Chemie. W. Foerst, Ed. Vol. 14: 306. Urban & Schwarzenberg. München, West Germany.
100. OSTER, R. H., C. J. CARR & J. C. KRANTZ. 1947. Anesthesin. XXVII. Narcosis with vinyl chloride. Anesthesiology 8: 359.
101. OSTERMAYER, H. 1967. Vinylchlorid. *In* Ullmanns Encyklopädie der technischen Chemie. W. Foerst, Ed. Vol. 18: 87. Urban & Schwarzenberg. München, West Germany.
102. OTTO, P., P. H. KRAUSE, H. G. JESTER, F. W. SCHMIDT & K. H. GLATZEL. 1972. Idiopathische portale Hypertension. Med. Klin. (Munich) 67: 447.
103. PARMEGGIANI, L. & C. SASSI. 1955. Rischi e patologia professionale nella produzione e nella lavorazione di alcune materie plastiche. Med. Lavoro 46: 14.
104. PATTY, F. A., W. P. YANT & C. P. WAITE. 1930. Acute response of guinea pigs to vapors of some new commercial organic compounds. V. Vinyl chloride. Public Health Rep. 45: 1963.
105. PATTY, F. A., Ed. 1958. Industrial Hygiene and Toxicology. Vol. I. General Principles. 2nd edit. Interscience Publishers. New York, N.Y.
106. PATTY, F. A., Ed. 1963. Industrial Hygiene and Toxicology. Vol. II. Toxicology. D. W. Fassett & D. D. Irish, Eds. 2nd edit. Interscience Publishers, New York, N.Y.
107. PEOPLES, A. S. & C. D. LEAKE. 1933. The anesthetic action of vinyl chloride. J. Pharmacol. Exptl. Ther. 48: 284.
108. POLISH, E., J. CHRISTIE, A. COHEN & B. SULLIVAN. 1962. Idiopathic presinusoidal portal hypertension (Banti's syndrome). Ann. Internal Med. 56: 624.
109. POPPER, H., F. PARONETTO, F. SCHAFFNER & V. PEREZ. 1961. Studies on hepatic fibrosis. Lab. Invest. 10: 265.
110. Popper, H. 1968. Pathology of portal hypertension. *In* The Therapy of Portal Hypertension. N. G. Markoff, Ed. Georg Thieme Verlag. Stuttgart, West Germany.
111. POPPER, H. & F. HUTTERER. 1970. Hepatic fibrogenesis and disturbance of hepatic circulation. Ann. N.Y. Acad. Sci. 170: 88.
112. POPPER, H. & S. UDENFRIEND. 1970. Hepatic fibrosis. Correlation of biochemical and morphologic investigations. Am. J. Med. 49: 707.
113. PUSHIN, G. A. 1965. O porashenii pečeni i želčnyikh putei u rabočikh' zanjatyikh w proizwodstwe nekatoryikh widow plastmass. Sov. Med. 28: 132.
114. PVC producers see good business ahead. 1969. Chem. Eng. News August 4: 18.
115. QUOOSS, H. 1959. Gesundheitsgefahren in der Kunststoffindustrie. Johann Ambrosius Barth Verlag. Leipzig, East Germany.
116. RAMALINGASWAMI, V., K. L. WIG & S. K. SAMA. 1962. Cirrhosis of the liver in Northern India—a clinicopathological study. Arch. Internal Med. 110: 350.
117. RAMALINGASWAMI, V. & N. C. NAYAK. 1970. Liver disease in India. *In* Progress in Liver Disease. H. Popper & F. Schaffner, Eds. Vol. III: 222. Grune & Stratton. New York, N.Y.
118. RAPPAPORT, A. M., M. KNOBLAUCH, R. G. BLACK & S. OHIRA. 1970. Hepatic microcirculatory changes leading to portal hypertension. Ann. N.Y. Acad. Sci. 170: 48.
119. RAVENNA, P. 1940. Banti syndrome (fibrocongestive splenomegaly). Definition, classification and pathogenesis. Arch. Internal Med. 66: 879.
120. REMMER, H. 1970. The role of the liver in drug metabolism. Am. J. Med. 49: 617.
121. REYNOLDS, T. B. & A. G. REDEKER. 1965. Hepatic hemodynamics and portal hypertension. *In* Progress in Liver Disease. H. Popper & F. Schaffner, Eds. Vol. II: 457. Grune & Stratton. New York, N.Y.
122. ROTH, F. 1957. Arsen, Leber, Tumoren (Hämangioendotheliom). Z. Krebsforsch. 61: 468.
123. ROUSSELOT, L. M. 1940. The late phase of congestive splenomegaly (Banti's syndrome) with hematemesis but without cirrhosis of the liver. Further observations on the etiology of Banti's syndrome and the effect on prognosis of certain variations in the portal venous pattern. Surgery 8: 34.
124. SAMA, S. K., S. BHARGAVA, N. GOPI NATH, J. R. TALWAR, N. C. NAYAK, B. N. TANDON & K. L. WIG. 1971. Noncirrhotic portal fibrosis. Am. J. Med. 51: 160.
125. SARAGOCA, A., M. R. TAVARES, F. B. BARROS & J. DA SILVA HORTA. 1972. Some clinical and laboratory findings in patients injected with thorium dioxide. Study of 155 cases. Am. J. Gastroenterol. 57: 301.
126. SCHAFFNER, F. & H. POPPER. 1963. Capillarization of hepatic sinusoids in man. Gastroenterology 44: 239.

127. SCHAUMANN, O. 1934. Ueber die Herzwirkung einiger Inhalationsnarkotica. Medizin & Chemie. Abhandlungen aus den Medizinisch-chemischen Forschungsstätten der I.G. Farbenindustrie A.G. Vol. II. Bayer-Meister Lucius. Leverkusen, West Germany.
128. SCHAUMANN, O. 1938. *In* Toxikologie und Hygiene der technischen Lösungsmittel. K. B. Lehmann & F. Flury, Eds. p. 130 (Monochloräthylen). Julius Springer. Berlin, Germany.
129. SCHMID, M. 1968. Zur Leberhistologie der verschiedenen Formen des portalen Hochdruckes. *In* The Therapy of Portal Hypertension. N. G. Markoff, Ed. Georg Thieme Verlag. Stuttgart, West Germany.
130. SCHMIDT, F. W. 1969. Geringer Aktivitätsanstieg der Serum-Transaminasen. Deut. Med. Wochschr. 94: 2250.
131. SCHNACK, H., L. STOCKINGER & F. WEWALKA. 1967. Adventitious connective tissue cells in the space of Disse and their relation to fibre formation. Rev. Intern. Hépatol. 17: 855.
132. SIMONDS, H. R., A. I. WEITH & M. H. BIGELOW. 1949. Handbook of Plastics. 2nd edit. D. van Nostrand Co., Inc. Princeton, N.J.
133. SLATER, T. F. 1972. Free radical mechanisms in tissue injury. Pion Ltd. London, England.
134. SMORON, G. L. & H. A. BATTIFORA. 1972. Thorotrast-Induced hepatoma. Cancer 30: 1763.
[illegible] inhalation. Am. Ind. Hyg. Assoc. J. 17: 129.
136. SOLOWAY, R. D., A. H. BAGGENSTOSS, L. J. SCHOENFIELD *et al.* 1971. Observer error and sampling variability tested in evaluation of hepatitis and cirrhosis by liver biopsy. Am. J. Digest. Diseases 16: 1082.
137. STEIN, G. [illegible] Nuklearmed. 11: 60.
138. STENGER, R. J. 1965. Fibrogenesis along the hepatic sinusoids in carbon-tetrachloride-induced cirrhosis. An electron microscopic study. Expti. Mol. Pathol. 4: 357.
139. STUTZ, F. H., D. C. TORMEY & J. BLOM. 1973. Hemangiosarcoma and pathologic rupture of the spleen. Cancer 31: 1213.
140. SUCIU, I., I. DREJMAN & M. VALASKAI. 1963. Contributii la studiul imbolnavirilor produse de clorura de vinil. Med. Interna 15: 967.
141. SUCIU, I., I. DREJMAN & M. VALASKAI. 1967. Etude des maladies dues au chlorure de vinyle. Med. Lavoro 58: 261; Abstr. Hyg. 43: 430 (1968).
142. SUMMERSKILL, W. H. J. 1974. Chronic active liver disease reexamined: prognosis hopeful. Gastroenterology 66: 450.
143. SUSSMAN, E. B., I. NYDICK & G. [illegible] mangioendothelial sarcoma of the liver and hemochromatosis. [illegible] 39.
144. SYMANSKI, H. Ed. 1963. Handbu[illegible] Ed. Vol. IV/2: Arbeitshygiene. [illegible] rbeitsmedizin. E. W. Baader, [illegible] renberg. München, West Germany.
145. TANDON, B. N., R. LAKSHMINARAY[illegible] 1970. Ultrastructure of the liver [illegible] , N. C. NAYAK & S. K. SAMA. ortal fibrosis with portal hypertension. Gut 11: 905.
146. TISDALE, W. A., G. KLATSKIN & W. [illegible] bleeding esophageal varices. Th[illegible] hepatic and extrahepatic obstructi[illegible] 209. [illegible] 59. Portal hypertension and the absence of both intrahepatic and extrahepatic obstruction. New Engl. J. Med. 261: 209.
147. TORKELSON, T. R., F. OYEN & V. K. [illegible] as determined by repeated exposur[illegible] soc. J. 22: 354; Bull. Hyg. 37: 357. [illegible] toxicity of vinyl chloride [illegible] imals. Am. Ind. Hyg. As-
148. TRIBUKH, S. L., N. P. TIKHOMIROVA, S. [illegible] truda i meropriyatiya po ikh ozd[illegible] khlorvinilovykh plastičeskikh mass. [illegible] . KOZLOV. 1949. Usloviya izvodstve i izpol'zovanii 18.
149. TRUFFERT, L. 1959. Aspects toxicologi[illegible] Arch. Maladies Profess. 20: 43. des matières plastiques.
150. U.S. DEPARTMENT OF HEALTH, EDUCAT[illegible] VICE: CENTER FOR DISEASE CONTROL. [illegible] giosarcoma of the liver among polyvinyl chloride workers—Kentucky. Morbidity Mortality Weekly Rep. 23(6): 49. [illegible] E—PUBLIC HEALTH SER-

Observer Error and Sampling Variability Tested in Evaluation of Hepatitis and Cirrhosis by Liver Biopsy

Roger D. Soloway, MD, Archie H. Baggenstoss, MD, Leslie J. Schoenfield, MD and W. H. J. Summerskill, DM

In 50 patients with chronic active liver disease, observer and sampling error in histologically evaluating hepatitis and cirrhosis after blind-needle biopsy of the liver was assessed from coded tissue. This was done by repeated readings of the same specimens by the same pathologist, by sequential biopsies from the same patients with cirrhosis, and by multiple simultaneous biopsies from adjacent areas of the liver. Observer error was small. The consistency of grading the individual histologic characteristics of hepatitis was 90%, and the reproducibility of the degree of either hepatitis or cirrhosis was 94%. Sampling error was also trivial in hepatitis, indicating that a single needle biopsy accurately reflects the type and degree of inflammation and necrosis in adjacent areas of the liver. By contrast, sampling error was of considerable magnitude when the presence of cirrhosis in patients known to have the lesion was sought, since confirmation by simultaneous or sequential biopsies was made in only 33% of the cases.

Accuracy of aspiration needle biopsy in evaluating or diagnosing hepatitis (1) and cirrhosis (2) has hitherto been assessed by multiple postmortem biopsies. Under these circumstances, accuracy in each condition can approach 100%, but such biopsies were not taken blindly. Since determination of histologic appearance, which is accessible only by blind biopsy of the liver, is of practical value in diagnosing and managing several chronic liver diseases, it is essential to define, for a single needle biopsy, the reliability of a single interpreter (observer error) and the representative nature of the tissue acquired (sampling variability). Results of blind evaluation of needle biopsies by three pathologists indicate that interobserver errors can be as much as 48% (3).

In particular, histologic changes in the biopsy tissue from the liver may be the most relevant and sensitive method of judging response to treatment of chronic active hepatitis. Corticosteroids, 6-mercaptopurine, or azathioprine may cause severe side effects, and adjustment of medication is influenced by histologic evidence of activity or suppression of the disease (4, 5). We have, therefore, assessed blind biopsies, taken during life, for observer error and sampling variability to provide a basis for diagnosing and managing patients with chronic active liver disease and other conditions.

MATERIALS AND METHODS

Transthoracic or subcostal liver biopsies, taken with a Vim-Silverman needle from 50 patients being

From the Gastroenterology Unit and the Department of Pathologic Anatomy, Mayo Clinic and Mayo Foundation, Rochester, Minn.

Supported in part by Research Grant AM-6908 and Training Grant T1-AM-5259 from the National Institutes of Health, Public Health Service.

Address for reprint requests: Section of Publications, Mayo Clinic, Rochester, Minn 55901.

treated for chronic active liver disease, were studied. The criteria for the diagnosis (5) included disease of at least 10 weeks' duration, with a persistent tenfold elevation of SGOT, or a fivefold elevation of SGOT coupled with a twofold rise in γ-globulin. In addition, all patients had severe hepatitis with moderate or severe piecemeal necrosis when observed on initial biopsy.

Biopsies were prepared using hematoxylin and eosin and van Gieson stains. Each biopsy was randomly assigned a different code number and was evaluated by the same pathologist without knowledge of clinical or biochemical data. Biopsies were examined for seven general features (each representing the summation of four to six specific characteristics), and for the degrees of hepatitis and cirrhosis. Each characteristic and feature (Table 1), together with the degrees of hepatitis and cirrhosis, was graded on a scale from 0 (normal) to 3 (most severe). Cirrhosis was diagnosed by the presence of regenerative nodules. Grade 1 indicated early or doubtful cases in which only segments of nodules were seen. Grades 2 and 3 indicated that cirrhosis was definite and that the normal lobular pattern had been largely or completely replaced by regenerative nodules.

To test observer error, the same 12 slides were submitted twice for evaluation of each histologic characteristic and feature, separated by periods of months to years, with different code numbers. To determine consistency in evaluating the degree of hepatitis and cirrhosis, 34 slides were submitted for a second reading in the same way.

Sampling error regarding the presence of cirrhosis (assuming the lesion to be irreversible) was tested in 19 patients from whom three to five biopsies were taken at intervals of 6 or 12 months after cirrhosis was demonstrated. Next, to test the representative nature of the diagnosis of hepatitis or cirrhosis from a single biopsy, two or three biopsies from adjacent areas of the liver were obtained simultaneously through the same intercostal site in

Table 1. Reproducibility of Histologic Grading (Same Biopsy)

Features*	No. of comparisons	Disagreement		Full agreement
		Marked†	Minimal‡	
Parenchymal necrosis (acidophilic, lytic, focal, diffuse, piecemeal)	60	2	20	38
Parenchymal infiltration (polymorphonuclear, acidophilic, lymphocytic, plasma cell, fibroblastic, Kupffer cell)	72	3	24	45
Bile stasis (liver cells, Kupffer cells, canalicular, central, peripheral)	60	0	7	53
Parenchymal miscellaneous (vacuolization of nuclei, multinucleated giant cells, collagen, fatty change, fatty change with cysts, pseudoductular proliferation, mitoses)	84	2	13	69
Portal tract inflammation (polymorphonuclear, acidophilic, lymphocytic, plasma cell, fibroblastic)	60	3	18	39
Portal tract ductular change (typical duct proliferation, atypical duct proliferation, ductular cell reaction, duct degeneration, reduced number of ducts)	72	7	16	49
Portal tract miscellaneous (size of portal tract, collagen, septal formation, granulomas)	48	0	13	35
Totals§	456	17	111	328

* All graded 0 to 3; characteristics are in parentheses.
† 2-grade difference between the 2 readings.
‡ 1-grade difference between grades 0 and 1, 1 and 2, or 2 and 3.
§ Whole table: $\chi^2_6 = 12.74$ ($P < 0.05$) = table is not homogeneous.

13 patients. The needle was pointed in three different directions during the procedure, and each biopsy was separated from the others by an angle of at least 30°. Eight patients had three, and 5 patients had two such simultaneous biopsies.

RESULTS

Observer error. The reproducibility of grading the same biopsy was established from 12 biopsies, with 38 specific histologic characteristics grouped into seven general features, all of which were evaluated twice (Table 1). There was full agreement in 328 of 456 comparisons (72%), a difference in only 1 grade in 25%, and a greater discrepancy of 2 grades in only 3%. There were no 3-grade differences. The group of comparisons which demonstrated 1-grade differences (25%) were composed of 44 (9%) in which the difference was between grades 0 and 1, and 67 (16%), in which the difference was between grades 1 and 2 or grades 2 and 3. Among the general features, errors were found more frequently in evaluating portal or parenchymal inflammation and bile ductular changes, whereas excellent accuracy was achieved in estimating bile stasis. Because of these deviations, the accuracy of diagnosis among general features (Table 1) was not homogeneous ($P<0.05$). Among 12 evaluations of diffuse necrosis, there was only one 1-grade difference (between grades 2 and 3), and in 12 evaluations of piecemeal necrosis, there were three 1-grade differences (between grades 2 and 3) of no practical significance.

To assess reproducibility of the degree of hepatitis or cirrhosis, 34 slides were evaluated twice. Overall severity of hepatitis was exactly reproducible in 30 of 34 slides (88%). Two specimens were interpreted differently between grades 2 and 3, and two, between grades 1 and 2. The grade of cirrhosis was exactly reproduced in 26 of 34 biopsies (76%). Five biopsies differed by only 1 grade; in two instances, the difference was between grades 0 and 1, and in the others, there was one 2-grade difference.

Sampling variability. Evaluation of multiple simultaneous biopsies (Table 2) showed that the presence and grade of hepatitis in a single biopsy accurately reflected hepatitis in adjacent areas. The only discrepancies noted were 1-grade differences in 4 of 21 pairs: 2 between grades 2 and 3, which had no diagnostic significance; and 2 between grades 1 and 2. By contrast, there were many discrepancies in the presence and grade of cirrhosis. In five sets of three and in three sets of two biopsies, in which grade 1 or 2 cirrhosis was seen in at least one specimen of the set, there was only one instance of uniform grading. Only three of seven sets showed cirrhosis in all biopsies (Table 2).

Further, to judge the consistency of the diagnosis of cirrhosis, three to five sequential biopsies from 19 patients were exam-

Table 2. Evaluation of Multiple Simultaneous Biopsies from Same Patient

	Biopsies					
	Hepatitis*			Cirrhosis*		
Patient	A	B	C	A	B	C
1	3	3	3	0	0	1
2	3	3	3	2	2	1
3	3	3	2	1	2	0
4	2	2	2	0	0	0
5	2	2	2	0	0	1
6	1	1	2	1	1	1
7	1	1	1	0	0	0
8	1	1	1	0	0	0
9	3	2	—	1	2	—
10	2	2	—	0	0	—
11	2	1	—	0	1	—
12	1	1	—	0	1	—
13	1	1	—	0	0	—

* Graded 0 to 3 (0 = normal).

ined during th
was diagnosed.
diagnosis consi
cirrhosis to be
ance of cirrhos
ently inconsiste
pling error. F
biopsy not de
preceded and fo
cirrhosis was p
consistent. Even
cirrhosis was co
of cirrhosis varie

D

From our ex
of an individua
hepatitis was si
interpretation
ther, there wa
the many featu
prising the his
in 72% of case
difference in in
ly contribute
tween grades 0
looked, as wit
change in chr
and, between
necrosis, imper
chronic persist
titis (6) could
Sampling erro
encompasses o
even less im
simultaneous
of the liver sh
tency and we
Others have re
(1, 7) from au
sional discrepa
magnitude to
mented earlie
evaluate inclu
severity of por

ined during the 1–3 years after cirrhosis was diagnosed. In only 8 patients was the diagnosis consistent (Table 3). Assuming cirrhosis to be irreversible, the disappearance of cirrhosis in 6 patients was apparently inconsistent and represented a sampling error. Further, in five instances, a biopsy not demonstrating cirrhosis, but preceded and followed by biopsies in which cirrhosis was present, was more clearly inconsistent. Even in the minority in which cirrhosis was consistently present, the grade of cirrhosis varied.

Table 3. Diagnosis of Cirrhosis* on Sequential Biopsies from Same Patient with Cirrhosis

	Months after original diagnosis				
Patient	0	6	12	24	36
CONSISTENT					
1	1	2	2		
2	—	1	1	—	3
3	3	3	—	2	1
4	2	—	3	3	3
5	2	2	3		
6	1	2	2	2	
7	1	1	3		
8	3	2	1	2	
APPARENTLY INCONSISTENT					
9	2	1	0		
10	1	1	0	0	0
11	2	—	0	0	
12	2	1	—	2	0
13	1	0	0		
14	2	0	0		
INCONSISTENT					
15	1	0	2	1	1
16	2	0	1		
17	1	0	1	0	0
18	1	0	1		
19	1	0	3	2	

* Graded 0 to 3 (0 = normal).

DISCUSSION

From our experience, the observer error of an individual in grading the degree of hepatitis was small, and the consistency of interpretation approximated 94%. Further, there was full agreement regarding the many features and characteristics comprising the histologic diagnosis of hepatitis in 72% of cases. However, even a 1-grade difference in interpretation may occasionally contribute to clinical errors. Thus, between grades 0 and 1, disease may be overlooked, as with the portal inflammatory change in chronic persistent hepatitis (6); and, between grades 1 and 2 of piecemeal necrosis, imperfect differentiation between chronic persistent and chronic active hepatitis (6) could lead to mistaken therapy. Sampling error by blind biopsy, which also encompasses observer error, is probably of even less importance in hepatitis, since simultaneous biopsies from adjacent areas of the liver showed a high degree of consistency and were therefore representative. Others have reported a similar conclusion (1, 7) from autopsy specimens. The occasional discrepancies found were of similar magnitude to the observer error documented earlier. The most difficult areas to evaluate included the type but not the severity of portal and parenchymal inflammation, because plasma cells and lymphocytes are difficult to distinguish on routinely stained specimens; it was also difficult to assess lesions of the bile ductules, a category previously reported to present difficulties in diagnosis (8).

By contrast, the diagnosis of cirrhosis was unreliable. Observer error (24%) was slightly greater than with hepatitis (12%), but not significantly different. But the magnitude of sampling variability, tested in two ways, was such that failure to demonstrate cirrhosis in patients with chronic active liver disease offers no assurance that the lesion is not present. This finding

differs from the results of series done from postmortem material (2), but accords with a report in which blind biopsy failed to demonstrate cirrhosis in 51% of 254 patients in whom the diagnosis was made at laparoscopy (9). In chronic active liver disease, cirrhosis is usually of the postnecrotic (macronodular) variety (10), and bioptic confirmation of this type of cirrhosis is acknowledged to be more difficult than is portal (micronodular) cirrhosis.

REFERENCES

1. Waldstein SS, Szanto PB: Accuracy of sampling by needle biopsy in diffuse liver disease. Arch Pathol 50:326–328, 1950
2. Braunstein H: Needle biopsy of the liver in cirrhosis: diagnostic efficiency as determined by postmortem sampling. Arch Pathol 62:87–95, 1956
3. Garceau AJ and the Boston Inter-Hospital Liver Group: The natural history of cirrhosis. II. The influence of alcohol and prior hepatitis on pathology and prognosis. N Eng J Med 271:1173–1179, 1964
4. Page AR, Good RA, Pollara B: Long-term results of therapy in patients with chronic liver disease associated with hypergammaglobulinemia. Amer J Med 47:765–774, 1969
5. Soloway RD, Baggenstoss AH, Elveback LR, et al: The treatment of chronic active liver disease (CALD). Gastroenterology 60:167, 1971, abstr
6. De Groote J, Desmet VJ, Gedigk P, et al: A classification of chronic hepatitis. Lancet 2:626–628, 1968
7. Scheuer PJ: Liver biopsy in the diagnosis of cirrhosis. Gut 11:275–278, 1970
8. Baggenstoss AH: Morphologic and etiologic diagnoses from hepatic biopsies without clinical data. Medicine (Baltimore) 45:435–443, 1966
9. Vido I, Wildhirt E: Korrelation des laparoskopischen und histologischen Befundes bei chronischer Hepatitis und Leberzirrhose. Deutsch Med Wschr 94:1633–1637, 1969
10. Baggenstoss AH, Stauffer MH: Posthepatitic and alcoholic cirrhosis: clinicopathologic study of 43 cases of each. Gastroenterology 22:157–180, 1952

Effect of Vagotor
Response Curves

S. J. Konturek, MD.

**Pancreatic dose-res.
*duodenal ulcer befo
was determined by
progressively increas
and pyloroplasty die
curves for the volur
of vagal nerves is c
exogenous secretin.**

The role of vagal inn
secretion in man has b
number of authors, and
vagotomy and pyloropl
crease of gastric secretor
tle attention has, howeve.
effect of this procedure or
tion (2, 3).

This report was desig
influence of vagotomy ar
the entire range of panc.
response to pure natural s

METHOD

Six men (28–41 years of age.
all volunteers who were hospi
ulcer disease, were selected for
The diagnosis, established .
tion of an ulcer crater, was co
at the time of surgery.

Prior to surgery, pancreatic
performed in the early morni
fast. A double-lumen Dreiling

From the Institute of Phy
Surgical Clinic, Medical Acade
Address for reprint requests
turek, MD, Institute of Physio
my, Krakow, ul Grzegorzecka 16