slow systolic-diastolic waves were noted. None of these cases had received cardiac therapy of any kind.

The low-frequency (inaudible) vibrations recorded by this method may be due to actual motions of the myocardium. Absolute proof for this is yet lacking. However, the tentative suggestion may be made that as the myocardium is compromised by a diminished coronary flow, and tends to become flaccid, slow vibrations may be provoked in the muscle mass by the violent onset of contraction. Whatever the cause, these slow waves as recorded are constant enough in coronary disease to warrant the suggestion that they may indicate flaccidity and weakness of the heart muscle, and may appear before definite signs of myocardial disease develop.

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Glycogen Content of the Human Liver.*

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In recent years much attention has been given to the value of the preoperative diet and its relation to the level of liver glycogen in surgical patients. Unfortunately, little is known about the effect of diet, anesthesia and liver pathology upon the glycogen levels of the human liver. Most of our knowledge has been obtained from analysis of human livers obtained at autopsy or from livers of experimental animals. In order to obtain direct information on liver glycogen levels in surgical patients, it was decided to determine the glycogen content of liver specimens obtained by biopsy, and this program was carried out on a series of 33 patients undergoing laparatomy during the winter of 1939-40.

A small section of the biopsy specimen taken during the operation was reserved for microscopic examination and the remainder, approximately 1 g, was immediately frozen in carbon dioxide snow and ether. While still frozen the specimen was ground very fine and transferred into a weighed centrifuge tube containing potas-

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sium hydroxide, then quickly weighed, and placed in a beaker of boiling water. After solution was complete, the glycogen was precipitated and hydrolyzed according to the Good, Kramer, and Somogyi modification[1] of Pflüger's method for liver glycogen. The total reducing carbohydrate was determined on aliquots in duplicate according to Shaffer and Somogyi.[2]

In Table I the glycogen values from 26 cases have been grouped according to the extent of liver damage, as shown by microscopic sections reported by the Department of Pathology, the anesthetic used, and whether or not supplementary glucose was fed. The condition of the liver was reported as normal or as showing advanced damage. In 7 additional cases, most of whom showed moderate damage, no grouping could be made because of a multiplicity of varying conditions. The values in these cases ranged between 1.22-4.65%. The average for Group A is 3.15% but if the highest value of 6.31% in this group is omitted, the average of Group A becomes 2.80%. All the individual values in both Groups B and C are above this figure, and the averages of Groups A and D are almost the same.

Conclusions. The glycogen content of biopsy samples of normal livers was highest in patients that were fed glucose prior to operation. When extensive liver damage was present, liver glycogen was low despite glucose feeding.

TABLE I.
Distribution of Values for Hepatic Glycogen.

	A	B	C	D
No. of cases	10	7	5	4
Range %	1.10-6.31 (—3.85)*	3.19-7.56	3.91-5.75	2.70-3.14
Avg %	3.15 (2.80)*	5.03	4.73	2.86

A—Normal livers, spinal anesthesia, no glucose feedings.
B—Normal livers, spinal anesthesia and oral administration of 200 g of glucose during the 12 hours immediately before the operation.
C—Normal livers, nitrous oxide and ether vapor anesthesia and 200 g of glucose similarly fed.
D—Advanced hepatic damage, spinal anesthesia in 3 and nitrous oxide and ether vapor anesthesia in 1, and 200 g of glucose fed.
*See text.

1 Good, C. A., Kramer, H., and Somogyi, M., *J. Biol. Chem.*, 1933, **100**, 485.
2 Shaffer, P. A., and Somogyi, M., *J. Biol. Chem.*, 1933, **100**, 695.