has been possible, by means of intermittent quinidine therapy, properly spaced, to maintain the normal rhythm for over five months, with coincident marked clinical improvement.

Intravenous injection of atropine sulphate (1.0 to 1.5 mgm.) in these patients, at a time when fibrillation was present and again when the normal rhythm prevailed, resulted in the usual increase in ventricular rate, but in no significant alteration in the cardiac mechanism or in the form of the electrocardiogram.

*Cases in which Restoration of the Normal Mechanism was Not Accomplished.*—Eleven courses of quinidine were administered to 8 patients. As in the group just decsribed, tachycardia was commonly the first effect observed. Ventricular premature beats, at times in the form of coupled rhythm, were more commonly seen than in those patients in whom the sinus rhythm was eventually established. Occasionally the fibrillatory waves became coarser. In two patients auricular flutter followed administration of the drug, but a larger dosage was not followed by the normal rhythm. In one of these cases auricular flutter persisted for three days and was followed, after administration of digitalis, by reversion to the fibrillatory mechanism. Paroxysms of ectopic ventricular tachycardia occurred three times. Although of short duration, they served to indicate that quinidine as a therapeutic agent was not to be administered with impunity, for ventricular tachycardia occurring in dogs poisoned by digitalis or strophanthin is not infrequently the precursor of ventricular fibrillation.

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## Studies on the acetonuria produced by diets high in fat.

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The following ratio was suggested to express the ketogenic balance of any diet:

$$100 \times \frac{1.5\ (\text{weight carbohydrate} + 25\ \text{per cent. weight protein})}{95\ \text{per cent. weight fat}}.$$

This ratio is based on the relative molecular weights of glucose and the higher fatty acids—stearic, palmitic, and oleic; in it it is

assumed that approximately half the glucose derived from protein is used up in burning the ketogenic material from the α-amino acids leucine, tyrosine and phenyl alanin occurring in the same protein; no allowance is made in the expression for the possible antiketogenic effect of the glycerol radicle present in the fats.

Diets high in fat were fed to a normal subject, and to arthritic patients undergoing the Pemberton[1] treatment. These diets were based on that suggested by Shaffer[2] which contained 10 per cent. of the total calories as protein, 10 per cent. as carbohydrate, and 80 per cent. as fat. The degree of acetonuria which corresponded with each diet was determined, and the results compared with the numerical values of this ratio.

From a study of these values which corresponded with a very mild degree of acetonuria it was concluded: one, that the phenomenon of ketogenesis could properly be regarded as a molecular reaction between ketogenic and antiketogenic compounds in the diet; two, that protein entered into the reaction only to the extent of the glucose which could be drived from the α-amino acids contained in it; three, that the glycerol radicle of fat figured as a source of antiketogenic material only to the extent to which glucose could be derived from it; and, probably, four, that the glycerol radicle probably did figure as a source of antiketogenic material to the extent to which it could yield glucose.

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## The glycogen content of the tissues of diabetic animals and the influence of adrenalin thereon.

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In a series of experiments on dogs rendered diabetic by means of phlorhizin, the glycogen content of the muscles was studied immediately after the animals were killed. The muscles of thirteen animals were analyzed at the end of two days of glucosuria.

[1] Pemberton, R., *Am. J. Med. Sci.*, 1917, cliii, 678.

[2] Shaffer, P. A., *J. Biol. Chem.*, 1921, xlvii, 449; Woodyatt, R. T., *Arch. Int. Med.*, 1921, xviii, 125.