

PHYSIOLOGICAL PSYCHOLOGY.

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It is proposed in this review to deal only with those aspects of physiological psychology that have an immediate bearing on the problems of human behaviour. Here again two limitations must preclude consideration of much important work in this field, in that endocrinology and the study of cortical potentials have been relegated to the special articles dealing with this subject.

The action of drugs on mental processes has been far too little studied in the past, and the three years under review have added little to our knowledge of a subject which properly understood should, in itself, furnish important clues to most of the problems of human behaviour.

Benzedrine.—The stimulating action of this drug has been the subject of much work from the therapeutic angle, but its alleged effects on mental processes are still very far from being objectively verified.

Mann and Quastel (1940) have been able to suggest a biochemical basis for the alleged stimulant action. They found that the oxidation of glucose by the cerebral tissue is inhibited by tyramine, isoamylamine and a number of similar amines. Benzedrine competes reversibly with tyramine and the related amines for amino-oxidase, and thus reduces the aldehyde-forming power of the amines and the resultant inhibition of glucose oxidation. It would be interesting to inquire in view of the almost specific therapeutic actions of benzedrine in neurology whether there is any evidence of abnormal aldehyde formation in this condition. These authors have studied the excretion and detoxication of the drug, which Beyer (1939) has shown to have a prolonged action on the general metabolism. Using a diazo titration colour reaction they found that less than half of the injected dose is excreted in the urine. The effect of benzedrine on intellectual activity has been the subject of much discussion. Davidoff and Reifenstein (1939) found that the results of benzedrine administration were characterized by uncertainty and unpredictability. Carl and Turner (1939) found that the drug had little effect on accuracy, but that speed of work was increased. Holch, Thornton and Smith (1939) found that the improvement effected by benzedrine on the tapping time, handgrip and steadiness tests was greater than that obtained by caffeine administration. Golla and Blackburn (1939) introduced a new method in their investigation of the effect of benzedrine on speed of movement and simple mental processes. They were impressed by the great difficulties encountered in any attempt to demonstrate alterations of time in performance with the usual tasks set in psychological experiments, due not only to awareness of the expected result, but to the difficulty of establishing a norm. For this reason whilst the subject was undergoing the usual standard tests, a number of habitual reactions

were timed without the subject being aware that this was being done. Thus the time taken in signing his name, getting up from chair, crossing the room and sitting down, taking up a pen, etc., furnished a series of habitual reactions which the subject was unconsciously offering as a test and which were unsusceptible of improvement on practice. They found a significant acceleration of performance under benzedrine, and a similar less pronounced effect with the closely allied drug isomyn.

Caffein has been shown to have a stimulating action on colour sensitivity and on certain psychomotor performances. Holck, Thornton and Smith (1939) found that caffein benzoate in doses of 300 mgm. produced an acceleration of tapping times and speed of reaction, together with an increase of the handgrip, whilst unsteadiness was also increased. Kravkov (1939) obtained a 40 per cent. increase in colour sensitivity to reddish orange and green after administration of 100 mgm. caffein.

Alcohol.—Mead (1939) found that 30 c.c. of pure ethyl alcohol administered in 150 c.c. of water decreased the percentage improvement in an artificial language test, but had no effect on a conditioned finger withdrawal test.

Barbiturates.—Robinson (1939) claims that tolerance to barbiturates is easily established, the patients requiring larger and larger doses, which may eventually lead to clinical evidence of toxicity. He has observed cases of barbital psychosis. Hambourger (1940), in an analysis of hospital cases, found that barbiturates accounted for more than 10 per cent. of the drug addicts, excluding alcoholics.

Tobacco.—Johnson and Short (1939) have investigated the reactions of the human body to epinephrin and tobacco smoke. They find a parallelism that they regard as furnishing evidence in favour of the view that toxic reaction to tobacco smoke is occasioned by an increased output of adrenalin.

Pain.—Goodell, Hardy and Wolff (1940) investigated pain sensibility by focusing the light from a 1,000 watt lamp on to a blackened area of the forehead 3.5 sq. cm. in area. The intensity of the radiation was measured by substituting a radiometer for the skin area. With this method they found that the pain threshold remained surprisingly constant for over a year. Heat and pain were differentiated by the action of acetosalicylic acid, which raised the threshold for pain and lowered it for heat. The peripheral end-organs for pain were found to be unaffected by ischaemia, whilst the thermal sense was abolished. Together with Schumacher (1940) these workers were able to show by their method that differences in the reaction of individuals to pain are not the result of individual variations in the pain threshold, but express the characteristic individual response. The reaction to pain at different times of the day may vary greatly while the threshold remains constant. Goodell, Hardy and Wolff (1940) showed that the pain threshold-raising power of morphia can be reduced or obliterated by severe pain experimentally produced before the administrations of the morphia, and a similar reduction could be effected by the previous administration of adrenalin. In spite of this reduction of the power of the drug to raise the threshold of pain, they find that the emotional reaction may still be markedly subdued. Lewis (1938) has drawn attention to the possibility of educating patients by the use of simple stimuli to differentiate

clearly pain from skin, muscle, finger, tendon and periosteum, and to contrast such pain with that originating in deep structures. He points out that information from a patient so educated may greatly facilitate diagnosis. In a review of experimental studies on headache Wolff (1939) concludes that distension of the cranial arteries produces headache, and that the headache of migraine arises almost always from distension of the branches of the external carotid. Kunkle, Ray and Wolff have contributed an important study on headache, in which they point out that increased intracranial pressure is neither a prime nor an essential factor in the production of headache, which results from traction on or displacement of pain-sensitive intracranial vascular structure and is independent of pure generalizing intracranial pressure changes.

Achelis (1939) instances a number of observations which appear to form a summation theory of pain, and to be incompatible with the specification of pain-mediating systems, whether from the anatomical or the functional point of view. Thus in a hyperalgesic zone pressure spots disappeared; he suggests their conversion to pain spots. On the other hand, if appropriate methods of stimulation are employed it is possible to elicit pure tactile sensations from the cornea—an area which has been supposed to be supplied with exclusively pain-mediating nerve-endings. Rowbotham comes to a similar conclusion.

Tickle and itching.—Chordotomy causes a loss of tickle sensation on the analgesic side though tactile sensation is unimpaired. After chordotomy itching may be lost as well as tickle sensibility in the affected area. Bickford (1939), using histamine puncture, provoked spontaneous itching at the point of puncture, accompanied by a gradually developing change in the surrounding skin, which is evidenced by the appearance of a generalized itching sensation. A certain degree of asphyxia will abolish the itching skin, whilst leaving spontaneous itching unaffected. This is held to indicate different nervous mechanisms for the two conditions.

The phenomenon of tickle vanishes with anaesthesia *pari passu* with the itching skin. Some evidence is advanced that itching skin and possibly tickle is moderated by axon reflexes in a plexiform arrangement of nerve fibres.

Vasomotor responses.—Mittelmann and Wolff (1939), using the Hardy radiometer, made a number of observations on 47 subjects. In all but two subjects a drop in the temperature of the skin of the finger occurred under emotional stress. Affective states were induced by a discussion dwelling on difficulties affecting the subject's life to which he reacted with signs of emotion. The physical environment was, of course, controlled as to temperature and humidity and the relaxed subject was lying lightly covered, and during the control period the skin temperature remained fairly constant between 31° and 36° C. When an affective state was aroused the temperature of the fingers occasionally fell below room temperature.

Self-regulation.—By self-regulation is meant the phenomenon of self-regulation by the organism of its intake, so as to balance deficiency caused either by the absence of certain constituents in the diet or their defective metabolism. It may be objected that this subject is not one that can logically be included under physiological psychology, inasmuch as the teleological orientation is not found to be conscious. On the other hand, a knowledge of the causation of this

behaviour type is not only important as a contribution to the physiology of metabolism, but from it one may legitimately hope to learn something of the conditioning of other desires and repulsions.

An excellent review of our present knowledge of this subject is contributed by Richter to vol. iv of the *Annual Review of Physiology*. The review deals with papers published on instances of self-regulating activity in connection with the nutritive functions and the neurological mechanisms involved, with instances in which the self-regulating mechanism breaks down. Among some of the very interesting points made are the following: The evidence to hand indicates that the self-regulating process in role depends largely on taste and not on other forms of experience. After section of the taste-nerves adrenalectomized rats failed to differentiate between sodium chloride solution and water. Rats accepted or refused solutions of electrolytes according to their dietary value in extraordinarily low concentrations to which normal rats showed no preferences. Reference is made to the work of Bornstein (1940), who has concerned himself with a review of our knowledge regarding thalamic and cortical gustatory systems, and he concludes that the functional relations which taste bears to smell predicate a convergence in the cortex of these two kinds of sensations rather than separate taste and smell systems. These studies on self regulation indicate that the effort to maintain a constant internal environment constitutes a fundamental behaviour drive. The self selection technique gives us a method of determining chemical and metabolic needs that could only be discovered by biochemical methods after prolonged experimentation. Instances of clinically observed markedly unusual behaviour may often be considered to be manifestations of efforts on the part of the patient to maintain his internal environment. Thus, of course, polydipsia in diabetes insipidus is an effort to compensate for an increased loss of water.

Emotion.—According to Spiegel, Miller and Oppenheimer (1940), the rage reactions appear to be lodged in dogs and cats in the olfactory tubercle, the amygdaloid nucleus and the hippocampus fornix system. Bilateral lesions of these structures left the animal susceptible to outbursts of rage, while lesions of the neocortex failed to produce this reaction. Destruction of the olfactory bulbs was, however, without effect on the animals' behaviour, and Wolff (1939) removed the neocortex from three cats, avoiding damage to the olfactory system. Though these cats were active they never showed any signs of rage when handled. Masserman (1943) has subjected the whole question of the hypothalamus as a centre for emotion to a critical and experimental review. He concludes that animal experimental work on the hypothalamus may possibly reinforce the effector neural responses controlling some of the sympathetic and motor manifestations of fear and rage; there is little or no basis for the thesis that the hypothalamus governs or even mediates the motor experiences themselves. He points out that in man symbolization has to a large extent replaced more visceral methods of expression, and this fact alone may explain the paucity of reported cases of "sham rage" in human beings.

Grinker (1939), on the strength of his oscillographic records of the potentials resulting from hypothalamic stimulation in the human, together with the emotional results of such stimulation, supports the view "that anatomical or

functional lesions in the hypothalamus may cause complete or partial release of cortical (ideational) control of the emotional content of higher activities. The dominant external activity would then be not a slow adaptive response produced by a process of long circuiting, but a less deliberate (impulsive) response with accompanying vasomotor correlates of emotional reactions."

Sleep.—Ranson has called attention to an interpretation of the function of the hypothalamus that should have been sufficiently obvious in that the so-called sleep centre is in reality a centre for maintaining the condition of wakefulness. Bilateral destruction of the hypothalamic area in monkeys was followed by profound somnolence. In view of the fact that descending tracts from the sympathetic nuclei of the hypothalamus traverse this area, it might appear that somnolence may be attributed to the elimination of the normal discharge from the hypothalamic centres. Harrison (1940) has substantiated the views of Ingram, Barres and Ranson by effecting bilateral lesions in the hypothalamus of cats and monkeys. Succeeding the lesion the animals present for a number of days an appearance of catalepsy and acute somnolence. In those animals surviving the lesion the somnolent condition gradually disappears, leaving the animals much more tractable and unemotional. In wild Rhesus monkeys bilateral extirpation of the mammillary bodies causes the animal to become friendly and tame. Serota (1939) measured the thermal relations of various areas of the cat's brain by thermo-electric couples. He found that in both the conscious and the anaesthetized animal the basal region was warmer than the cortex. When emotional states were induced, such as fear or rage or anticipation of food, the temperature of the hypothalamus increased. During sleep the hypothalamic temperature sank, and rose again on awakening, earlier, and to a greater relative degree than that of the cortex, the caudate nucleus or the cornu Ammonis. These changes were not thought to be due to increased blood flow such as Gibbs has recorded in active cortical areas, but to increased metabolism.

Nygaard (1939) recorded the brain volume with a tambour over the skull opening of patients who had undergone temporal decompression. He confirms previous work in finding an increased brain volume during sleep, which is accompanied by slowing of the heart-beat and slight decrease of blood pressure. It was found that all these circulatory changes actually preceded the onset of sleep. Schnedorf and Ivy (1939) have revived the now generally discarded hypnotoxic theory of sleep. They withdrew 8 c.c. of cerebrospinal fluid from a normal dog and replaced it by an equal amount from dogs kept awake 7-16 days. The reaction is said to have been the induction of sleep or a sleepy state in the normal dog. It is obvious that without further chemical and physiological data these findings cannot be accepted as direct evidence of a hypnotoxic explanation of sleep.

Sexual behaviour.—Apart from the mass of endocrinological evidence which will be considered under the appropriate heading, there is some recent experimental evidence throwing light on the relations of the central nervous system to sexual behaviour. The observations of Brooks (1940) support the theory of the neurogenic control of the gonadotropic hormone in relation to ovulation. Not only does he confirm the observation of Westman and Jacobsohn that

ovulation in the rabbit is absent after coitus in animals whose pituitary stalk is sectioned, but he observes that rabbits with the transected stalk fail to show any alteration in the amount of luteinizing hormone in the anterior hypophysis after mating, whereas it decreases regularly in unoperated control animals. Westman showed that when the excitability of the brain is abolished by novocaine, ovulation is temporarily suppressed provided that novocaine is injected intracisternally a few minutes after copulation. If it is injected later ovulation occurs (Gellhorn, 1942).

These and similar observations are of interest in the study of sexual psychology in that they suggest that abnormalities of sexual conduct may be neurologically conditioned, and that the endocrine influence may occasionally be a secondary factor. Brookhart and Dey (1941) found that male guinea-pigs subjected to hypothalamic lesions show either a greatly reduced or no sexual activity, although spermatogenesis continues and the sexual vesicles remain intact. Papanicolau and Seward (1939) produced proof that hormones may act directly on the central nervous system by inducing oestrus after double hysterectomy when oestrin is injected. The experiments of Dempsey and Rioch (1939) show that sexual behaviour in the female guinea-pig is controlled by a centre located caudal to the anterior margin of the mammillary bodies and rostral to the intercollicular level. They bring evidence to show that the site of action of both oestrone and progesterone is in this area. Further experiments localized an afferent pathway for sexual impulses in the posterior columns of the spinal cord which decussates below the posterior corpora quadrigemina and runs through to the roof of the mesencephalon. They further showed that normal mating behaviour was not abolished by brain lesions in the septal and preoptic regions or after section of the pituitary stalk.

Cortical function.—Little has been added to our knowledge of cortical localization of function during the years under review.

The frontal lobes.—The results of prefrontal leucotomy as a method of treatment of certain psychoses are discussed elsewhere. They have naturally stimulated renewed interest in the function of the prefrontal area. Rylander (1939) undertook a detailed investigation of a number of cases of unilateral frontal lobectomy, and on the basis of 32 cases and an equal number of controls he considered that it was possible to give some quantitative expression of the degree of mental defect caused by the lobectomy. Thirty of the 32 patients showed a mental condition after operation which was markedly different from that presented before the onset of the pathological condition necessitating the surgical procedure. The two exceptions were a case of a cyst and of an abscess. In both of these cases there had been no mental disturbance before coming to the operation. Only cases in which the operation had been successful in removing the tumour were considered. The patients were observed for months and in some instances years after the operation. Changes both in emotional tone and loss of emotional control were the most common symptoms noticed. Euphoria and loquacity were present in 20 cases. There was a general loss of shyness, perversity and compulsion states in a number of the patients. Rylander found that all patients of pyknic habitus and syntonic personality reacted with euphoria, while those who were definitely leptosome and schizoid showed

some degree of depression. Mixed types of physique showed a corresponding mixture of affective tone. Lack of tact and consideration for the feelings of others was one of the commonest syndromes. Twelve patients showed impairment of initiative, of which they were themselves fully aware. Rylander agrees with Brickner and Goldstein (1940) in considering that the power of abstract thinking is affected. No difference between the results from right or left frontal lobectomy could be ascertained. Stookey, Scarff and Teitelbaum (1941) report a series of 23 cases of unilateral frontal lobectomy in which the intelligence was tested after operation. They report that "when the lesion was completely removed little or no impairment of intellectual function resulted; emotional and social adjustments were satisfactory, and there was no loss of inhibition or spatial orientation." It may be summarily stated that all the evidence so far adduced in the period under consideration is in favour of Jefferson's original conclusion that if no marked symptoms are present before operation, none are to be detected after unilateral frontal lobectomy.

Bifrontal lobectomy is necessarily an uncommon operation, but should afford unequivocal evidence as to the functions of the premotor area. Brickner has published a follow-up investigation of his famous case of bilateral lobectomy performed by Dandy in 1930. He believes that lack of control over emotion is the dominant symptom. The patient is alternately boastful, angry, aggressive, negativistic, etc. In the intellect there is some degree of impairment of the power of synthesis and analysis. Slowness and rigidity of thought is accompanied by lack of insight, and the patient attempts to mask his defective comprehension by facetiousness and gross humour.

The operation of prefrontal leucotomy is discussed elsewhere. Most of the evidence summarized in the monograph of Freeman and Watts and the recent papers by Hutton, Fleming *et alii* point to a diminution of self-consciousness without intellectual impairment in those cases where no dementia had manifested itself before the operation. The latest cortical area phylogenetically to develop, it is perhaps not unreasonable to think that the prefrontal area subserves the last acquired mental process of self-consciousness. Brickner (1940) has recently made an addition to our knowledge of the functions of the frontal lobes by the discovery of an area of the frontal cortex, stimulation of which causes repetition. This is area 6 on the mesial side of the left hemisphere just above the junction of that area with the posterior part of area 32. A patient who was operated on under local anaesthesia was instructed to repeat the alphabet; when the stimulus was applied the patient stopped his sequence of letters at the letter which was being enunciated at that moment, and proceeded to repeat that particular letter again and again for such time as the stimulus lasted. As soon as the stimulus was removed he resumed his alphabet recitation. It is difficult to formulate any hypothesis as to the nature of the neural events underlying this phenomenon. It is to be hoped that a more psychologically directed analysis of the patient's experience during the period of perseveration will throw light on this problem when the experiment is repeated. It is, of course, highly desirable to ascertain whether the perseveration is confined to verbal action, or may manifest itself during any other sequence of movements.

Temporal lobes.—The ablation of the temporal lobes has taught little as to their function from the point of view of the psychologist. Klüver and Bury (1939) performed bilateral temporal lobectomy on the monkey; they noted psychic blunders and unintelligent reaction to environment stimuli. There was some evidence of a loss or diminution of fear and anger responses and of the occurrence of hypersexuality. There were no major sensory or motor disturbances. After adequate training the animals were able to exercise simple visual discrimination. No other operation procedures gave rise to the same syndrome except complete interruption of the occipito-temporal communications. Hebb (1939), after extirpation of the right temporal lobe in men, noted an impairment of abstract thinking, though the patient remained alert and responsive.

Corpus callosum.—The tentative treatment of some cases of epilepsy by section of the corpus callosum in the hope that the discharge might be thereby rendered unilateral has given an opportunity to learn something of the mechanism responsible for the co-ordination of the cerebral hemispheres. Previous evidence on this subject had been complicated by the fact that all cases reported as having had section of the corpus callosum performed were suffering from gross lesions of the central nervous system. In a number of cases suffering from idiopathic epilepsy without any symptom indicating the existence of a gross cerebral lesion, the operation of division of the corpus callosum was performed by Van Wagener and Herring (1940), and the psychological state of the operated patients was investigated by Akelaitis and Parsons (1942). Neither apraxia or astereognosis was noted in any instance. Personality changes were not noted more frequently than in patients in general who undergo serious operations in the hope of relief from seizures. Speech records showed no variation from the normal in tone, pitch or other qualities. Studies of visual fields made when the splenium was divided showed no alterations. Such patients do not exhibit the temporary confusion noted after bilateral prefrontal leucotomy. None of these statements are true of patients who had a pre-existing, compensated lesion of one hemisphere. In such cases a confusional state may be produced. It cannot be claimed that these observations have fulfilled such expectations as might have been entertained of arriving at some understanding of the function of the corpus callosum in the normal individual. It may be suggested that if reaction time experiments are performed on any future cases both before and after operation, disturbance of the reaction time for bilateral movements is likely to be found.

MENTAL IMAGERY.

During the past year an investigation of the physiological concomitants to mental imagery has been undertaken, checking the old respiratory observations of Golla and Antonovitch by electro-encephalography.

Golla, Hutton and Walter (1943) claim that by correlating the respiratory tracings with the evidence obtained from electro-encephalography and the patient's introspective account of his imagery, it is possible to show that the habitual type of imagery can be recognized from an inspection of the electro-

encephalogram, and thus for the first time it has been possible to correlate variations of cortical potentials with mental processes.

EXPERIMENTAL NEUROSES.

The production of experimental neurosis in animals has lately, it is claimed, thrown much light on a certain type of abnormal behaviour.

Such experiments must necessarily prove of great interest to students of physiological psychology. Masserman (1943) publishes a monograph giving a detailed description and analysis of the experimental results for 212 animals for a period of over seven years. On the strength of his experiments he formulates fundamental principles of animal behaviour :

1. Behaviour is motivated by the biological needs of the organism.
2. Behaviour is contingent on, and adaptive to, the meanings of the " objective " and " social " environment as interpreted by the individual organism.
3. Behaviour relieves bodily tensions not only by direct, but also by substitutive or symbolic activity.
4. When psychobiological motivations or environmental meanings become excessively confused or conflictual, behaviour likewise becomes abnormally substitutive, symbolic and biologically inefficient, that is, neurotic or psychotic in character.

It may be doubted if the foregoing propositions represent in any degree the fruits of so much prolonged and patient work with this particular method, which at first sight appeared promising.

BIBLIOGRAPHY.

- ACHELIS (1939), *Arch. ges. Physiol.*, **242**, 644.
 AKELAITIS *et al.* (1942), *Arch. Neur. Psychiat.*, **47**, 971.
Idem (1942), *ibid.*, **48**, 914.
 BEYER (1939), *J. Pharmacol.*, **66**, 318.
 BICKFORD (1939), *Clin. Sci.*, **4**, 159.
 BÖRNSTEIN (1940), *Yale J. Biol. Med.*, **13**, 133.
 BRICKNER (1939), *Arch. Neur. Psychiat.*, **41**, 580.
Idem (1940), *J. Neuro-physiol.*, **3**, 100.
 BROOKHART and DEY (1941), *Am. J. Physiol.*, **133**, 551.
 BROOKS (1940), *Research Publ. Nerv. and Ment. Dis.*, **20**, 525.
Idem and LAMBERT (1939), *Am. J. Physiol.*, **128**, 57.
 CARL and TURNER (1939), *J. Psychol.*, **8**, 165.
 DAVIDOFF and REIFENSTEIN (1939), *N.Y. State J. Med.*, **39**, 42.
Idem (1939), *Am. J. Psychol.*, **52**, 56.
 DEMPSEY and RIOCH (1939), *Am. J. Physiol.*, **126**, 758.
Idem (1939), *J. Neuro-physiol.*, **2**, 9.
 FREEMAN and WATTS (1942), *Psychosurgery*. Baltimore.
 GELLHORN (1942), *Autonomic Regulations*. New York.
Idem (1941), *Am. J. Psychiat.*, **97**, 1204.
Idem and MINATOYA (1943), *J. Neurophysiol.*, **6**, 161.
 GOLLA, BLACKBURN and GRAHAM (1940), *J. Ment. Sci.*, **86**, 48.
 GOLLA, HUTTON and WALTER (1943), *ibid.*, **89**, 216.
 GOODELL, HARDY and WOLFF (1940), *J. Clin. Invest.*, **19**, 649.
 GRINKER (1939), *Psychosomat. Med.*, **1**, 19.
 HAMBOURGER (1940), *J.A.M.A.*, **114**, 2015.
 HARRISON (1940), *J. Neurophysiol.*, **3**, 156.
 HERB (1939), *J. Gen. Psychol.*, **21**, 457.
Idem and PENFIELD (1940), *Arch. Neur. Psychiat.*, **44**, 422.
 HOLCK, THORNTON and SMITH (1939), *J. Ab. Soc. Psych.*, **34**, 96.
 JOHNSON and SHORT (1939), *J. Lab. and Clin. Med.*, **24**, 590.
 KLÜVER and BRAY (1939), *Arch. Neur. Psychiat.*, **42**, 979.

- KRAVKOV (1939), *Acta Ophthal.*, **17**, 89.
LEWIS (1938), *Brit. Med. J.*, **1**, 321.
MANN and QUASTEL (1940), *Biochem. J.*, **34**, 414.
MASSERMAN (1943), *Arch. Neur. Psychiat.*, **49**, 43.
MEAD (1939), *J. Gen. Psychol.*, **21**, 3.
MITTELMAN and WOLFF (1939), *Psychosomat. Med.*, **1**, 271.
NYGARD (1939), *J. Exper. Psychol.*, **24**, 1.
PAPANICOLAU and SEWARD (1939), *Psychol. Bull.*, **36**, 535.
ROBINSON (1939), *J. Missouri State Med. Assoc.*, **34**, 374.
RYLANDER (1939), *Acta Psychiat., Neurol. Suppl.*, **20**.
Idem (1941), *Acta Scandinav.*, **85**, 213.
RICHTER (1942), *Ann. Rev. Physiol.*, **4**, 561.
SCHNEDORF and IVY (1939), *Am. J. Physiol.*, **125**, 491.
SCHUMACHER (1940), *Science*, **92**, 110.
SEROTA (1939), *J. Neurophysiol.*, **2**, 42.
SHOCK (1941), *Am. J. Psychiat.*, **97**, 1374.
SPIEGEL, MILLER and OPPENHEIMER (1940), *J. Neurophysiol.*, **3**, 538.
STOOKEY, SCARFF and TEITELBAUM (1941), *Arch. Surg.*, **113**, 161.
VAN WAGENER and HERRING (1940), *Arch. Neur. Psychiat.*, **44**, 740.
WHITE (1940), *Assoc. Research Nerv. and Ment. Dis. Proc.*, **20**, 844.
WOLFF (1939), *Proc. Interstate Post-Grad. Assembly, Chicago*.