

**SYMPOSIUM: THEORETICAL-EXPERIMENTAL APPROACHES TO MEMORY**

**The Material Basis of Memory**

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Any system—and I shall not define system more closely than as a group of somehow integrated subordinate units that forms an entity of its own—must have a certain architecture, structure, or morphology, which is reasonably constant in time. The way the system is put together, I like to call its “being.” This system interacts with its environment, responding to stimuli—whether at the more complex level of organisms giving behavioral responses or simply a rubber band yielding to a weight—and most of these responses are in effect ephemeral, reversible changes in time. The system yields and restores itself, and this I like to call its “behaving.” But under certain conditions the interaction of the system and its environment leads to irreversible changes; the system has altered as a result of its experience. It fixes its experience and so becomes something different, and this I like to call “becoming.” So this addiction to alliteration gives us architecture or being, functioning or behaving, and development or becoming.

Becoming subsumes, of course, development of the individual, evolution of the species, history of the particular society or social group of any kind, and learning in the individual. And learning may include, if you accept a broad definition, changes as varied as: the hypertrophy of a muscle with exercise; the horny hands of a laborer; and the many other material changes that record the past—as in that lovely couplet on weatherbeaten trees:

Is it as plainly in our living shown,  
By slant and twist, which way the wind  
hath blown?

That is memory in trees. I would like, then, to approach the problem of fixation of experience, which is the basis of becoming and which includes in it memory, from the point of view of the interaction of the organism and its environment.

This occurs ordinarily, of course, at the interface. Start with some kind of a system; it is acted upon by the environment, probably mostly reversibly. Sooner or later, however, there are irreversible changes and there is a different system than before. The environment continues to act upon and interact with it to give a still different system. And there is a steady flow through time of the system plus its past, as influenced by all the things that have happened to it. At any one stage, the new system is some product of what was in it at that time, which might be called its inheritance or heredity, plus what was done to it, its environment. This progressive specification or alteration with time is, in the broadest sense, fixation of experience. This process is operating, of course, from the very start. We ordinarily think of organisms as having a fixed genetic past, carried in what happens to the gene complement of a particular individual; but, more profoundly, the kinds of genes that are present represent the influence of the environment on ancestors of that organism—by selection or any other

mechanisms that happen to be valid. The environment starts, then, ordinarily with a heterogenous system and does things to it.

In principle, however, even a homogenous system, able to respond differentially to differences in the environment, will develop heterogeneity. Recall the dramatic experiments with frog eggs early in this century. The fertilized egg divides into two, and the two blastomeres become the left and the right sides of the body. From their medial portions come the backbone and the spinal cord and other midline structures. But if these two half-embryos are separated they do not die; they continue to develop and produce identical twins. The left side of the left cell becomes a left side, as it did before; and the right side of the right cell develops as before. But the right side of the left cell no longer becomes a center; it now forms a right side—and the left side of the right cell becomes a left side. This was so mysterious that Driesch, who first was concerned with this, gave up mechanism and said this was obviously vitalistic; that some kind of guiding spirit or entelechy determines which way these cells develop.

Then the zoologist Child got the clue to the explanation in terms of concentration gradients. With the two cells stuck together at the central plane, to form a sphere, oxygen must diffuse from the surface to the center and its concentration would be lowest at the midline. Carbon dioxide, formed in the cells, would have the reverse concentration gradient. If a low oxygen concentration (or a high one of carbon dioxide) favors differentiation of protoplasm into midline structures and the reverse favors lateral structures, each cell will become a side of the embryo and, eventually, of the adult. But with the two cells separated, same gradients will form between surface and center of each cell—and two complete bilateral individuals will result. So the environment imposes structure on homo-

geneity by virtue of such a simple mechanism as the concentration gradient!

As the embryo develops further, with many cells, the position of one cell relative to another has certain structural consequences. Where a piece of central nervous system nears the skin, the skin turns in and forms a lens. Where the endoderm adjoins the heart it develops into liver, otherwise into gut. Such relationships have been worked out in great detail. At each stage of development there is further and further differentiation, with more loss of totipotency, and commitment to a particular path from which there is no return. Moreover, the period of flexibility between successive stages of differentiation may be very brief in the life of the embryo; until there is an endoderm no possibility exists to become gut or liver, a few minutes later the path to one or the other has been taken.

Comparably, at a later stage when a nervous system is established, it is possible to rotate a piece of the neuroaxis, the spinal cord and medulla, so that the dorsal side is ventral or so that the caudal end is rostral, and the neurons reorganize to form the normal shapes and connections—they were still totipotent as to general neural structure. Later, this does not work; but the number and size of the nerve fibers growing out at one segment or another can still be altered by moving the developing limb bud. If this is shifted from the normal shoulder region down to the rib region, the normally small intercostal nerves become the great brachial plexus and the normally great cervical ones remain small. Chemical specification may occur without obvious structural change, as when a nerve artificially attached to some atypical muscle comes to carry messages to it from the spinal cord whenever a reflex activates the normal muscle of the same type. There is even evidence that sex differentiation is carried from the chromosomes—*XX* for female and *XY* for male—by differences in the hypothalamus

of the developing nervous system. If the hypothalamus of the early male embryo is transplanted into a female embryo, the latter develops as a male, and vice versa; whereas if the pituitary is transplanted at the same stage of development, the embryo retains its own genetic sex—which indicates that the hypothalamus determines the pituitary which, in turn, determines the somatic sex characteristics.

Moving on from the embryo to postnatal stages, the comparable phenomena are more easily recognized as learning and memory. Imprinting, despite some question of precise interpretation, determines which object a chick or duckling adopts as its mother—a further step in neural differentiation, now indicated by behavior. In mammals comparable phenomena have long been recognized. A newborn kid, taken from its mother for only half an hour after birth, is no longer accepted by the mother as her offspring. Some interaction that should have taken place between these two systems does not take place, the kid fails to get the proper label; and ever after is rejected. If it survives at all it becomes an undersized miserable creature with clearly disturbed behavior. The more recent work, raising baby monkeys with surrogate mothers, has given comparable results; if the young grow up at all, they turn out to be bad mates and mothers.

In man, a study of responses to frustration supplied evidence that childhood experience determined whether norepinephrine was liberated, and anger outwardly expressed, or epinephrine was liberated, and anger turned in as self-criticism. With a weak or absent father or a social milieu that frowned on exhibiting feelings, as in brahmin Boston society, the “anger turned in” reaction was likely; with a dominant father and an expressive milieu, as in Brooklyn gangs, “anger turned out” was the norm. These reactions are pretty irreversible; many experiments on conditioned visceral reflexes in animals—vascular or

respiratory responses to a shock—show these to remain stable after months or years of inactivity.

I still like to tell of a case that came to my attention many years ago at the University of Chicago. Freshmen, undergoing routine medical examination, came down the line with their chests bared, and the physician slapped his stethoscope over the heart of each. One student fainted, so my friend brought him back for examination and finally spent quite a bit of time with him. The long and short of it was that, with the student in front of a fluoroscope and the doctor as far away as the room permitted, the student's heart could be seen to stop whenever he could recognize as a stethoscope some object gradually being brought into view. This is clearly a conditioned response, and the conditioning must have happened early in childhood; unfortunately, no psychiatric study was made so I have no idea what the traumatic experience was.

The fixations last considered involve the nervous system quite specifically; let us move on to more ordinary kinds of behavior. What we can perceive depends on our early experience. Recall the dramatic experiments on newborn chimps, kept in darkness or in unpatterned light for some weeks, and unable later to distinguish visual patterns and jump about in trees. In humans, similarly, a clouded cornea at birth may be replaced in later years by an optically perfect transplant, without bestowing pattern vision. One might think that seeing a circle or a square is a simple business; but it is not. The central nervous system has to learn such discriminations with bitter, hard, carefully built up experience; and, without such perceptual experience in infancy, these people are quite unable to recognize a circle from a square by sight, although making the identification by touch immediately. They may learn this in time but mostly never achieve the fine visual discriminations required for adequate reading.

On the motor side, the same learning of patterns by use occurs in the nervous system. The great athletic champions have practically all, I believe, started to learn their skill very early in life. For top level performance, patterns must be built into the nervous system when it is most plastic; later in life plasticity is largely lost—perhaps it falls off exponentially from the zygote stage. An adult Japanese cannot learn to distinguish *r* and *l*, not distinct phonemes in their language; I saw on a fine piece of electrophysiological apparatus built and labeled in Japan, the word Stimulator. Nor can an adult occidental learn to squat comfortably, for any time, on his haunches with his legs crossed under him, as a Japanese can. A Frenchman, in adult life, cannot learn the English *th*; nor can an English-speaking adult learn the French *r*. These are learned habits, to be sure, but they are actually built into the structure of the developing nervous system.

Some structuring by experience of course continues in adulthood. We lay down new engrams, if I may call them that, throughout life. A striking demonstration of this can be made in the rat. If both occipital lobes are removed simultaneously, pattern vision is entirely lost; if one lobe is removed first and the other two weeks later, pattern vision remains; but if, during that two weeks, the rat is kept in the dark and so denied any further visual experience, pattern vision is again entirely lost. This evidence suggests that existing engrams, built into the brain by visual experience, constitute some sort of scaffolding on which new experience can, so to speak, crystallize itself. Both the nucleus on which to build and the experience to do the building are necessary.

So much for the general behavioral level; it is time to move into the nervous system for a more precise look at the laying down of engrams. First, if a system gives a different performance than before under otherwise similar conditions, and this difference is main-

tained, it can only mean that the system has undergone a material change of some sort. It is entirely comparable to the change in an hypertrophied muscle or the change in a membrane that makes a cell permeable or not. Whether or not we are as yet able actually to see the structural alteration is of some interest and importance; but whether we see these material changes or not, they are there and they are real. A dynamic, rather than structural, change in the nervous system will not do (this is the question that first interested me in memory problems). One could argue that a memory was something that had been set spinning, like nerve impulses reverberating in closed neuron loops, and that the spinning constituted a dynamic memory. If this were the case, then stopping neural activity should lead to irreversible loss of the memory. To be sure, going to sleep or being under anesthesia does not cause forgetting, but physiological activity of the nervous system is not stopped under those conditions.

We took advantage of the fact that hamsters can be made to hibernate: we taught them a fairly simple maze, cooled them down to 5° C, at which temperature extensive soundings revealed no electrical activity in the brain, warmed them up, and retested on the maze. As expected, they remembered it perfectly well. This was, perhaps, the first clear-cut demonstration that memory does not depend on a dynamic process going on in the brain, but on some change that had become structural. Later we thought of an easier way of stopping neural activity—not by “freezing” it in its tracks but, just the reverse, by activating all neurons simultaneously by electroconvulsive shock. This would stop any messages that depended on sequential activation of neurons in chains or in loops, since all would become refractory. Again, the hamsters remembered.

So there is a material change. Further, this change is localized. There is much work along

these lines, the most dramatic perhaps being on split-brain cat preparations. The optic chiasm is divided, so that the left eye remains connected only to the left brain, the right eye to the right, and a pattern discrimination is learned through, say, the left eye. On testing through the right eye, performance is then perfectly good while the corpus callosum remains intact but collapses after this connection between the hemispheres is cut. Left eye performance remains intact. This offers pretty strong evidence that the engram had been formed in the left brain (occipital lobe) and that the right brain had access to it through connecting fibers. Another imaginative experiment, along the same line, took advantage of the spreading depression, produced by concentrated potassium ion, to block one hemisphere in the rat, say the left one, while the animal was given massed learning experiences. Later, with the right hemisphere blocked, even though the left one was now functional, the rat could not perform; when the block of the right hemisphere passed off, performance returned. So learning with the left hemisphere blocked left the engram only in the right hemisphere; again, a clear example of a localized change having been left behind by the learning experience.

How is it brought about? Our experiments with hamsters, and later with rats, were almost not performed, we were so sure of the answer. But a graduate student finally undertook them and turned up a large unexpected dividend. Instead of first having the animal learn and then determining whether it remembered after cold or shock, it was given spaced learning experiences, with interspersed shocks. Every day, at the same time, the animal made massed maze runs and, at another fixed time, was then given an electroconvulsive shock through the head. Comparable foot shocks, incidentally, were ineffective. When several hours elapsed between maze runs and shock, the learning curve was essentially the same as in controls without

shock; but as the interval between runs and shock was decreased below an hour, the learning curve fell off until, at some fifteen minutes, no learning occurred. Repeated shocks can produce minor damage, but that could have no relation to this timing phenomenon. Clearly, some process must continue in the nervous system after having an experience before an enduring memory is established; rather as if, after exposing a film, one had to wait a while before developing it or no picture would remain. A certain time is required for the fixation of experience and we have called it the "fixation time." This was a new and exciting discovery for us but we later found others had run into the phenomenon, and by now it is familiar and well studied.

Granted a fixation time, what is going on during that interval; what is the mechanism of fixation? Has the experience started some kind of chemical process in the neuron which then goes on more or less automatically, as light starts progressive changes in a photographic emulsion, or must there be some kind of maintained physiological activity? I think there is good reason to believe that fixation depends on a continuing physiological activity. Hebb and I suggested independently in 1949 that during fixation there is a continuing reverberation of nerve impulses in neuron loops. In 15 minutes, if one assumes reverberation at 50 a second, there would be some 100,000 repetitions. This is the kind of repetitious impact of an experience that is likely to fix it. It is the old story of one drop of water on the ground disappearing, but a million drops digging a channel, an irreversible material change.

The fact that blocking activity by an electric shock permanently stops the fixation fits better with the notion of some kind of continuing physiological activity as its basis rather than some continuing chemical reaction. There is no a priori reason for an electric shock to stop a chemical reaction and keep it from resuming, but making neurons

refractory should end reverberation. Further, we determined the temperature coefficient of the fixation time by combining the electroshock and the cooling techniques. Hamsters were given the learning experience, then cooled down to a low temperature, kept there for a certain time, warmed up again, and then given electroshock. If kept cool during the learning-shock interval, a shock after an hour or two was still effective in blocking fixation. The fixation time was, thus, prolonged at low temperatures; in fact, the temperature coefficient came out at 2.9. This happens to be the temperature coefficient for conduction velocity of the nerve impulse, which well fits the reverberation idea but should not be given too great quantitative weight. Another thing, certain drugs act upon fixation time about as one would expect from their effect on thresholds. Some depressant drugs would tend to prolong fixation time, by making synapses harder and slower to excite and so permitting fewer reverberations per unit time. This has been found by us and others. Conversely, strychnine, which lowers thresholds and speeds excitation, shortens fixation time. Finally, the reverberation suggestion makes physiological sense because there are many changes in nerve fibers, and even more dramatic ones in nerve cells, produced by repeated activity. Changes in potential, in threshold, in chemical and thermal responses may greatly increase in magnitude and, even more (by 1000-fold), in duration as a result of repeated activity; and altered reflex responsiveness, as in post-tetanic potentiation, can last for hours. All these items favor the view that continued physiological activity—and the easiest one to assume is reverberating circuits—lays down the irreversible change.

What sort of a change is it? We are dealing here with storage of information, in contradistinction to transmission of information; and, whereas the latter depends on all-or-none messages going along nerve fibers at fast rates, the storage depends essentially on threshold

changes of the neurons and on how these respond to the messages when they arrive. The threshold change may depend on a shift in membrane potential on the dendrites, on altered neurohumor liberation, on many other factors; but it most probably involves changes at the receptive component of the synaptic mechanism. And the change might further involve simple inhibitory connections, or chemical action (epinephrine can both raise and lower thresholds), or the diffuse nervous system, the reticular formation and other deeper structures, which plays upon cortical neurons. All these mechanisms could well be involved, but they are only part of the story; sooner or later the issue comes down to a molecular mechanism. Neurons change spatially, they throw out pseudopods and pull them back again, at least in tissue culture, and may increase connections, as by swelling of terminal knobs; and they turn over chemically. Some kind of molecular continuity seems essential and many have guessed as to what kind of change this might be. Almost necessarily it has to be at the macromolecular level, ions and simple metabolites are too evanescent, so attention falls on proteins or polynucleotides or perhaps lipids, although not many take the last seriously. Proteins are formed under the influence of nucleotides, and RNA is a likely candidate. Hyden first, I think, explicitly published such a notion and, as have others, has reported that RNA, also seen as Nissl substance in the cytoplasm of neurons, increases under moderate activity, decreases in fatigue.

Many of you know the evidence from planaria studies. If a flatworm is conditioned and then cut in two, the head end regenerates a tail, the tail end regenerates a head, and both worms retain what was learned. If the regenerative process occurs in the presence of ribonuclease, the head end remains educated, the tail end does not. This suggests that RNA is involved in carrying on memories during the generation of new neurons, as in

regeneration of the tail but not the head. There has been other evidence that RNA is involved in memory and our recent work has been directed rather specifically to this point.

Given an engram, it should be possible to identify it more precisely. An ingenious experiment by Morrell involved producing an epileptogenic focus by placing alumina cream on one spot in the motor cortex, mainly in rabbits. If this spot were excised after a week or ten days, epileptic convulsions still continued, because the mirror spot on the other hemisphere had also become epileptogenic. If the corpus callosum had been cut, this mirror focus never formed, so this was clearly a result of physiological activation, able progressively to build up changes which then endured on their own. Here was a nice localized engram of a sort; and it contained an increased amount of RNA.

We took advantage of a comparable phenomenon, first studied some decades ago by DiGiorgio. A unilateral lesion in the cerebellum produces a postural asymmetry in the legs, clearly due to an asymmetrical barrage of impulses coming down the two sides of the cord. Therefore, cutting the cord, which

abolishes this asymmetrical discharge, should abolish the asymmetry. Such is, indeed, the case, unless the asymmetry has endured for some time; then it may remain after the spinal section. We explored the phenomenon in some detail, for it offered a means of again coming to grips with fixation time. Chamberlain, especially, standardized the preparation in the rat and did careful timing. The fixation time emerged embarrassingly sharply; if the cord is cut within 45 minutes of the time the asymmetry has begun, it will disappear; if after 45 minutes, the asymmetry will remain indefinitely. Figure 1 shows the discontinuity.

The next step was to try and alter the fixation time by modifying the rate of synthesis of ribonucleic acid. Others have tried this kind of experiment on learning rates, using RNA antimetabolites, and have obtained slowing. In our animals, 8-azaguanine prolonged the fixation time from 45 minutes to 70 minutes, still with a surprisingly sharp break (Fig. 2). This was encouraging, but a depression could easily result from nonspecific damage, even though the doses used did not alter the rats' behavior in an exercise wheel nor affect their weight, nor seem to bother

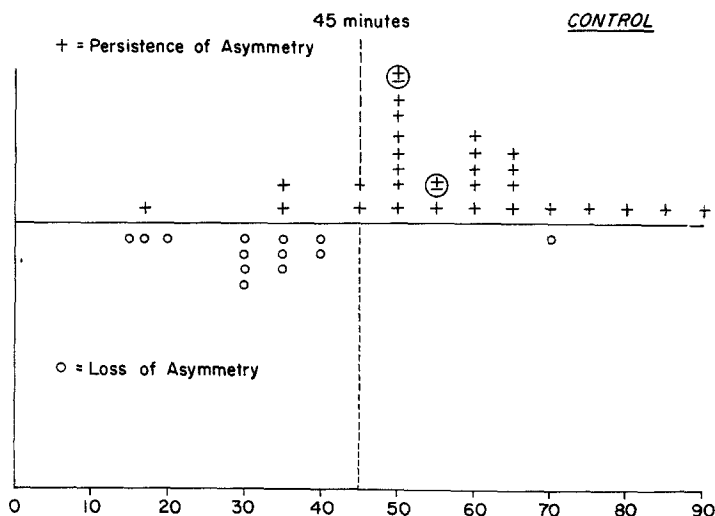


FIG. 1. Interval between development of asymmetry and cord section (minutes) (each symbol represents one animal).

them in any way. Nonetheless, a shortened fixation time with a drug that increases formation of RNA would be far more convincing. For over a year we fussed with a malonitrile dimer reported by Grinnell to speed up RNA formation in neurons and made available by Upjohn (U-9189). In time, Chamberlain worked out satisfactory condi-

tions and found the fixation time shortened to 25 minutes (Fig. 3). Finally, Rothschild has shown a comparable improvement in avoidance-conditioning by U-9189. The rat is put in a vertical cylinder, jumps up and receives a strong shock, and is taken out. Next day, when put in the cylinder, many animals do not jump, indicating learning, or

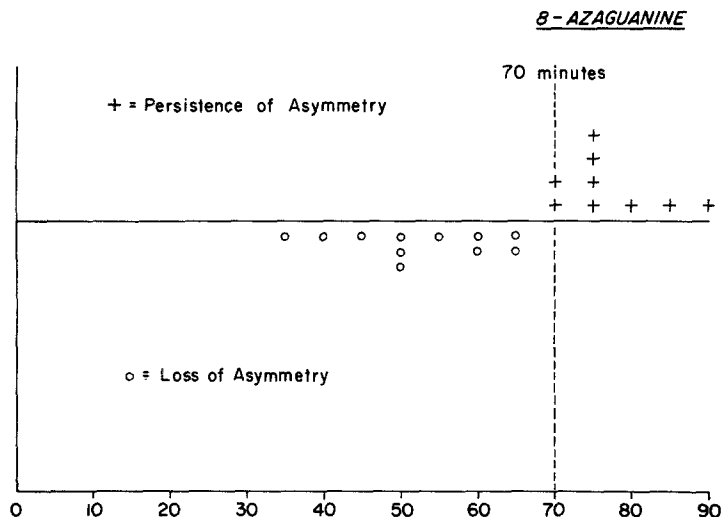


FIG. 2. Interval between development of asymmetry and cord section (minutes) (each symbol represents one animal).

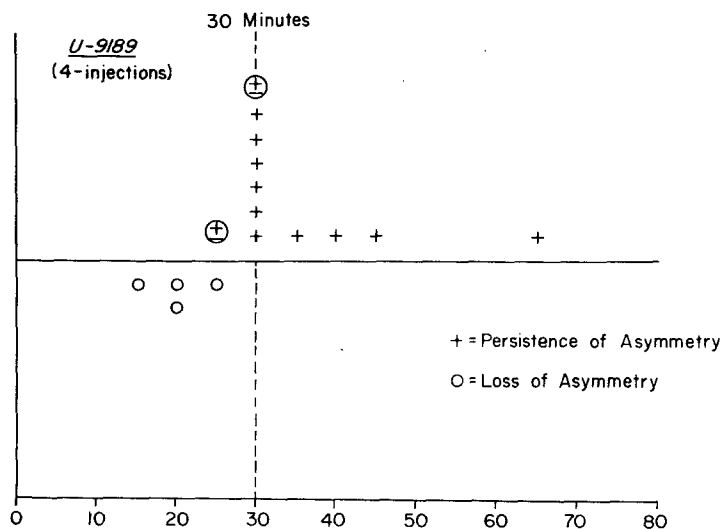


FIG. 3. Interval between development of asymmetry and cord section (minutes) (each symbol represents one animal).



the latency of jumping is longer. On both counts, U-9189 favors learning.

Even if we accept a role of RNA in establishing the memory trace, some of the most important problems still remain, even in connection with the nature of the engram. For one thing, even though polynucleotides might be involved in the process, stored protein molecules, formed by RNA, might actually constitute the enduring material code. Or, different or additional mechanisms might be involved: (*a*) in the very short dynamic memory that lasts seconds or perhaps a very few minutes (e.g., remembering a telephone number to call) but then vanishes without real fixation; (*b*) in the fixed memories that we have been considering; and (*c*) in the really ancient memories, that seem even more enduring under conditions of aphasia or other serious memory losses. It may be that additional mechanisms in the latter case give it the extra stability—as a nut left screwed up tightly on a bolt for many years will rust in place. The sharp endpoint in our cord fixation experiments would suggest that temporary memory is, indeed, different in kind rather than in degree; were it not for the fact that, when the amount of asymmetry is plotted rather than its simple presence at criterion level, there is a rather more continuous curve across the end of the fixation time (Chamberlain, Haleck, and Gerard, 1963a; Chamberlain, Rothschild and Gerard, 1963b).

The really difficult question as to engram formation—it will not be possible even to touch upon the other unsolved one of the specificity of recall—has to do with the transduction mechanism of event to structure to event congruent with the initial one or, in terms of information, of flow to storage to congruent flow. As discussed elsewhere (Gerard, 1963), this raises the same difficulties as does adaptation in evolution. In biological evolution, the answer seems clear that a selective process, as postulated by

Darwin, is operating; in social evolution, an instructive process, as proposed by Lamarck, seems equally established. Individual learning and memory share some aspects of both kinds of evolution, and the exciting question is, therefore, whether particular changes in RNA induced by experience are simply a case of selecting out one type from many possible ones or of actively modifying a given one in a certain direction. In either event, much additional understanding and evidence will be required before the material engram is understood sufficiently to translate molecular and cellular changes into patterns of nerve impulses and organismic behavior.

The functioning of collectivities of neurons, always involved in normal behavior, brings us to another major area of physiology, which demands at least passing attention in any treatment of memory. Wherever molecular and cellular changes may be located, and however specific or nonspecific, it must still be true that many neurons are involved in each engram and, equally, that every such neuron is involved in many engrams. It is impossible quantitatively that one memory be in one spot (there just are not enough such spots even in the human brain to cover a life span of recallable memories), and extensive destruction of brain regions does not lead to memory scotomata, but to either no impairment or quite general impairment. That localization of some sort occurs has already been evidenced; and one might add the well-known findings of temporal lobe stimulation in the unanesthetized human, in which different memories were evoked from nearby cortical regions. But these are still a long cry from the position of one input, one memory. Nerve nets or assemblies are required for reverberating circuits of physiological activity; nerve masses or sheets are necessary for somatic or steady potentials and for the transmission of excitation waves of the sort examined by Beurle. These latter are especially interesting in that they may cross one

another in a cortical sheet and leave, at the locus of intersection, a sensitized region with physiological properties that would permit recall and association of those particular waves. (Moreover, only groups of neurons can exhibit the extremely general and important phenomenon of lateral, or reciprocal, inhibition.)

There is good electrical evidence that in the course of learning activity at first occurs over much of the cerebrum, but that the paths of activity then become neatly channeled. While an animal is learning a problem, and making frequent mistakes, electric responses can be picked up in widely separated regions; when it is performing with skill and correctness, activity is very sharply circumscribed—only to flare out widely again when uncertainty or confusion returns during a trial. This channeling of activity is probably one instance of the action of a type of lateral or feedback inhibition, which is more obviously seen in perceptual and motor control, and equally clearly present although not yet located and worked out in the sudden, almost all-or-none, shift from one mood to another, or from one idea to a different one, or as one plan after another commands attention.

A muscle or a limb is normally thrown into a clean pattern of action in connection with any reflex or other act. Certain fibers in a muscle and certain muscles in a limb contract, while others may remain or become relaxed, under the precisely patterned messages from the motor neuron nuclei or pools in the spinal cord. Activity reaches such a pool from many sources, yet the delimitation between the active and the inactive neurons is always sharp in each case although it shifts from case to case. Small neurons have been shown to be activated by discharges in the motor fiber from a large motor neuron, and to send their own message to neighboring motor neurons, which are inhibited, thus trimming the edges of the active group. The receptor elements in the eye, similarly, send collaterals

to neighboring elements, which they also inhibit. This has the same influence in dissecting out a perceptual entity, for it serves to markedly accentuate the edge between two continuous patches of illumination. On the bright side, all receptors are stimulated to fire rapidly but are inhibiting each other considerably—except that those near the edge, having only slight inhibition from one direction, are considerably more active than their fellows deeper in the patch. An equivalent effect takes place on the dim side; none is firing rapidly because of light nor is being much inhibited by its fellows—except those near the bright edge, which are being strongly inhibited from the bright side. At the edge, therefore, the bright receptors are firing more and the dim receptors less than their fellows in the evenly illuminated patches.

These neural mechanisms for dissecting out entities are largely the product of racial experience and genetic mechanisms, but individual experience, left in material changes which subsume functional ones, are also strongly involved. Such building of the program by activity on an existing structural scaffolding was discussed above in connection with pattern vision development, and the motor story is comparable. The nervous system starts with primitive units of organization and puts these together in ever larger constellations. Simple acts, essentially reflex patterns, are combined into more complicated ones and these into still more elaborate performance patterns; as someone put it, the motor cortex plays upon the spinal units as a typist on the keys of her typewriter. Old studies in learning revealed the same progressive groupings; an operator learning Morse code telegraphy was found to improve rapidly at first until a plateau was reached at a rather low speed of message reading. Then, often after a surprisingly long time, a further rapid improvement set in, to reach a new asymptote; and in time a third step of improvement would be superimposed. It was

recognized that the first learning curve represented the recognition of letters, the second one the recognition of words as units, and the third one the recognition of phrases and sentences. This is an early demonstration of the recent formulation of perception by grouping "bits" into "chunks."

It is obvious that the motor neurons which are active in one motion are not entirely different from those active in another motion; intersecting sets of neurons are involved, with some common to many acts, others different in each. In exactly the same way, integrations at higher functional levels, playing upon the subordinate units, also engage these in individual but overlapping patterns. There is, thus, competition and interaction of neurons in progressive organizations or assemblies and, to the extent that a given memory is itself represented by the grouping or pattern of neurons which operate together, memories gain some of their interesting properties from this. I suspect that the phenomena of association, of interference, of proactive and retroactive inhibition, of the gradual shift in recall over time, and many of the other particular facets of memory of especial interest to psychologists, are more dependent on these interlocking neuron systems than they are on the particular chemical or physical change in any particular neuron or neuron group. Perhaps, even, there is a similar physiological explanation for the fact that older and more frequently used memory patterns are prepotent in determining behavior and in emerging into conscious memory and are best retained under various insults to the organism. If two different perceptions reaching consciousness, or plans leading to action, engage neuron pools which partly overlap; then, by the various inhibitory and other integrative mechanisms, one will come through in toto and the other be entirely suppressed. The one that will take precedence will be that which captures the moot interneuron group and so completes its full complement of active neurons. A stronger

input through one set than another will normally lead that one to prevail. But, other things being reasonably equal, the grouping with the best established connections—the junctions with the best transmitting properties—will be the one to capture the interneurons and complete the system. If the firmness of fixation continues to increase, even a little, on repeated use (which should be greater in older than in newer acquisitions), then ancient or rehearsed learned patterns will tend to prevail.

This symposium is concerned with memory as a scientific problem, but I cannot close without drawing an important practical conclusion from this knowledge of how experience writes itself into the structure and performance of the human brain. Clearly, it is of the greatest importance to expose the young individual to as much relevant experience as possible. It is now pretty obvious (and becoming rather widely accepted) that many of the more complex intellectual functions can be enhanced by early exercise, just as pattern vision is developed by looking at visual patterns. Man's great biological virtue is in his malleability, in which he clearly is the present culmination of the major sweep of evolution. A nervous system increased the malleability of organisms, and man's overstuffed brain, with twice as many uncommitted neurons, perhaps, as other brains (the functional neuron reserve), has given him the learning capacity of the group as well as of the individual. Man possesses collective and transmitted learning, culture, to a unique degree; the use of symbols, language, science, and, just upon us, computers and automata, makes possible a richness and complexity of interaction unmatched by any other system that we know. The problems of human interaction have mounted with its intensity; it seems obvious that it will be necessary to exploit man's potential intellectual capabilities to the fullest—by vastly improved educational methods—for him to keep up with his

own machines and to avoid technological  
obsolescence, if not biological elimination.

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