

Konorski's notions of conditioning. p. 86-90

Plasticity as a physical property of cerebral cortex  
Between the centre of CS & centre of US previously  
non-existent functional connections are formed.

Not a growth of new, previously non-existent nerve paths  
(can get condg w. single axon of adq. 5)

Formation of new intercentral conn. = a synaptic process

Potential interneuronic conn. exist before

a CR is established.

In condg the potential connections are transformed  
into actual connections.

[ Learned R. is always possible first time, so not  
entirely new pathways ]

The "emitting" neuron centre becomes  
coupled to the "receiving" + " "

Functional (dynamic kinetic) vs morphological changes

assumption - bringing into activity previously inactive  
closed, self-reexciting chains of neurons in highest  
divisions of M.S. (Young, Jz, Korbovsky, Hausholder, etc)

cessation of the circuit = forgetting

incomprehensible to Konorski how these could survive  
deep narcosis, cerebral ischemia, etc. that knocks out simple  
reflexes - whole body of engrams is completely  
preserved in such states.

|| R. favors formation and multiplication of new  
synaptic junctions. (like Cajal, Kappers, Child, Pughill, etc)

fading or atrophy of synaptic conn. denotes  
forgetting & loss or extinction of CR's

[ Young's principle of double dependence (1946) have to 52 above  
3 halves otherwise get atrophy ]

K. H's Bykov in believing there are no reciprocally unrelated areas whatever in cerebral cortex, i.e. can condition anything to anything.

Paulovian CR = simplest & best known type of plasticity

The condit'd brain center & uncondit'd brain centre.

|| The interconnections not direct & simple = extremely complex.

Background along w. specific S. figure in receiving & emitting centres - extremely diff'd & complex - [so complex, the value of the theory is here lost]

|| Growing permanence of a reflex w. repetition = growing stabilization of the synaptic contacts

Once accepted that synapses are formed thru conditioning and that they are subject to atrophy when CR not trained, no difficulty in accepting that training leads to increased stability.

Contracts there's a physical of lower centers & Pavio of cortex. Plasticity as an attrib. of cortex.

For U.C. need inborn, permanent Rn

|| Pav. that a strong excitat'n reaching cortex attracted weaker ones reaching cortex simultaneously

CR = result of the meeting of the waves irradiated from diff't points

CR will die out from lack of reinforcement - or can be inhibited by ext. S w. its own uncondit'd R.

Paulov had effects irradiating across cortex from pt of impact. in a field-like gradient  Then get a flow back toward center & concentration.

Get irradiation of inhibition & of excitation.

Get wider induction effects as well as direct irradiation (when direct irradiation)



Beritoff theory as alternative (Georgian physiologist  
Individually aged activity of CNS N. 1932

(severe & ruthless criticism of P.'s theory)

K. goes along w. B.'s criticism, but thinks his  
positive aspect, his own theory = unsatisfy.

B.'s concept of "interdependent irradiation of excitation"  
irrad. in all directions like elec. current in a  
ramified conductor  
controlled by bio-electric currents



conduction becomes more & more covered  
here w. repetition

This theory hasn't even induced P.'s ffns to revise  
their views - only real service - drawn attention  
to the gulf between the physiologies of the higher  
and lower nervous activity.

Every axon possesses scattered synapses over some,  
but on only small zone only synapses from diff. axons  
Work of Lloyd & Renshaw requires direct inhibitory <sup>synapses</sup>  
Direct inhibition, K. says, must account for most of  
inhibition phenomena in conditioning.

Not clear whether same axon can excite some cells  
& inhibit others. K. prefers to operate on assumption that  
an axon carries impulses of only one character (excit. or inhib.)

Extinction of orientational <sup>taking to ground</sup> reflex - acute Ri + permanent,  
irreversible  $\Delta$  in centres = extinction. Later = evidence  
of cortical plasticity, found almost exclusively in cortex.  
Cortical excitability = widely studied, plasticity hardly at

Plasticity is found only in normal, i.e. unanesthetized  
animal.

"Cumulation" strengthening of CR in repetition, regression without, stabilization over long periods,

But it never occurred to Pavlov that the capacity to form plastic changes is a property <sup>of its own kind</sup> in general, which can be correlated w. other characteristics of N.S.  
[ p. 86 see 1st few pgs of notes ]

Can get conditioning of 2<sup>nd</sup> order but takes a strong UCR & CS of 1<sup>st</sup> order = weak

Growth of synapses with training and atrophy, regression, w/out reinforcement. RL  
- 1948 -

Anokhin

Konorski, Jerzy 1948 Conditioned reflexes and neuron organization. Translated by Garry.  
Cambridge University Press

Konorski's 1<sup>st</sup> type = classical (saliv) - somatic + autan  
" 2<sup>nd</sup> " = instrumental - only somatic