Mechanisation of Thought Processes

VOLUME II

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A Simple Computer for Demonstrating Behaviour
DR. W. ROSS ASHEY, Barnwood House Hospital, Gloucester

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MACHINA REPRODUCATRIX

AN ANALOGUE MODEL TO DEMONSTRATE SOME ASPECTS OF NEURAL ADAPTATION

by

DR. A. J. ANGYAN

MEMORY has always presented an important problem to physiologists and neurologists, and there have been many attempts to interpret the brain and its memory function by physical analogues or illustrations. The early ones included Dubois Raymond, Pavlov (ref. 16) who used telephone analogues for both unconditioned and conditioned reflexes, and the Hungarian neurologist, Jendrassik (1912) who used analogies of physical induction and resonance. More recent theories of conditioned reflexes Young (1938, ref. 22), Hilgard and Marquis (1941, ref. 9), Konorski (1948, ref. 11), and Hebb (1949, ref. 8) used similar ideas in attempting to make hypotheses on plastic adaptation using various electronic circuits. All-or-nothing features of synapses were detected by microelectrode studies and discussed by McCulloch and Pitts (1943, ref. 13), and by Eccles (1953, ref. 6). Cragg and Temperley (1954, ref. 5) used electromagnetic phenomena, i.e. field processes.

However it is clear that all these analogies are only very tentative ones, even if they do show some features of the brain. They are useful in helping to explain the meaning of biological terms, and, not less important, they may help to clear up errors and inconsistencies in our terms, and of the physical analogues associated with them. Physiologists are obliged to define clearly the concept of memory as a mechanism of a highly organized complex living system.

Machina Reproducatrix, which is shown here (photo, fig. 1) is a model which attempts to demonstrate some, but not all, of the recently formulated concepts of nervous adaptation. It is based on Dr. Grey Walter’s Machina Speculatrix and its development Machina Docilis. It is a relatively simple analogue model of a simplified pattern of the innate and acquired reflex connections of a living being. It is a simple model compared with the more refined concepts of Dr. McCulloch (ref. 14) or Dr. Rosenblatt’s perceptron, Dr. Uttley (ref. 19) and his Conditional Probability system or Dr. Ashby (ref. 4) and his habituation and homeostat models.
The model has two or three receptors, for light, sound, and mechanical input. The signals from these receptors are relayed through two control "centres" each of which controls a motor; this dichotomous control serves to demonstrate a basic innate pattern of behaviour. (There is in fact a very important dichotomy inherent in all behaviour, between well-oriented and directed goal-seeking and random searching.)

Like most living things, the model, if once aroused, searches for a goal for one of its tropisms or inborn unconditioned reflexes. It searches for light because of a connection between its photo-receptor and effector motors. This behaviour, which is dominant in the system, is disturbed or inhibited by any other stimulus impinging on its mechanical or acoustical receptor systems.

But extreme strength or duration of the original dominant stimulus causes the model to seek a new dominant cue. This illustrates another principle of reflex activity which never allows an organism to follow any drive continually: by switching off the drive at some level or after some time it starts a search for a new stimulus. Analogies of this can be found both in whole organisms and in any artificially separated part of their nervous system. Two teleological principles seem to be involved, but by introducing the concept of positive or negative feedback the organization can be seen to be very simply determined.

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**Fig. 2**
A logical concept of conditioning and learning was built up by Dr. Grey Walter in his 'CORA' and we must study the action of 'CORA' briefly before going on to the next stages. Figure 2 is a block diagram drawn in similar form to Machina Reproducatrix. The first three logical elements of Dr. Grey Walter's model can show a behaviour analogous to that which may happen in every instant of alert activity of the brain centres. Two stimuli, which originally were temporally or spatially separated, may meet on one or more synapse of the brain, ensuring alertness which is a basis for receiving communications from the external world. In 'CORA' this can happen if (a) the original driving or tropistic stimulus is differentiated(1)* corresponding to the on-off effects produced by appropriate synaptic connections at sensory inputs to the brain. (b) a second non-dominant or neutral stimulus preceding the dominant one is delayed in its effect(2) (corresponding to "after discharge" or widespread non-specific activation of synapses). (c) the two signals satisfy the necessary conditions, e.g. coincidence, summation(3).

So far there are no difficulties in explaining these features of neuron behaviour, but for building up a conditioning process a further logical step has to be introduced. This involves a temporal summation of the overlapping coincidence areas or the probabilities of the two stimuli. In the model this can be represented quite simply by a capacitance circuit(4); nevertheless in a nervous system it may involve a more refined mechanism e.g. it may work on principles similar to Dr. Uttley's conditional probability system.

Dr. Grey Walter points out that a nervous system, working as a learning box must rule out sheer chance and reach a threshold of coincidence for conditioning. There have been many hypotheses of conditioning, e.g. in the neurophysiologically almost correct work of Eccles (ref. 6) he attempted to localise it as a specific feature of the central grey matter or of the cerebral cortex because of the multitude of input and output connection in this region. But Pavlov pointed out (ref. 17) that in appropriate conditions the "Summation reflex" (i.e. association of two stimuli) may occur and give effects on any level of the nervous system, especially the less co-ordinated ones. Dr. Uttley (ref. 19) found that, in his conditional probability computer, the activation of a unit corresponding to the firing of a neuronal network does not suffice to distinguish actual occurrence from computed probability, and he introduces the postulate for a regenerative loop.

The next steps in Dr. Grey Walter's logical scheme are activation(5) and the preservation(6) of information by an oscillatory circuit with very light damping (an analogy of cortical alpha-rythym). These steps are

* Numbers in brackets refer to points on the block diagrams.
followed by gating the oscillation and the original neutral stimulus to produce a response corresponding to the original dominant or unconditioned stimulus. The response is analogous with cortical neuron output.

At first glance this seems to be a correct representation of conditioning and of the memory function of the brain and most hypotheses explaining conditioning on the basis of neural connections are similar.

We decided, in our model Machina Reproducatrix, to reproduce conditioning in the same way as 'CORA' does, but instead of an oscillatory circuit we employed a neon tube with two relays and a thermistor (the slow rise of temperature of which produces a memory for paired stimuli for almost the whole running period). This solution was preferred since the original one was too sensitive to mechanical disturbances. This may also be said about the neural network which is exemplified by this circuit. In the introduction of the thermistor we may see a very slight analogy with the newly-established fact that the excitability and activity cycles depend on the activation system and thence the cortex on vascular effects. There is so far no justification for taking these analogies too far.

Though Dr. Grey Walter's learning box displays some surprisingly correct imitations of life it shows that until the last few years the explanation of brain processes in conditioning and learning followed a very simple scheme. Compared with any animal during conditioning, we must conclude that the CORA circuits omit some important characteristics. These are:

(a) the conditioning trials do not lead to habituation of the neutral stimulus,
(b) other stimuli related to the conditioned one produce no effect, i.e. no generalization occurs,
(c) memory is lost as the oscillations gradually decay in the memory circuit and is finally extinguished. We must repeat the stimulus combination to obtain recovery which is never spontaneous as it is in animal conditioning experiments,
(d) a new disturbing stimulus may cause gross deficiencies in the function of such a delicate automaton depending on external influences. They seem to produce neurotic-like behaviour, but no marked external inhibiting or disinhibiting effects can be distinguished.

We therefore attempted to supplement CORA to overcome some of these defects. Machina Reproducatrix responds to three stimuli, light, flute and whistle. Six or eight repetitions of flute and light cause conditioning and the model turns it front towards the sound (just as the previous model did to light). But once the capacitive memory sustained by the thermistor circuit decays, no more conditioning effects are observed. If we repeat the combination of flute plus light conditioning occurs at once. If we now try the whistle, we may observe a conditioned effect i.e. a generalization.
But after several (about 18) repetitions of whistle without light no more such effects occur, whereas the flute (which with light was the original stimulus) remains a conditioned stimulus. This is simple discrimination and if we carry on fluteing only, the effect of the flute is also extinguished or habituated and no tropistic behaviour is seen. But, in our model, the conditioned effects of the flute recover in a few minutes, but the whistle remains as an effectiveless stimulus. If a new sharp sound is intonated, even the flute may be depressed; this is external inhibition, and after a few instances the whistle will regain its effect, this is disinhibition. The flute also recovers its conditioned effect comparatively soon.

Fig. 3

Figure 3 is a block diagram of the original model, Machina Docilis, and the supplementary parts which have been added to make Machina Reproducatrix achieve these features. The three inputs $N_1$, $N_2$, $N_3$ have a common input to a counting device (8) connected with another thermistor circuit (9)(10)(11), the outputs of which are connected to the gating relay. If $N_1$ and $N_2$ occur together conditioned reflexes are obtained from any input which contains either, but stimulations due to $N_1$ or $N_2$ alone are counted and cause blocking for a given time of the conditioning effects. If a sufficient number of unpaired stimulations of $N_2$ occur it leads to permanent
Inhibition of unreinforced stimulations of $N_2$ until $N_2$ is disinhibited. Thus we build up a discrimination. If a new stimulus $N_2$ is given, and it is strong enough, it connects a transistor circuit to produce disinhibition of $N_2$ and also inhibits the conditioning effects of $N_1$.

We realise that this model is somewhat crude, but it was conceived not only for demonstration purposes, but also to draw attention to some important questions. The supplement to "Machina docilis" was conceived to represent the phenomenon of habituation which results from any biological stimulus. It is a basic mechanism by which extinction of conditioned reflexes and their internal inhibition leading to discriminations is built up physiologically. But its effect is temporary, and may be thought of as a negative feedback to the changes in neural connections of every nervous adaptation. In fact, the conditioned reflex cannot be fully represented by taking account only of association, combination or summation of two stimuli, since the mechanism of habituation occurs in parallel and counteracts any accumulation of corrections. (Thus, it ensures the conditions necessary for the coexistence of a "conditional probability" and a "conditional certainty" system in the brain). Experimental observation on the mammalian and human brain especially in the Neurophysiological Institute of Pecs and also in the Neurophysiological Clinic in Debrecen, (Lissak and Grastyán ref. 12, Kajtor et al, ref. 10), clearly suggested that it is the hippocampal system which may act like a counting device for any sensory inputs. This agrees with Green and Arduini's electrophysiological observations. They observed that, following stimulation, the hippocampus may inhibit conditioned reflex activity, independently of the origin and dominance of conditioned or unconditioned stimulations, and this may be demonstrated by studying orientation reflex phenomena of the model. Now it seems that the supplementary parts of the model may be compared with the negative-feedback effect which the hippocampus has on neocortical and brain stem phenomena in conditioning. We should leave further neurological discussion of the structural analogy and point only to some observations by which the validity of this analogue can be tested. The electrophysiological observations of Morell, Jasper et al (ref. 15) have shown that a significant impairment of electrocortical conditioning occurs only with archipallial (hippocampal) lesions and Penfield and collaborators (1958, ref. 18) pointed recently to the fact that distinctive, bilateral lesions of the hippocampus only wipe out the recollection of recent memory experiences. But even if we control the effect of such lesions or that of any extended cortical lesion in a conditioned reflex experiment, we may find that the association process by itself is less impaired than discrimination and appropriate recall (Angyan, 1956, 1957 ref. 2). This somewhat too docile impaired learning mechanism is modelled by "Machina Docilis". We get the same result by comparing the brain adaptation with any model taking account mainly of the conditioned summation reflexes.
Our model demonstrates (1) that habituation and internal inhibition must occur in parallel and must to some extent inversely regulate the conditioned summation or probability computation process. (2) that discrimination is based on a common mechanism with habituation but it may be further improved if specific inputs with specific analysing mechanisms are considered. Some degree of discrimination may be obtained by filter mechanisms, but it is only effective if it is regulated by extinction based on habituation. (3) that spontaneous recovery (or recall of forgotten or extinguished conditioned reflex) cannot be based on preferential states for more recent stimulations (as in Dr. Uttley's concept). Our model though far from being a sufficient analogy, allows the repeated recall of an extinguished conditioned reflex during the whole span of its memory.

The fact that our model is still of insufficient complexity is shown by the effect of external inhibition. By causing a block through the orientation—habituation mechanism, this wipes out both the effects of positive and negative stimulations. To obtain a recovery of discrimination, we need to build up again the extinction or discrimination process. In our opinion, this problem can be adequately developed by constructing a model which incorporates another very important and experimentally well-founded fact of nervous adaptation— the Sherrington principle of reflex antagonism and mutual induction. Pavlov has pointed out that this is the third basic mechanisms (with excitation and inhibition) in the maintenance of dynamic equilibria of every complex of innate and acquired behaviour.

If we assume that, in parallel with the extinction process or the development of any internal inhibition, that the stimulus to be discriminated build up a connection with another specific stimulus in opposition to that already conditioned (e.g. summed with a negative stimulus), it would not be too difficult to build a model which demonstrated electronically this mutually or reciprocally inductive antagonism (perhaps in the sense of Wiener's 'moth and bedbug' model combination). If the two summation systems are coupled together mutually in an inverse feedback manner, an automatic mechanism is obtained which resets the original state of discrimination immediately after the disturbing stimulus disinhibited it. In fact, this occurs in uninjured, normally adapting, animals, with a speed and repeatability which is very characteristic of the individual. It is one of the most important transformations which sometimes seems to almost entirely override the rules of conditioned association in everyday psychic activity. It is usually the first mechanism to be impaired following functional or structural disturbances of the brain. We are convinced that this mechanism of 'direct' inhibition is an inherent structural feature of nervous organization. Every unconditioned reflex has a positive 'to' and a negative 'fro' aspect (seen in defence, feeding and sexual activities). These form the basis of unconditioned reflexes in their lower and higher manifestations.
Plasticity (acquired individual adaptation) is however maintained by habituation, or by indirect inhibitory mechanism, which never allow ‘summation reflexes’ to build up above a certain limit on the basis of the former ones as a result of new stimuli impinging in their networks. Plasticity also counteracts the development of fixed correlations between the summation reflexes, in contrast, e.g. with the spinal cord’s reflex organization. In an ideal model system which also contains the development analogies it should be shown, as in the mammalian brain and in general in animal of the main phyletic line, that (a) these mechanisms are acting along spatial axes according to Child’s gradient (b) the habituation mechanism in its interaction with a summation of probabilities is never allowed to be static (c) the opposing coupled basic reflex systems cannot be static. If it satisfies these conditions the model system fulfils the requirements of Dr. Ashby’s concept of homeostasis maintained by ever-changing dynamic equilibria and regulated by partly deterministic and partly probabilistic perception systems. But our supplemented model has functional correlates in the high organization level of the mammalian brain. The dichotomy of its design is also based on simple experimentally-demonstrable properties of neurons just as in the hypothesis of Eccles – impulse threshold changes, synaptic use and disuse, reciprocal antagonism and feedback principles. There are also probably differences in the axosomatic and dendrodendritic connection of neuronal adaptation similar to those of summation and habituation on the higher organizational level.

CONCLUSIONS

Our model cannot exclude the possible generalization that learning is simply due to a coupling of two mechanisms – say a positive or excitatory feedback and another negative feedback on inhibition. This coupling can occur at every structural level of biochemical, electrophysiological, or functional anatomical organization, and to show its special adaptive features we must find out the principles for further development which are directing the mechanism whose nervous system quickly adapts to its environment. Recent observations of the author (Angyan et al 1957, ref. 3) on the behaviour of flatworms during regeneration of the cephalad and caudal parts of their primitive nervous system shows that the two aspects of nervous function referred to above may be separated from each other functionally and structurally by simple experiments. It seems that both developmental and behavioural mechanisms show interesting polarization along the developmental axes of Child’s gradients. Perhaps the conditioned reflex is no more than a temporal expression of an axial developmental mechanism propelled by an excitatory summation which shows more general rules of polarization and is limited by inhibitory habituation.
Since in complex organisms we are dealing with at least three special dimensions or axes (and perhaps a temporal one as well) we are inclined to suppose that a more complete analogy of learning and conditioning would be represented by a model in which three antagonistically-coupled CORA or Reproducatrix systems were linked and their varying dominance cancelled by an adequate internal scanning or temporal summing mechanism. This idea forms the basis for further development of our model (Angyan, Zemanek and Kretiz, 1959).

In our opinion, models are only useful in that they help to draw a better concept of present knowledge of brain function the realization of which may show whether our ideas and terms may correspond or should be excluded from the interpretation of life.
REFERENCES


