50 days of age, when they are still highly susceptible. These animals continue to have seizures after that, if they are tested.

Mice of the second strain, when tested daily from 15 to 50 days of age, also have a high level of incidence of seizures, but the seizures are of the clonic type only. In this case, too, the mice do not die, because this type of seizure is rarely fatal (*11, 12*). In both these strains the latent periods of the seizures (i.e., the time elapsing between the start of the stimulus and the start of the seizure) are quite short (1–10 sec; av 5.3 ± 1.3 sec). In the original stocks the average latent period was 35 ± 2 sec. Thus testing is greatly expedited in these strains. The reduction in the latent period is probably the result of the increased susceptibility to seizures (*14, 15*).

Mice of the third strain, when tested from 15 to 50 days of age, have seizures at the 90–100% level between 17 and 24 days of age, but have 0–5% seizures between 30 and 50 days of age. These animals are valuable particularly in maturation studies, because the same animal is susceptible at one age and nonsusceptible only a few days later.

Mice of the fourth strain—by far the most difficult to produce—when tested similarly, have a very low incidence (5–10%) of seizures and have a large number of individuals that have no seizures at all. In the 22 offspring of one pair of these mice, for instance, 16 of the animals had no seizures. These nonsusceptible mice are valuable for comparison with the highly susceptible mice and for use in studies in which seizures are undesirable.

Details of the selection procedures and development of these strains will be presented elsewhere.

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A Metabolic Cage

THE Krebs tricarboxylic acid cycle is frequently referred to as a "furnace" in which intermediates are oxidized to CO_2 and H_2O. The initiated, who know enough not to take such statements literally, automatically include the subsequent electron transfer reactions, in which the ultimate acceptor is oxygen, as part of the system. Many students, however, have difficulty in understanding the relations of the two sets of reactions. As a means of demonstrating these relationships more clearly, the author has constructed a model (Fig. 1).

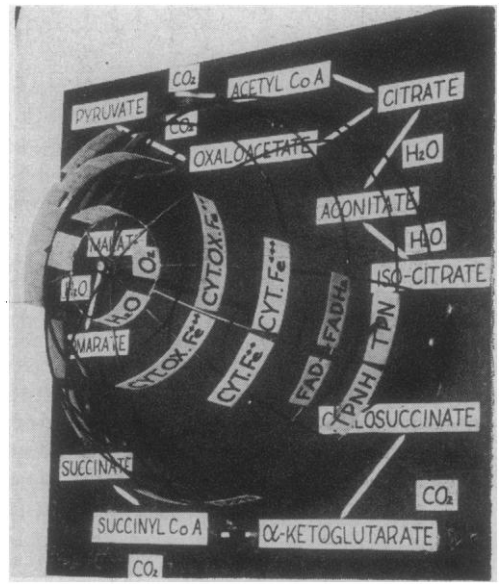

FIG. 1.

The reactions of the tricarboxylic acid cycle are represented as proceeding in a clockwise direction. Nonoxidative reactions are indicated by white arrows pasted flat to the base. Oxidation reactions in the cycle are indicated by raised red arrows which symbolize the loss of electrons. The transfer of electrons to the pyridine nucleotides is indicated by blue arrows, which are bowed toward the base. The series of electron transfers from the pyridine nucleotides, to the flavins, cytochromes, and finally to oxygen, is represented by the ascending groups of cards, each set with its red and blue arrows to indicate the electron transfers. One such group, starting with the isocitrate-oxalsuccinate: TPN-TPNH couple, faces the camera. Another series, beginning with the malate-oxalacetate: DPN-DPNH reaction, is shown from the rear. These two series illustrate the relationship adequately; hence the others have not been completed on the model. Blank cards are placed over the succinate-fumarate arrow as an admission that we do not as yet know all the answers.

To avoid confusion, many reactions and other details have been omitted. Thus, the transamination reactions, linkage with glycolysis, fatty acids, etc., could easily be added if desired. Likwise metals, cocarboxylase, phosphate, adenylic nucleotides, etc., are not depicted. Once a student grasps the central ideas, he has little difficulty adding these details to the conceptual superstructure. The cage does bring out the

sources of CO_2 and H_2O and the role of oxygen in respiratory reactions.

This particular model was constructed from a conic projection obtained from war surplus. Other cages could readily be constructed from the wire baskets obtainable at nurseries.

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[1] The author wishes to acknowledge the aid of John Kemp in the construction of the model.

Sensitivity of Male dba Mice to the Toxicity of Chloroform as a Laboratory Hazard

FIFTY mice were sacrificed in this laboratory in a closed vessel containing some gauze soaked in chloroform. In the room in which the mice were killed 55 male dba mice were housed; within 6 days all but 5 of these mice were dead or dying. Autopsy disclosed large, pale, swollen kidneys, which were seen microscopically to have gross tubular necrosis. A considerable number of mice of strains A, C, C3H, and CAF1 hybrid, of both sexes, and 55 female dba mice remained quite healthy, although kept on the same rack and under the same conditions as the male dba mice.

The renal changes observed were similar to those reported by Eschenbrenner and Miller (*1*), who also noted that in strain A mice the males were more susceptible than the females to chloroform poisoning.

To confirm that the death of the male dba mice was indeed due to chloroform poisoning, 6 dba mice of each sex were placed near a beaker containing 5 ml of chloroform for 30 min. After 8 days 3 of 6 males were dead, and all had lesions resembling those seen in the original group. The female mice showed slight fatty degeneration of the liver but were otherwise well.

It would appear, therefore, that the male dba mouse is sensitive to the toxic effects of chloroform to such a degree as to constitute an unusual laboratory hazard.

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Book Reviews

Pharmacology in Clinical Practice. Harry Beckman. Philadelphia–London: Saunders, 1952. 839 pp. Illus. $12.50.

This book supersedes the author's *Treatment in General Practice*—a text which has seen six editions over a period of 22 years, the last edition appearing in 1948. Like the older one, the new book is oriented not about various classifications of drugs, but about diseases and other clinical situations that call for the use of drugs, as encountered in internal medicine and in the other medical and surgical specialties, as well as in dentistry.

In many instances, the disease or clinical situation is introduced by a concise paragraph highlighting the clinical problem. The drugs employed in treatment are then recorded, and their administration, clinical effects, absorption, excretion, and toxicity are described. Emphasis is on the practical use of these agents, rather than on their chemical and physical properties, which have been relegated to a separate section that includes some representative commercially available preparations. To conserve space, consideration of the historical development of drugs and of the relationship between chemical structure and biologic activity has been omitted, and consideration of the mechanism of drug action has been abbreviated.

Many of the more recent developments in therapy are presented, including the use of synthetic curare substitutes, newer antibiotics, isonicotinic acid hydrazides, stilbamidine (for some mycotic diseases), autonomic blocking agents, ion exchange resins and N-allyl-normorphine. The author demonstrates a good sense of proportion in his consideration of new drugs, as well as in his evaluation of the usefulness of ACTH and cortisone, and of anticoagulants. Drugs which are unproved or subject to conflicting claims are so described. Where any one of several agents may be utilized, the author frequently indicates which, in his opinion, is superior.

Although these opinions are, in the main, reasoned and sound in the light of current practice, it is unfortunate that the author has not provided better documentation and more references than are listed in the "suggested excursions into the literature." This would put in proper perspective a few of the author's comments that are open to question—for example, that "coronary disease is not a contraindication to dihydroergocornine's use" (in hypertension), that "tolerance to hexamethonium has not been demonstrated," that "many experienced men strongly oppose intravenous administration of any fluid in cases of massive hemorrhage" (from peptic ulcer), and that "atropine should always be at hand . . . but should never be injected with the prostigmin" (in the diagnostic test for myasthenia gravis). Statements such as these are few, however. The great majority of recommendations concerning therapy are in accord with sound current practice, so that this book, which is written in a clear