A FINGER-GUARD

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This protective device I have found very practical and useful in all cases of contusions received at the ends of fingers and toes, as well as after amputations of these members. Particularly will the guard be indicated at these points named where cicatricial tissue has been deposited or is being deposited during the healing process. The guard will be found useful in this class of injuries received by mechanics and workmen who are obliged to continue at their work for economic reasons while the fingers are still "very tender." All blows or contacts to the injured member, it will be noticed, are received on the metallic breast-work or conical point, and the force of any jar and impact is thus carried down and received on the body of the finger or the healthy, normal tissue, and the shock

Finger-Guard.

to the affected part is, consequently, greatly lessened and better distributed than if received directly on the primary injury.

At tinsmiths may be secured strips of tin or zinc, about 1/16 or 1/8 inch wide and approximately 4 inches in length (strips of cardboard so cut will answer nicely if the former material is not at hand). Two of these pieces or strips should be bent to fit over the end of the finger in loop or horse-shoe form, each loop being placed at right angles or crossing the other, flat side down, so that there is an anterior and a posterior, as well as two

lateral bars (four in all), extending downward nearly to the second phalangeal joint, or lower, if this point is involved in the injury. A strip of adhesive plaster about 1/2 inch wide is now bound firmly around (it may be split between the bars if member tends to swell much without impairment of anchorage) the finger covering the distal ends of bars to the same depth, or it may lap over the distal ends on the tissue of the finger; the cage or guard will then be securely held in a firm position.

A turn or two of gauze, to be first applied when strips are bent, will protect skin of finger from edges or ends of tin and prevent laceration, or the edges may be bound or rolled in adhesive plaster for the same purpose. A space of from 1/4 to 1/4 inch may be left between the end of the finger and the inside of the guard through which the wound may be dressed from time to time.

The whole contrivance may then be closed with turns of a bandage or may be left, if desired, with strips of metal exposed

ACETONURIA AND THE RELATION OF
ACETONE TO "ACID INTOXICATION" *

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The purpose of this paper is to report progress on some investigations regarding the nature of acetone and of its relative importance in the symptom-complex known as "acid intoxication." The present conception of this condition is that the toxemia is due to the acid qualities of the acetone bodies, and that the acids, namely, diacetic and beta-oxybutyric acids, although not in themselves toxic, produce a condition of toxemia by uniting with the fixed alkalies of the blood. The blood no longer being able to extract the poisonous carbon dioxide, the subject succumbs to "internal suffocation" from its accumulation in the tissues, the acids being excreted as ammonia compounds, resulting in a larger excretion of ammonia and a consequent diminution of urea.

Ewing¹ reports a study of acid poisoning by injecting intravenously eighth-normal to fourth-normal hydrochloric acid into rabbits. He found that they could stand large quantities of acids slowly administered (one receiving 1,485 gm. in eleven days), drowsiness being the chief symptom, with no dyspnea and no fatty degenerations of the liver or kidneys. He gives the following objections against the purely acid nature of diabetic coma: Munser and Strasser reported cases with acetone and diacetic acid but no oxybutyric acid; coma may improve while the urine is still acid; alkalies may not reduce the urinary ammonia; miliary tuberculosis and erysipelas have been found to reduce the carbon dioxide of the blood. Ewing concludes that some process is responsible for both coma and acids. He is of the opinion that none of the principles of the theory of acid intoxication have been proved, namely, carbon-dioxide asphyxiation, degenerations of vital organs by withdrawal of alkalies and reduced alkalinity. He concludes that more fundamental knowledge of the source, occurrence, chemistry and physiology of the acetone bodies is needed before correct interpretation can be obtained. The appearance of these bodies in such a variety of toxic and debilitating conditions demonstrates that they must play an important part in the pathology of metabolism. An attempt, therefore, to know more of their nature and to become more familiar with their properties is at least worth while.

From the experiments of Knoop and others it seems to be definitely settled that the acetone bodies are derived principally by faulty oxidation of fat and fatty acids, although they can be formed to some extent from proteids. This abnormal splitting of fats can take place during abnormal catabolism or during faulty digestive processes in the intestine. As proof of the latter contention can be offered the following data: Geelmuyden noticed acetonuria after adding olive oil and butter to a normal diet. Neuberg and Blumenthal found that butyric acid by mouth increased acetone output, while subcutaneous injection did not, ascribing this to activity of intestinal bacteria. Beer and Blum fed organic acids to diabetics and found an acetone output in each instance. Schwartz found that fatty diet increased acetone output in diabetes but not in health. Geelmuyden

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1. Ewing: Arch. Int. Med. 1908, 11, 330 and 448.