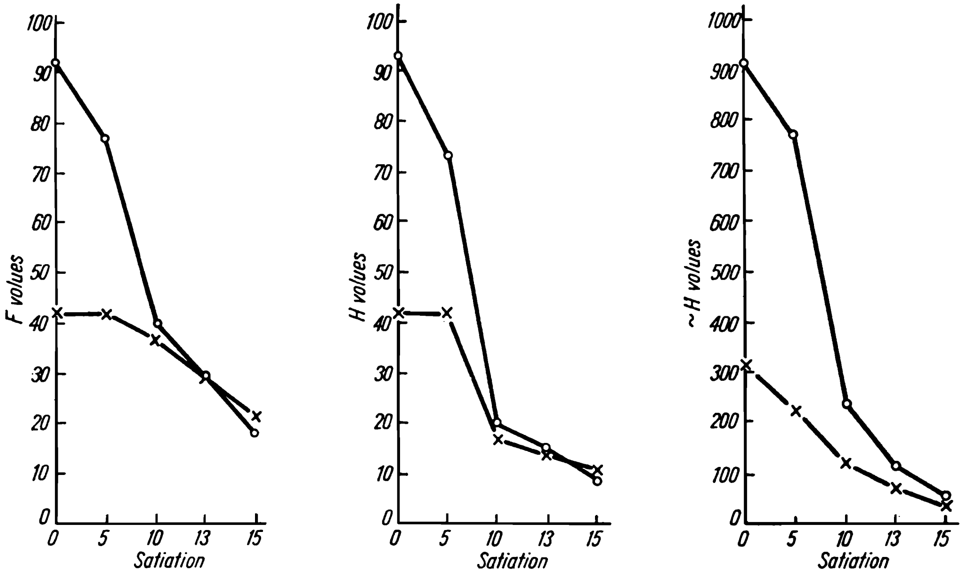

Fig. 5. The comparison of responses to the FUS in Experiment I (crosses) and in Experiment II (circles). Abscissae: the levels of satiation; ordinates: left graph, F values, middle graph, H values, right graph, ~H values.

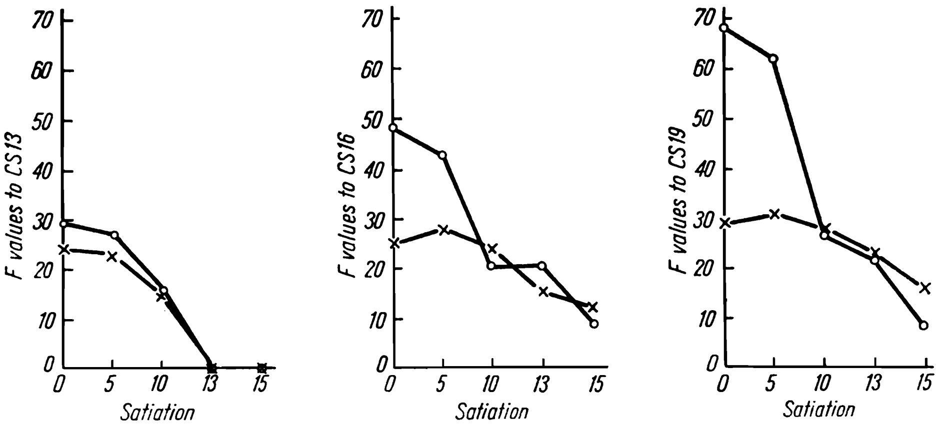

Fig. 6. The comparison between FCRs in Experiment I (crosses) and in Experiment II (circles). Abscissae: the levels of satiation; ordinates: F values. Left graph, CS 13, middle graph, CS 16, right graph, CS 19.

In Fig. 5 the comparison of responses to the FUS is presented. It may be seen that in Experiment II at the highest humoral hunger (Sat 0) there is a considerable increase of F values, H values and ~H values. Almost the same increase is observed at Sat 5, a very small increase is observed at Sat 10, whereas at Sat 13 and Sat 15 all values are the same in both experiments.

The fact that the F values are considerably increased at the high levels of humoral hunger in Experiment II and remain unchanged at the low levels radically changes the slopes of the corresponding curves: whereas the relation between the FUR at Sat 0 and at Sat 15 in Experiment I is roughly 2:1, it grows to 5:1 in Experiment II.

If we turn now to the comparison of responses to FCSs in both Experiments (Fig. 6), we may see that with regard to the strong CS (19), the picture is roughly the same as that with regard to the FUS: there is a considerable increase of responses at the high level of humoral hunger and no increase at all at the low level. The increase of responses is, however, less pronounced with regard to the moderate CS (16) and it is quite insignificant with regard to the weak CS (13). These relations are represented in Fig. 6 only for F values, but they are analogous for H values and ~H values.

Since the increase of FCRs at the high humoral hunger concerns mostly the response to CS 19 (68 instead of 29), in a lesser degree the response to CS 16 (48 instead of 25) and only insignificantly the response to CS 13 (29 instead of 24), the equalization phase so well pronounced in Experiment I at Sat 0 gives way to a well pronounced principle of strength of the CSs in Experiment II (Fig. 4, upper left graph).

To sum up, we see that weakening of the inhibitory H— ~H bond produces strong increase of the F values, ~H values and H values; this increase is, however, observed only at high humoral hunger (Sat 0 and Sat 5) and in response to the US and to strong and moderate CSs. On the other hand, when the humoral hunger is low, or the CS is weak no increase of these values is observed.

How are these data to be explained?

The weakening of the inhibitory connection between the ~H subcenter and the H subcenter obviously leads to a partial release of the H subcenter from the suppressing influence of the ~H subcenter. Accordingly, in all those cases in which the ~H subcenter is in operation, that is when the F subcenter is excited, excitation of the H subcenter must be higher than in Experiment I. This increased excitation produces in turn stronger facilitation of the F subcenter, and this leads to a stronger excitation of the ~H subcenter.

It is clear that all these effects are most pronounced when the stimulation of the F subcenter is strong, because this leads to strong excitation of the ~H subcenter, which fails to inhibit significantly the excitation of the H subcenter and the facilitation of the F subcenter. On the contrary, when the stimulation of the F subcenter is insignificant, as is the case with weak FCS or with weak humoral hunger, then the excitation of the ~H subcenter is weak and therefore the experimental condition are roughly the same as in Experiment I. Thus we see that the inhibitory bond ~H–H plays normally a most important stabilizing role for the whole circuit and restrains its links from excessive excitation. In this way both the virtual "equalization phase" of FCRs under the high humoral hunger, and the weak dependence of the FUR (and FCR to a strong FCS) on the humoral hunger observed in Experiment I are now understood. We can easily overturn these relations by mere weakening the ~H–H inhibitory bond.

To end this discussion we should consider the effect of the weakening of the ~H–H bond on the activity of the ~F subcenter. Comparing Fig. 3 and 4, upper right graphs (or Tables I and II, upper right tablets), we may notice that the ~F values in Experiment II are practically the same as those in Experiment I; if there are some changes, they have an irregular, unpredictable character. This result may seem to be paradoxical in view of the fact that the strong increase of F values produced by facilitation of the F subcenter should rather suppress the ~F subcenter.

The solution of this seeming paradox is this. Facilitation produced by the H subcenter influences not only the F subcenter but also the ~F subcenter. In consequence, the net activation of the ~F subcenter is increased and therefore its resistance to the inhibitory influence of the F subcenter is augmented. Certainly, by manipulating the weight of the F– ~F inhibitory bond we could obtain some other results than those observed in Experiment II, but this was not the aim of our experimentation.

What is the correspondence between the results obtained in Experiment II and those which are observed in normal dogs? Unfortunately, the problem of the relation between the consistence of food (sliminess versus hardness) was not systematically investigated. According to the data obtained in this experiment we should predict that if exactly the same food is given in big morsels requiring grinding or in the shape of minced pulp, hunger will be stronger with the former food and the salivary reflexes (both conditioned and unconditioned) should be larger. Intuitively it seems that it is just so. Incidentally, one would note that the FUR, which in Experiment I was largely independent of the level of satiation, here depends very much on this level. It would be interesting to see whether the same is true in normal dogs.

WEAK BOND BETWEEN ~T AND ~F (EXPERIMENT III)

It is a well known fact that whereas in many dogs the magnitude of the FCRs to strong CSs is always lower than that of the FUR (as is the case in Experiments I and II), in others both magnitudes are equal. According to our present analysis the systematic difference between the FCR and FUR is due to the fact that during the operation of the US the ~F subcenter is silent, while during the operation of the CS it is activated and partially suppresses the F subcenter. Accordingly, we have decided to make a model of a dog in which the activity of the ~F subcenter would be diminished by weakening the weight of the connection between the ~T and ~F. We used for this aim the model in which all other connections were more or less the same as in Experiment II (Table II and Fig. 4); in this model the connection ~H–H was relatively weak, simulating the food reinforcement which does not strongly inhibit the H subcenter.

The results of this experiment are presented in Table III and Fig. 7. Comparing Table III with Table II and Fig. 7 with Fig. 4 we may see that the consistent differences between Experiment III and Experiment II concern ~F values, F values and ~H values. The H values are in both experiments similar.

As seen in Fig. 8, the ~F values in Experiment III are strongly reduced in comparison with those in Experiment II. In Experiment II the ~F subcenter was never completely inhibited; in Experiment III its total inhibition is observed in 11 cases out of 20 ~F items. Zero values are observed: in response to CS 19 at all satiation levels, except Sat 0, in response to CS 16 at satiation levels 10, 13, 15, and in response to CS 13 and CS 11 at satiation levels 13 and 15. In other words, the higher the satiation or the stronger the CS, the greater the probability of the complete suppression of the ~F subcenter.

As far as the F responses to the CSs are concerned, almost all of them are increased in Experiment III in comparison to Experiment II (Fig. 9); this increase, however, does not concern FURs. The extent of the increase of F values is not visibly related either to the levels of satiation or to the strength of CSs. It should be also noted that whereas in Experiment II the CS 11 was completely subliminal as far as FCR is concerned, in Experiment III it produced the conditioned response under high humoral hunger (at Sat 0 and 5) (Table III, upper left tablet).

Another important difference between Experiment III and Experiment II with regard to the F values is this: in Experiment II the F responses to FUS were always larger than those to the CS 19 (Fig. 4, upper left graph or Table II, upper left tablet); in Experiment III this difference remains only for Sat 0 (Fig. 7, upper left graph or Table III, upper left tablet). Examining the ~F responses to CS 19 (Table III, upper right tablet), we see immediately that only at that level is the ~F

Table III

Experiment III. Weakening of the ~T– ~F bond

F

S / Sat	CS				us	Sum
	11	13	16	19		
0	13	35	62	84	96	290
5	11	33	58	87	90	279
10	0	19	35	38	40	132
13	0	0	22	28	29	79
15	0	0	8	17	18	43
Sum	24	87	185	254	273	823

~F

S / Sat	CS				us	Sum
	11	13	16	19		
0	64	51	33	22	0	170
5	62	50	31	0	0	143
10	54	38	0	0	0	92
13	0	0	0	0	0	0
15	0	0	0	0	0	0
Sum	180	139	64	22	0	405

H

S / Sat	CS				us	Sum
	11	13	16	19		
0	940	390	150	124	130	1734
5	790	362	140	86	96	1474
10	390	196	51	20	20	677
13	51	35	20	15	15	136
15	9	9	8	9	10	45
Sum	2180	992	369	254	271	4066

~H

S / Sat	CS				us	Sum
	11	13	16	19		
0	7	195	661	841	870	2574
5	6	150	515	805	814	2290
10	0	28	94	179	220	521
13	0	1	21	79	103	204
15	0	0	10	35	45	90
Sum	13	374	1301	1939	2052	5679

Note that CS 11 which was subliminal in Experiments I and II and therefore was not included in Tables I and II is now supraliminal for Sat 0 and Sat 5. It is included in this table instead of 0 value of the CS (intertrial interval).

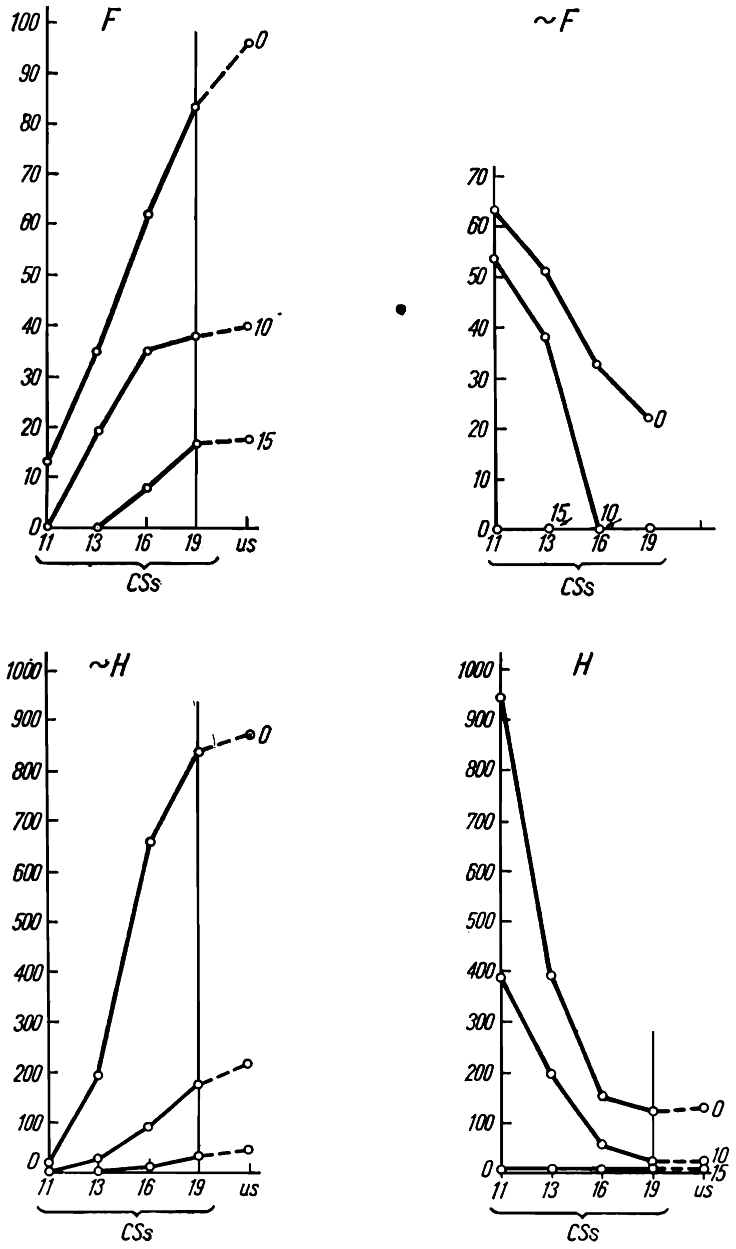

Fig. 7. Experiment III. Attenuated bond between ~T and ~F. Explanations as in Fig. 3.

value larger than zero. (To be precise all other F responses to the CS 19 are a little smaller than those to the US, but this is due to the fact that the stimulation of the F subcenter by the CS 19 was a little weaker than that by the taste stimulus).

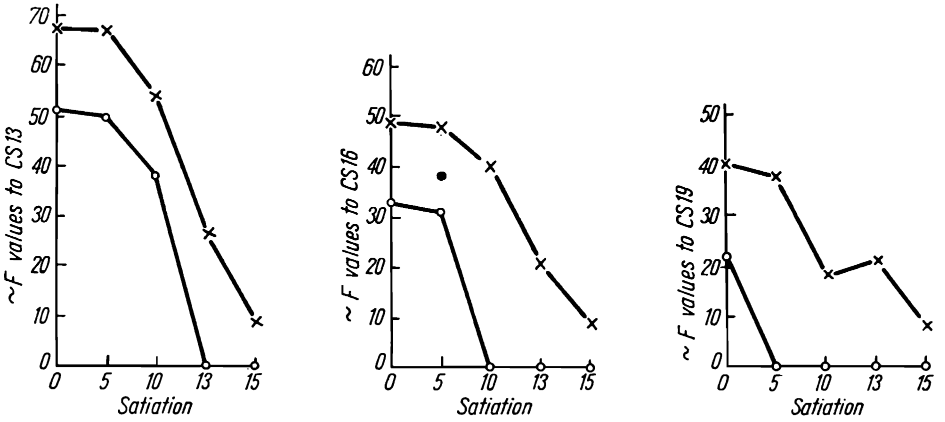

Fig. 8. Comparison of ~F values to CSs in Experiment II (crosses) and Experiment III (circles). Abscissae: levels of satiation. Ordinates: ~F values to CS 13 (left graph), CS 16 (middle graph) and CS 19 (right graph).

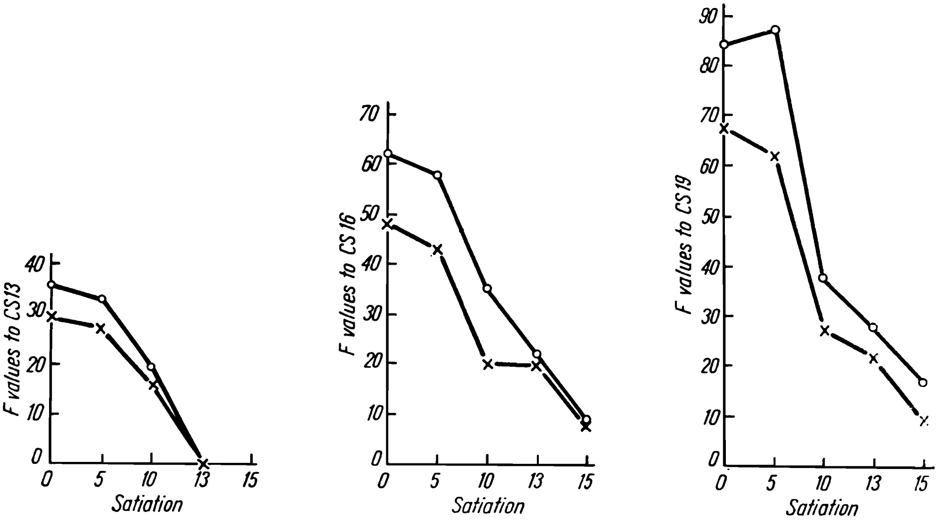

Fig. 9. Comparison of F values to CSs in Experiment II (crosses) and Experiment III (black circles). Abscissae: levels of satiation. Ordinates: F values to CS 13 (left graph), CS 16 (middle graph) and CS 19 (right graph).

Finally, as seen in Fig. 10, the ~H responses to all CSs (but not to the FUS) are uniformly smaller in Experiment II than in Experiment III. In fact, the sum of the ~H values to CSs in Experiment II amounts to 2785, whereas the sum of these values in Experiment III amounts to 3627.

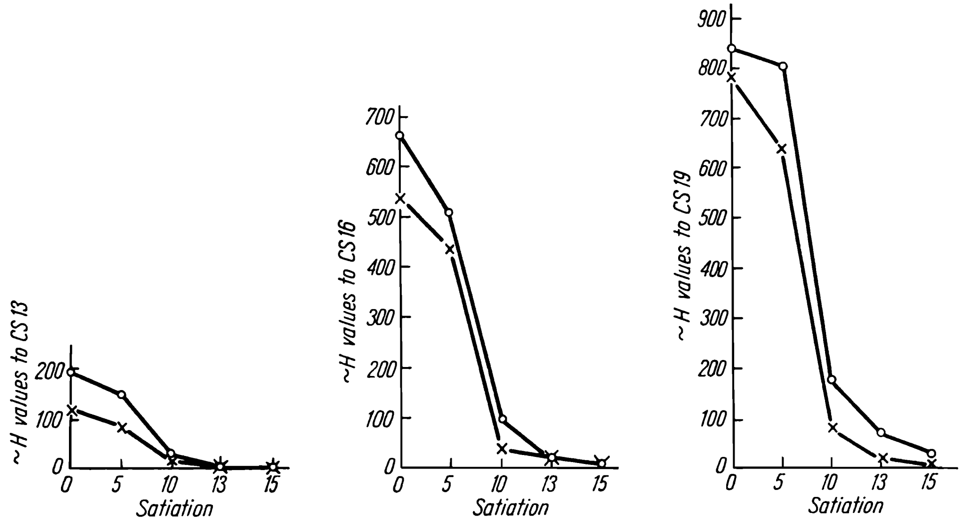

Fig. 10. Comparison of ~H values to CSs in Experiment II (crosses) and Experiment III (circles). Abscissae: levels of satiation. Ordinates: ~H values to CS 13 (left graph), CS 16 (middle graph) and CS 19 (right graph).

Turning to the explanation of these results we should start with the analysis of changes in the ~F activity. Of course, its decrease is directly dependent on the weakening of the ~T– ~F bond. It, is, however, conceivable that this decrease is augmented by the increase of the inhibitory influence of the F subcenter upon the ~F subcenter due to the predominance of the former.

The increase of the F responses to the CSs is due to the weakening of the antagonistic excitation of the ~F subcenter. Since at CS 19 the activation of the ~F subcenter dropped to zero (except at Sat 0), F responses to that CS, which in Experiment II were always lower than those to the US, are now of the same magnitude.

This finding indicates that the smaller magnitude of the F responses to the CS 19 in comparison to that to the US, regularly observed in Experiments I and II (Tables I and II, upper left tablets), is in fact dependent on the inhibitory influence of the ~F subcenter.

It is also interesting to note that whereas in Experiment I and II the CS 11 was subliminal at all levels of humoral hunger, in the present experiment it becomes above threshold at the two highest levels of hunger. This is a clear evidence of the fact that in the two preceding experiments the subthreshold character of this stimulus was not genuine, but it was caused by the strong inhibitory influence of the ~F subcenter.

The increase of the F responses to the CSs leads naturally to the increase of the excitation of the ~H subcenter. This increase should attenuate the excitations of the H subcenter and thus influence the excitability of both F and ~F subcenter. In our experiment, however, this did not occur because, as mentioned before, the ~H–H bond was attenuated. Had the situation been in Experiment III such as in Experiment I, the feedback of the loop F– ~H–H– F would be well pronounced and this would strongly stabilize the system diminishing the differences between the two experiments.

If the present experiment is a true simulation of the normally existing relations, than the results obtained are highly important. For, if in spite of the fact that the animal has no food in the mouth its ~F subcenter is silent while its F subcenter is activated, the situation does not differ from that when food is actually in the mouth. Speaking psychologically we can say that the animal has such a strong image (or may be hallucination) of the food in the mouth produced by the CS that the perception of the absence of food is completely inhibited. We may call such phenomenon eidetism and a subject who is prone to experience it the eidetic type. By the way it may be seen in our experiment that the high degree of humoral hunger does not necessarily favor the appearance of eidetic phenomena. As a matter of fact with a very high hunger the absence of food in the mouth may be perceived so acutely that it would not allow the image of food in the mouth to prevail.

DISCUSSION

As it was said at the beginning of this series of papers (Konorski and Gawroński 1970) a great body of experimental data in the field of alimentary behavior of animals led to the formulation of a theory concerning its central mechanisms. Since these mechanisms appeared to be rather complex, it seemed reasonable to represent them in the form of a model, whose elements would simulate the uniform pools of neurons or "centers", and the connections between them would reflect the organization of the alimentary system postulated by us. Our first task was to see whether the model is workable, that is whether the experimental data obtained on this model would agree with the data observed in experimental practice.

The results of our study turned out to be amazingly successful. In the present paper we dealt only with classical CRs and studied their dependence on the strength of CSs and the level of humoral hunger. We were able to replicate very precisely the main principles of the CR activity as they were discovered and worked out in Pavlovian laboratories. Here belong:

1. The level of satiation being constant, the positive dependence of the magnitude of CRs on the physiological "strength" of CSs.

2. The strength of CS being constant, the negative dependence of the magnitude of CRs on the level of satiation, or the positive dependence on the level of humoral hunger.

3. The insignificant slope of the first regression with high humoral hunger (the so called equalization phase) and its steep slope with moderate humoral hunger.

4. The insignificant slope of the second regression with strong CSs and its steep slope with moderate CSs.

Even more important was the circumstance that since in our artificial system not only the food conditioned response, being the indicator of the excitation of the F subcenter, was available, but also we could measure the activity of the ∼F subcenter, H subcenter and ∼H subcenter, the analysis of data obtained in our experiments could go much deeper than it was possible in natural experiments.

The next problem which emerged in our study was this.

Starting to experiment with a number of dogs Pavlov was struck by the fact that within the above general principles each dog exhibited his own individual properties not identical with those in other dogs. Exactly the same was observed in our experimentation with Dewan. It was sufficient slightly to modify the organization of the system by changing the weights of some connections to obtain dogs with quantitatively different properties. In this way we could easily simulate the Pavlovian "types" of the nervous systems by creating Dewans with different functional characteristics.

Here we concentrated our attention only on one problem concerning the "typology" of the nervous system, which to our knowledge was never raised before in the CR experimentation. We drew attention to the fact that the CR operates by definition when the mouth is empty, that is, when the ∼F subcenter is activated by the off-taste receptors. This subcenter reciprocally inhibits the F subcenter not allowing the FCRs to attain equal values to those of the FUR. Now, it may be supposed that there are constitutional differences between the subjects depending on their "suggestibility". There may exist subjects, denoted by us as eidetics, in whom the ∼T–∼F connections are not very potent and therefore a strong activation of the F subcenter may completely inhibit the ∼F subcenter.

Other subjects, which may be called realistic ("matter-of-fact dogs" of Pavlov), have strong ~T- ~F connections and therefore in them the perception of lack of food in the mouth cannot be suppressed by a FCS. It is clear that in the eidetic type of subjects the FCRs are stronger and more reliable than in the second group; moreover, the CR to a strong CS may equal the UR, the phenomenon never occurring in the realistic type of subjects.

Another point of our interest was concentrated upon the problem of the significance of the quality of food reinforcement for the properties of CRs.

We have discerned two different qualities of food eliciting different unconditioned responses:

1. The slimy or liquid tasty food which, when placed in the mouth, stimulates immediately the whole taste receptive surface and requires very little or no grinding. The pulpy minced meat or milk represents this type of food.
2. A food which when put into the mouth stimulates at the beginning mainly tactile receptors, but after thorough grinding is transformed into a tasty bole ready for swollowing. Bones or tasty biscuits are the examples of such food. It was supposed that this type of reinforcement may be modelled in Dewan by weakening the ~H–H inhibitory bond: in fact, it may be assumed that digesting this kind of food is connected with strong satisfaction, but in the same time the hunger drive is not considerably inhibited.

It was found that this relatively small change influences to a great extent the whole activity of the alimentary system. What was interesting and completely consonant with our intuition was the considerable increase of the satisfactory (pleasurable) character of the food reinforcement (increase of ~H). Both the FUR and the FCRs increased considerably and they became much more strongly related to the intensity of hunger than in the case of slimy food. Accordingly, the influence of the humoral hunger upon the FUR and FCR activity was considerably increased. As noted before, such experiments were not performed on normal animals and therefore our conclusions reached on the basis of experiments on Dewan are so far only predictions in regard to the CR activity in normal dogs.

The question is still open as to whether or not the simplification of our model or its essential modifications could explain the same experimental facts. We are not able to give a general answer to this question. We have made only one attempt (not published) to simplify the system by removing reciprocal bonds between F and ~F, and H and ~H.

It has been shown that such a model is completely useless, since it fails to obey the rules listed above. It worked mostly according to the all-or--nothing principle, all the subcenters being either maximally excited or not excited at all.

Let us turn now to some general characteristics of the functioning of our model, discussed in general terms in the preceding paper (Gawroński and Konorski 1970b).

First property is that a small change in the weight of one or two connections is sufficient to modify the activity of the whole system, occasionally in an unpredictable way. Accordingly, Dewan fully falls into the category of wholistic systems, and if its internal structure were not known we would see something misterious in its organization. Anyhow, any narrow localizationistic theory of its activity would certainly not hold, and therefore, not knowing its internal structure agnostically minded people would have a tendency to attribute to it some unexplainable properties. Thus we see that a wholistic system, a system which always reacts as an entity, must not be misterious and it is fully prone to the analytical approach.

A second property of the system is its strong intrinsic stability. When looking over our tables representing the responses of Dewan to various CSs under various levels of satiation we are amazed by the lack of linearity of their relations. Very often a considerable change of one of the independent variables fails to affect the response, and time and again we are confronted with instances, when the response is "paradoxical", that is, it breaks the monotonous character of the regression.

The stability of the system is due to the types of feedbacks built in the system. We had a good experimental evidence of it when by destroying the inhibitory ∼H–H link in the F– ∼H–H F loop the stability of responses of particular subcenters was abolished.

Finally, the third property of the system is that when looking at the oscillographic records of the response of particular subcenters we see that only very rarely are they composed of regular sequences of impulses (cf. Konorski and Gawroński 1970, Gawroński and Konorski 1970b). Most often we have to do with complex rhythms of impulses due to complex interactions between the subcenters concerned. Of course the reciprocity of ∼H–H and ∼F–F connections, as well as convergence of impulses of various frequencies, plays here an essential role. It should be emphasized that these changing rhythms of impulses are very similar to those observed in electrophysiological studies both in macro- and microelectrode technique.

These complex rhythms of responses seen in all our records should be an important warning to all those students who have a tendency to imagine that these rhythms, being of course constant for a given configuration of the input to a given pool of neurons, convey essential information about its contents, and that we shall be able to read out this contents from the "melody" of impulses. If the evidence supplied by Dewan is relevant, this conclusion should be regarded as totally wrong. The complex rhythm of impulses is simply the resultant of the interplay of excitatory and inhibitory impulses sent to a given neuron through many internuncial links and it is a byproduct of their convergence. There is, however, no evidence to claim that this rhythm gives any information about the messages conveyed to the nervous centers.

To sum up, we see the following general advantages of experiments performed on Dewan.

1. The verification of hypotheses concerning the central organization of a given functional system by testing whether the experimental facts can be reproduced in Dewan.

2. The explaining away of some superstitions connected with the study of the activity of the central nervous system. Here belong:

a) The belief that only localizationistic approach allows us to "understand" the function of the nervous system and that the wholistic properties of that system escape a rational analysis and compel us to recur to some vague concepts like equipotentiality, mass action, gradients, etc.

b) The belief that since the action potentials of the brain and sequences of impulses are endowed with complicated patterns, these very patterns are the carriers of information conveyed by the nervous system.

It is very important, to our mind, to get rid of these superstitions by showing their true nature.

SUMMARY

1. The model of the alimentary system described in the preceding papers of this series (Konorski and Gawroński 1970, Gawroński and Konorski 1970ab) was applied for simulating the experiments on classical food conditioning with two independent variables: the strength of the CSs and the intensity of humoral hunger (being the reverse of the level of satiation).

2. Our model closely imitates the CR activity of a typical well balanced dog. In. particular, the dependence of the food conditioned responses on the strength of the CSs and on the level of humoral hunger was reproduced. The Pavlovian "equalization phase" of CRs at high humoral hunger was found.

3. When the inhibitory connection leading from the $\sim$H subcenter to the H subcenter was attenuated, which was considered to imitate the situation in which the hard food is given as reinforcement, the FCRs

and the FURs were strongly augmented, the "principle of strength of the CSs" was more accentuated and the role of hunger in both FCRs and FURs was increased.

4. When the connection linking the off-taste receptors with the $\sim$F subcenter was attenuated, the FCRs were slightly augmented while the FURs were unchanged. The FCR to a strong CS became equal to the FUR. This condition was considered to simulate the "eidetic" type of a dog in which the conditioned food response is not restricted by the messages from the mouth receptors informing the absence of food.

5. The significance of the present model for the better understanding of the mechanism of the alimentary system is emphasized and it was shown that the "wholistic" approach to central nervous functions does not prevent the precise analysis of the underlying nervous structures.

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