Interaction of Neuromodulatory Systems in Modulating Memory Storage

James L. McGaugh, Ph.D., Larry Cahill, Ph.D.

An implicit assumption guiding many studies of neurochemical systems involved in learning and memory in animal subjects is that animal and human memory systems use the same or similar mechanisms. Because acquisition and retention performance can be influenced by many processes other than information storage, special effort is required to distinguish influences on memory processes from other factors influencing performance. This article reviews the findings of recent studies investigating the effects of memory, of drugs affecting adrenergic, opioid peptidergic, GABAergic and cholinergic systems. The review focuses primarily on studies using posttraining treatments and tests for retention given no sooner than a day after the training. Extensive evidence suggests that such drugs interact within the amygdaloid complex and that projections from the amygdala influence memory storage in other brain regions. The assumption that comparable processes occur in animal and human subjects is supported by evidence that, in human subjects, emotionally influenced memory is blocked by a β-adrenergic blocker and by lesions of the amygdaloid complex.

1. Introduction

Many, if not most, experiments investigating drug effects on learning and memory in animals are based on the implicit assumption that the findings will contribute to understanding drug effects on memory in human subjects and that such findings might eventually be useful in developing treatments for disorders of memory. This assumption applies, in particular, but not exclusively, to studies of drug enhancement of learning and memory. In all such studies there is a common methodological problem that must be addressed: The effects of the treatments on learning and memory must be distinguished from other influences of the treatments on performance. It is essential that effects on sensory/perceptual, motivational and motoric processes be excluded if it is to be concluded that a drug improves performance through influences on neurobiological processes underlying learning and memory [53]. However, the distinction is not required if the research is aimed only at finding drugs that enhance cognitive performance. In human subjects, for example, drugs that increase alertness, attention and motivation should be expected to enhance cognitive performance and such effects may also influence learning and subsequent retention. But, such effects should be distinguished from direct influences of the drugs on neurobiological processes underlying learning and memory. Similarly, in studies of drug effects in animal subjects it is critical to distinguish drug effects on learning and memory from influences on processes such as shock sensitivity, appetite, degree of arousal and ability to

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From the President

This is my final Presidential Column. I have been extremely privileged to have such a great Board of Directors to work with. The Board has been very active throughout the year and without the teamwork that has been displayed throughout the year, great things would not have come about. As I look back over the year, we achieved a victory in the Industrial Medical Council's policies on biofeedback in certain pain disorders, our web page continues to look great and links us to other sites of interest, and our new administrative office is humming along efficiently. Probably our crowning achievement is the wonderful conference that has been planned for November 14-16th at the Orange County Airport Hilton. I hope you will be able to attend. As I step down from the presidential post at the November conference, I am excited to say that we are already lining up some great events in 1998. Thanks again, not only to the Board of Directors for all of their work but to everyone else who has helped in some way to keep the BSC thriving and supporting the spirit of biofeedback.

Stephen Francis, MA

Editor's Special Report

Moves to change the relationships between physicians, health practitioners and the HMO insurance companies

Reports from several media sources indicate that there is a recent renewal of the motivation and drive to change some of the laws which govern the relationship between health maintenance organizations or HMO insurance companies, health practitioners and patients. The stronghold of these companies and their goal to maintain the lowest possible costs of health management by herding more and more people into the HMO system is causing an organized response in Sacramento. It is reported that legislators are deliberating how to change the way in which the insurance companies' agendas deal with professionals and patients. These criteria are supported by groups of associates consisting of client/patients/customers' groups together with the professionals who treat them.

There is opposition to the dynamic efforts of these consolidated individuals and groups of course, from the HMOs and those who share a common anxiety that if costs escalate any more, small-business employers will not be able to afford coverage for their workers. It is ironic that this move to change the laws is originating in California, the state which initiated the HMO idea! Statistics revealed how 50% of the 33 million California residents obtain their health care from one HMO or another. The reason for the new movement in legislation is the perceived lowering of the quality of attention and service which the HMO patient/customer receives. Difficulties in making timely appointments or being kept on telephone hold for long periods are becoming unacceptable to many individuals. Denial of approval for specialist treatment is a major source of discontent. Generally, it is felt that the HMO priority is profit first and patient care a low second. Jamie Court, director of Consumers for Quality Care, an advocacy group in Santa Monica was quoted as saying— "The popularity of these issues with legislators shows that they

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Special thanks to Gloria Passarella for all her work in securing ads for the California Biofeedback newsletter for 1997.
California Biofeedback: International Comment

The following is an item received from an EMG biofeedback practitioner who received “California Biofeedback” in Hong Kong. His contribution is titled: “Improvement of Existing Biofeedback Devices: Instrumental Aspect.” by Wong Yiu Ming, M.A.

The practice of electromyographic biofeedback is not a very popular modality of rehabilitation in HK because some therapists find the EMG signal to be very unstable. Since many EMG devices imported from the United States are shipped with a notch filter for 60 Hz and the HK power line frequency being 50 Hz, signal noise cannot be filtered out effectively. Surprisingly, some user and even distributors are unaware of this critical point. If any manufacturers of EMG monitoring equipment want to promote their products in HK, they would do well to provide an optional notch filter at 50 Hz.

Most portable biofeedback instruments (e.g., electromyography) that contain a light display for visual feedback showing physiological change generally have no capacity to trigger an appliance like a walkman or toy-train for optional feedback. Just adding a light-activated switch to an appliance you select, will enable that appliance to be triggered and activated immediately when the light display is on when the pre-set threshold is reached. Because the light-activated switch has a feature that can only be turned on under the presence of light.

Example: EMG-Controlled Walkman

The light-activated switch (TY-11, Sound Master Electronics, HK) costs less than US$6.00 and can be turned on by the light as little as the green LED (light-emitting diodes) of the Myotrac (Thought Technology, USA). The switch can be connected to a walkman that provides music as feedback. Placement of the photocell of the switch toward the LED depends on the treatment goal such as muscular strengthening or relaxation. An example of positioning might be in front of the red or green LED. When the pre-set EMG threshold is reached and the LED is triggered to an “on” position, the walkman will also be triggered on immediately to provide musical feedback. One must be aware that if surrounding lighting is very bright, the switch can be turned on too by this intensity, so simply covering the LED with a cloth over the LEDs and photocell can solve this problem.

Advantages and disadvantages

The above-mentioned design has been used for two years in my clinic and was found to be accurate and reliable, inexpensive, portable and easy to install. However, the design provides time-dependent and rewarding feedback but not a volume or intensity-graduated proportional one, i.e. it is digital meaning either on or off.

For anyone interested in further information or discussion on the topic of creative EMG feedback applications please feel free to contact Mr. Wong Yiu Ming at e-mail: emg@net.polyu.edu.hk or P.O. Box 79767, Mongkok Post Office, Hong Kong.

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make the appropriate motor response. Before concluding that a drug has cognitive-enhancing effects, it is essential to control for other influences of the drug that might enhance performance.

2. Drug and hormone influences on memory storage

A paper in 1917 by Lashly [43] was the first to report enhancing effects of a drug on learning. He reported that strychnine administered to rats prior to each day’s training in a maze enhanced their rate of learning. Because the drug was administered before training, the effects may have been due to influences other than learning and memory that influence maze performance. Subsequently, the findings of many experiments indicated that memory in maze tasks, as well as other training tasks, can be enhanced by posttraining injections of stimulant drugs, including strychnine, picrotoxin, pentylentetrazol, bemegride and amphetamine [21,51,55]. These findings strongly supported the hypothesis that drugs can enhance memory by modulating posttraining neurobiological processes underlying memory storage. The findings also strongly suggested that memory storage may normally be modulated by endogenous neuromodulatory systems activated by learning experiences [17,19]. It is well established that the adrenergic hormone epinephrine is released by stimulation of the kind normally used in learning experiments using rats and mice. Furthermore, there is extensive evidence that, in a variety of training tasks, including both appetitively and aversively motivated tasks, memory is enhanced by posttraining administration of epinephrine [18,36,57,74]. Such findings suggest that epinephrine released by training activates brain processes regulating memory storage. Evidence concerning the brain systems that may mediate the epinephrine effects on memory is discussed in a subsequent section.

It is also now known that, in rats and mice, memory can be influenced by other neuromodulatory systems, including opioid peptides, GABAergic, and cholinergic systems [52] and that these systems interact in modulating memory storage (see Table 1). Findings of many studies indicate that, in experiments using posttraining systemically administered injections, retention is enhanced by opiate antagonists, including naloxone and naltrexone [15,37,39,62] and impaired by morphine and β-endorphin [11,27,39]. Furthermore, evidence from several studies suggests that opiates affect memory storage through an interaction with a β-adrenergic system. The β-adrenergic antagonist propranolol blocks the memory enhancing effects of naloxone [40], and the effects of epinephrine and naloxone on memory are additive [29] as are the effects of propranolol and β-endorphin. Similar interactions are found with the GABAergic system.

3. Brain systems and modulation of memory storage

Most of the drugs used in the experiments summarized above readily enter the brain. Paradoxically, an important clue to the locus of action of these drugs in influencing memory came from studies of epinephrine, a hormone that does not readily enter the brain. It is well established that the amygdaloid complex is activated by emotionally arousing stimulation. And, it is known that electrical stimulation of the amygdala after

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INTERACTIONS

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All treatments were administered systemically immediately after training inhibitory avoidance training and retention was tested 1 day or longer after training. +, +, +, =, +, + +, + denote memory enhancement, additive or potentiated enhancement, attenuation (or blocking), impairment and additive or potentiated impairment respectively.
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training can produce memory impairment or enhancement, depending upon the experimental conditions used (20,41,56). Furthermore, retention is impaired by posttraining inactivation of the amygdala with lidocaine [13].

Retention is also impaired by posttraining intra-amygdala injections of propranolol [16]. Additionally, intra-amygdala injections of a low dose of propranolol block the memory enhancing effects of peripherally administered epinephrine [45]. Epinephrine effects on memory are also blocked by systemic injections of the β-adrenergic antagonist sotalol, a drug that acts peripherally, suggesting that epinephrine effects on memory are initiated by activation of peripheral receptors [36]. The findings of several experiments suggest that the adrenergic receptors are located on vagal afferents projecting to the nucleus of the solitary tract (NTS) and that projections from the NTS release norepinephrine within the amygdala [69,78,79]. Considered together these findings clearly suggest that retention should be influenced by administration of noradrenergic agonists into the amygdala. Evidence from several studies support this implication. Retention is enhanced by posttraining intra-amygdala injections of low doses of NE and the noradrenergic agonist clenbuterol [34,47]. Furthermore, and most importantly, as is shown in Fig. 1, footshock stimulation comparable to that used in inhibitory avoidance training induces the release of NE within the amygdala of rats [54]. This finding is consistent with evidence from other laboratories indicating that stress induces NE release in the amygdala [22,75,77]. Other evidence implicating the amygdala in mediating adrenergic influences on memory include the findings that, in rats, lesions of the amygdala [5] block the epinephrine effects and that lesions of the stria terminalis, a major amygdala pathway, block the memory enhancing effects of epinephrine as well as those induced by intra-amygdala injections of noradrenergic agonists [34,46,47].

Furthermore, as is shown in Fig. 2, the adrenergic agonist clenbuterol blocks the memory-imparing effects of β-endorphin in an inhibitory avoidance task [22] and a water-maze spatial task [213] when both drugs are administered intra-amygdally. And, in both tasks, low and otherwise ineffective doses of β-endorphin and propranolol impair memory when injected together intra-amygdally [33].

The consistent finding from studies using systemic as well as intra-amygdala injections of drugs affecting adrenergic, opioid peptidergic, and GABAergic systems is that memory is enhanced by treatments that induce the release of NE and that memory is impaired by treatments that reduce NE release or block NE receptors in the amygdala. Thus, noradrenergic activation within the amygdala appears to integrate converging neuromodulatory influences initiated by training. However, muscarinic cholinergic activation in the amygdala also appears to play an important role in mediating the noradrenergic influences.

Furthermore, as would be expected on the basis of the evidence summarized above, muscarinic cholinergic drugs also override the memory modulating effects of drugs affecting opioid peptidergic and GABAergic systems [12,26]. The effects appear to be mediated by efferents from the amygdala because lesions of the stria terminalis block the effects of muscarinic cholinergic drugs on memory [30]. The evidence that there are muscarinic cholinergic receptors and neurons within the amygdala also supports the view that amygdala noradrenergic influences on memory are mediated by activation of cholinergic neurons within the amygdala [48,64].

4. Locus of memory storage modulated by the amygdala

Although there is extensive evidence suggesting that the amygdala is a locus of neural changes mediating emotional memory [14,44], the converging evidence reviewed above strongly suggests that the sites of neural plasticity modulated by amygdala activation are in located brain regions influenced by the amygdala and are not located within the amygdala. Other recent findings of experiments investigating the effects of amygdala lesions induced after training provide additional support

Fig. 1. Effect of a 0.55mM/1.0 s footshock on norepinephrine release within the amygdaloid complex. *P<0.005 vs. 100% of baseline NE. Time has been corrected for tubing length from the microdialysis probe to the collection vessel and for the flow rate of the CSF in order to represent the actual time of release. (From Gaddy, Musches and McGaugh, 1996)

Fig. 2. Effects of intra-amygdala injections of β-endorphin (0.1 ng) and clenbuterol (10.0 or 30.0 ng) on retention of inhibitory avoidance training (A) and water maze spatial training (B). Data are expressed as means ±SEM. For (A), **P<0.01 vs. all other groups. For (B), *P<0.01 vs. groups given buffer, clenbuterol (10.0 or 30.0 ng) or clenbuterol (30.0 ng) + β-endorphin and 0.05 vs. clenbuterol (110.0 ng). (From intraini-Collison, Ford and McGaugh, 1995.)
for this view. In a series of experiments [72,73] rats were first given 10 or 20 escape training trials in a two-compartment straight alley. In different experiments neurotoxic (NMDA) amygdala lesions were induced either 1 week or 21 days after the training. On subsequent retention tests the animals showed significant retention of the prior training as indicated by learning of inhibitory avoidance in the same apparatus. And the retention performance reflected the amount of prior escape training. Clearly, the lesions of the amygdala did not block retention of the escape training. Comparable results were obtained when degree of original training was varied by giving the animals training footshock of different intensities [71]. The role of the amygdala in retention was addressed by another recent experiment in which rats received posttraining injections of amphetamine into the amygdala, caudate, or hippocampus immediately after they were trained in one of two tasks in a water maze: a spatial task, or a cued object task [65]. The animals were trained, in a series of trials on 1 day, to swim either to a submerged platform located in a constant position (the spatial task) or to a visible platform placed in a different location on each trial (the cued task). Retention was tested (by retraining) the following day. The findings indicated that amphetamine injected into the caudate selectively enhanced retention of the cued task whereas amphetamine injected into the hippocampus selectively enhanced retention of the spatial task. In contrast amphetamine injected into the amygdala enhanced retention of both tasks. Furthermore, inactivation of the amygdala prior to the retention test did not block the memory enhancing effects of the posttraining intra-amygdala injections of amphetamine.

These results provide additional evidence that the amygdala is involved in regulating memory storage and that the amygdala is not the locus of long-term memory influenced by amygdala activation. The finding that activation of the amygdala enhanced a cuedependent learning task as well as a hippocampal-dependent task [66-68] strongly suggests that the amygdala influences neural activity, and possibly memory storage, in these brain regions.

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Results fit well with findings indicating that retention is impaired by posttraining inactivation (with lidocaine) of the basolateral nucleus of amygdala [70] as well as evidence that lesions of the basolateral nucleus block the memory impairing effects of benzodiazepines [76].

5. Emotionally influenced memory in human subjects

The findings summarized in this article provide substantial evidence that memory storage is modulated by activation of the adrenergic system and that such effects are mediated by the amygdala. Findings of recent studies using human subjects provide additional support for these conclusions. In one experiment, subjects viewed slides of a narrated story which induced emotional arousal in the middle of the story (see Fig. 3). Control subjects given a placebo before viewing the story showed enhanced retention of the information associated with the emotional arousal. Other subjects were given propranolol prior to viewing the story. Propranolol impaired memory for the emotional portion of the story but did not affect memory for emotionally neutral portions of the story [6]. Thus, in human subjects, as in animal subjects, retention is influenced by activation of the adrenergic system. These results are consistent with other recent evidence that chronic adrenergic blockade also attenuates the enhancing influence of physically induced arousal on memory [63]. Additional evidence suggests that activation of the amygdala in humans is critical for enhanced memory associated with emotional arousal. A recent experiment examined memory for the emotionally arousing story described above in a patient with bilateral degeneration of the amygdala [4]. Although this patient reported the story as being emotionally arousing, he displayed no evidence of enhanced memory for the emotionally arousing portion of the story.

6. Conclusions

A fundamental question in studies of drug effects on animal learning and memory is that of whether the findings have any implications for understanding drug effects on human learning and memory. The findings summarized in this paper provide support for the view that findings of animal studies do provide information of significance for human studies. The findings of the animal and human studies provide strong and consistent evidence that memory storage is modulated by adrenergic activation and that the amygdala is critical for integrating the neuromodulatory influences activated by learning. Further, the findings of the animal and human studies provide strong evidence that an intact and normally functioning amygdala is not essential for long-term retention and are thus consistent in suggesting that the amygdala modulates long-term memory storage in other brain regions.

References on request from the editor at (415) 647-2642.
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are salient issues with the public.”

Criteria are quite comprehensive including the matching of reimbursement to mental health professionals with that of physical health services. Another criterion is allowing new mothers to have a longer stay in the maternity beyond 48 hours post-delivery. A universally acknowledged issue is the returning of the decision-making capacity in matters of health care from the HMO business employee to the appropriate professional. There are many examples of unnecessary suffering because of bureaucratic cost-cutting decisions being the priority. One motivating factor in this new “movement” is the first-hand experience of so many influential individuals who themselves, their families and friends have received shoddy cost-cutting services, denials of payment for necessary medications, not to mention effective preventive integrative medicine approaches such as biofeedback and applied clinical psychophysiology and other services. Physicians are complaining and are perceived as becoming stronger in their united efforts to change the scenario of sacrificing quality care for low-costs and larger profits. Of course, the incomes of health practitioners in all fields of care are suffering thus, reducing the acknowledgement of the extensive training and importance of the work they do including the loss of professional esteem for health professionals, collectively.

The personal physicians of numerous legislators are sharing their negative experiences as a consequence of the HMO environment with each other and the unification of professionals and patients is reported to be getting stronger. When a physician prescribes a medication or refers a patient to a biofeedback therapist as a case of medical necessity and the HMO says “no,” people are getting upset and feel that the situation is wrong. Right now, a bill is being sponsored which will require all decisions regarding approval or denial of a health service to be made or supervised only by medical directors licensed in California and they would be held responsible for the outcome of these decisions. One measure is for HMOs to clarify and explain how the insurance company arrives at its “financial formula” for paying health professionals and how quality care can be provided on the remuneration they receive.

One side of the argument is that if health service restrictions are relaxed by the HMOs this would result in increased premiums and make lower income people unable to afford basic health insurance thus, making those who need care the most unable to afford it. As pressure in the state capitol Sacramento increases to change the current situation it is believed that some form of change will take place. Some health care topics which have already been passed at least one house of the Legislature are: after hospital discharge, the patient must have sources of adequate care — physician-health plan contracts to be made requiring 120 days of notice before termination with a requirement to state the exact reason for the termination — a significant reversal in waiting times either on the phone and for same-day scheduling with the professional — disclosure of criteria leading to denial of health care and that decisions to be made only by a qualified physician — mental health care to be paid at the same basis as physical care — disclosure of HMO payment and how quality care can be provided for this amount — prohibiting incentive payments to physicians to induce denial, reduction of or delay of medically necessary and appropriate services. In conclusion, it looks like some positive changes will result from the current legislators and their collectivity of drive and motivation and when the changes do come we, biofeedback professionals in our various fields will feel some benefits too.

As a final editorial note from me, this being my last issue as editor of “California Biofeedback” it pleases me to be able to report some positive news regarding potential new changes in the current HMO laws and jurisdictions. I have enjoyed very much the experience and honor of being editor for the last year and offer my very best wishes to the new editor for 1998 and to the members of the BSC individually and collectively.

Thomas G. Browne, M.A.
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