MEDICAL GRAND ROUNDS

PARKLAND MEMORIAL HOSPITAL

February 6, 1964

ASCITES

Part of extracellular fluid volume. Same tonicity as other body fluids.

Electrolytes (small differences due to Donnan effect) glucose, urea, other components of non-protein nitrogen have approximately same concentrations in ascitic fluid and in serum.

Color and appearance

1. Most fluids are clear and slightly yellow.
2. Bloody taps - try to exclude trauma - with trauma there is frequently progressive clearing of fluid as it drains. Uniform distribution of blood throughout specimen. Usually indicates other process - neoplasm most common.
3. Milky effusions - see below.
4. Watery, whitish fluids with slight opalescence, virtually pathognomonic for nephrotic syndrome.
5. Foul odor - when present is usually associated with infection or neoplasm.

Coagulability - function of protein content

Cytology

Examine as soon as possible, for cells degenerate with standing.

Add aqueous sodium citrate, 20 per cent solution; 1 ml to each 10 ml of serous fluid for most satisfactory cytology examination.

Oxalates should not be used since they distort cellular morphology.

40 to 80% yield positive findings for malignancy by cell block or Papanicolaou cytologic examination. Higher proportion positive when peritoneal surface implanted with tumor.

False positives occur in heart failure, tuberculosis and peritonitis.

Specific Gravity

Specific gravity directly proportional to protein content of fluid. However, discrepancy between specific gravity and protein occurs so frequently as to limit its value.

Hydrometers standardized to read accurately at specific temperature. Most at 20°C; some at 15.5°C - others at 25°C. Such instruments will record values that are

spuriously low when used in fluids warmer than temperature of standardization. Correction factor of 0.001, subtracted or added for each 3°C below or above temperature of standardization.

Error of reading hydrometer scale ± 0.002.

Single value, usually taken as 1.016, as sharp dividing line in classification of transudates and exudates is gross oversimplification.

Routine study of ascitic fluid

Color

Appearance

Clot

Protein

Cytology

Culture

Cell count with differential

REFERENCES

Page, L.B. and Culver, P.J. Syllabus of laboratory examinations in clinical diagnosis. Cambridge, Mass., 1960. Harvard University Press.

Spak, I. On the clinical value of chemical analysis of ascites. Acta Chir. Scandinav. Supp. 261, 1960.

Loeb, R.F., Atchley, D.W. and Palmer, W.W. On the equilibrium condition between blood serum and serous cavity fluids. J. Gen. Physiol. 4:591-595, 1922.

CARCINOMA

	# pts.	Total protein g/100 ml	Bloody	Positive Cytology
Paddock, 1940	15	1.5-4.2		
Rovelstad, 1958	54	3.2 or greater in 34/54		52%
Tavel, 1959	30	<1.0 to >5.1 3.0 or less in 19 3.1 or greater in 11	15/53	77%
Spak, 1960	86 peritoneal carcinosis	3.97 ± 0.77	5/86	70%
	15 metastatic Ca liver	1.48 ± 0.66		
Hyman, 1963	11	3.1 or greater in all	5/11	

Wide range of protein concentrations. Protein levels appear to be higher when peritoneal surfaces extensively involved by neoplasm.

CARDIAC FAILURE

	# pts.	Total protein g/100 ml
Epstein, 1914	11	1.6 - 4.7 mean 3.2 9 were 3.1 or greater 1.6, 2.1 other 2
Paddock, 1940	21	1.5 - 5.3 mean 3.7
Tavel, 1959	16	1.1 - 4.5 10 were 3.0 or less 6 3.1 or greater
Spak, 1960	13	3.23 ± 0.72
Pillay, 1963 (Durban)	35	0.5 - 5.5 24 were 3.0 or less 11 3.1 or greater

Wide range of protein concentrations. Large number with relatively high values.

a. Originates from hepatic lymph?
b. Increased capillary permeability due to anoxia
c. Diuresis with fluid lost proportionately faster than protein

CARDIAC CIRRHOSIS

Total protein gm/100 ml

	Serum	Ascites
Spak, 1960	8.0	1.8
	7.1	1.5
	5.7	1.8
	5.5	1.6
	7.1	4.0

CHIARI'S SYNDROME
(Hepatic Vein Thrombosis)

	Protein gm/100 ml		Serum	
	Ascites	Total	Albumin	Globulin
Stuart and Bras, Quart. J. Med. 26:291-315,1937	2-2.5		>3.1 sometimes >4.1	
Little and Montgomery, Ann. Int. Med. 37:197, 1952	2.2	8.6	3.3	5.3
Norris Lancet 1:232, 1956	2.4	6.0	3.4	2.6
N.E.J.M. Case Report 258:749, 1958	2.9	6.2	4.2	2.0
Thompson Arch. Int. Med. 80:602,1947	1.0			
2 cases cited by Tavel, 1959	1.0 4.0			
Spak, 1960	1.5	6.0	2.9	3.1
Parkland Memorial Hospital,1959	2.6	6.0	3.2	2.8

Ascites is an almost invariable feature.

Despite hepatic engorgement, most protein values less than 3.0.

CIRRHOSIS

	# pts.	Total protein gm/100 ml
Paddock, 1940	69	0.6 - 3.2 mean 1.2
Rovelstad, 1958	32	29 2.5 or less 32 3.2 or less
Tavel, 1959	38	2.5 or less
Burack, 1960	18	17 2.5 or less 1-5.7*(staph. sepsis)
Spak, 1960	23	1.02 ± 0.48
Berendsohn, 1962	27	1.16 ± 0.70
Hyman, 1963	14	12 2.9 or less 1 4-4.9; 1 5-5.9
Pillay, 1963 (Durban)	23	17 3 or less 6 greater than 3
Parkland Memorial Hospital	53	51 2.5 or less 1 2.8 1 1.8 and 3.0 on same date

Almost all are 3.0 or less. For any serum albumin or total protein value, cirrhotics have less albumin or protein in ascitic fluid than patients with heart failure or tumor for example. Occasional values reported to be high. Would always suspect superimposed disease process in these patients.

CIRRHOSIS and TUBERCULOSIS

	# pts.	Total protein gm/100 ml
Burack, 1960	18	11 were 3 gm or less 7 were greater than 3 gm

CONSTRICTIVE PERICARDITIS

Andrews, G.W.S., Pickering, G.W. and Sellors, T.H. The aetiology of constrictive pericarditis, with special reference to tuberculous pericarditis, together with a note on polyserositis. Quart. J. Med. 17:291-321, 1948.

	Total protein gm/100 ml	
	0.5	
	2.5 - 3.5	
	4.3 - 5.3	Sterile and disappeared promptly after pericardectomy
	0.5 - 0.7	
	1.0 - 1.6	
	0.5	Constrictive pericarditis + peritoneal tubercle in some + probability of abdominal tbc in all
	0.5	
	0.6 - 2.2	
	0.5	
	4.0	
	3.0	
Hyman, 1963	2.2	

Wide range of protein concentration from low to high. Don't know if any had coexistent congestive cirrhosis, but unlikely to be many. Protein content of peritoneal effusion is no guide as to whether serous membrane is site of tuberculosis infection. Same statement pertains to pleural fluid.

HEPATOMA

	# pts	Total protein gm/100 ml	Alkaline phosphatase
Spak, 1960	13	1.27 ± 0.39	
Hyman, 1963	4	1 less than 3.0	
	3 bloody	1 3-3.9	5-15
		1 4-4.9	
		1 5-5.9	

Parkland Memorial Hospital

All had coexistent cirrhosis.

	Peritoneal metastases	Bloody	Tumor Cells	Protein: Serum Total	Serum Alb.	Serum Glob.	Protein: Ascites	Alkaline Phosphatase
1.	+	+	+	6.7	4.0	2.7	4.7	2.6
2.	0	N.R.	0	6.7	2.5	4.2	0.8	8.5
			Portal and hepatic vein thromboses					
3.	0	0	0	8.0	3.1	4.9	2.6	15.4-22.8
4.	0	+	0	6.0			2.3	3.1
			Portal vein thrombosis					
5.	+	0	N.D.	6.7	1.3	5.4	1.3	17.7 18.3
6.	0	0	N.D.	5.6	2.4	3.2	1.3	7.2-13.6
7.	0	N.R.	N.D.	7.1	2.7	4.4	1.4	
			0	6.9	2.7	4.2	0.9	5.3-12.9

N.R. = Not recorded
N.D. = Not done

Most patients have protein in range of uncomplicated cirrhosis. Few high values, greater than 3. Probable that these high values seen in patients with bloody fluids, and these may indicate peritoneal involvement. Usually have large, hard livers and alkaline phosphatases in range of uncomplicated cirrhosis. For comparable clinical picture with metastatic carcinoma, alkaline phosphatase levels tend to be higher.

HYPOTHYROIDISM

	# pts.	Total protein gm/100 ml
Kocen, 1963 Lancet 1:527	11	1.8-5.1 9 greater than 3.0 1 - 1.8; 1-2.4
Levine, 1960 Am. Heart J. 60:456	1	5.0

Clinical signs of hypothyroidism may not be obvious. Ascites may occur alone or with pleural and pericardial effusions. Latter usually have high protein concentration, too. Ascites present 2 months to 11 years. Responds to thyroid replacement within weeks to a few months.

Felt to be related to increased capillary permeability.

NEPHROSIS

Thirteen patients from literature. Protein concentration 0.1 - 0.9 with mean 0.3. White, watery fluid with slight opalescence.

PANCREATITIS

Barua, R.L., Villa, F. and Steigmann, F. Massive ascites due to pancreatitis. Am. J. Digest. Dis. 7:900-906, 1962.

Often produces small amounts of peritoneal fluid, 500-2000 ml. Fluid is usually serous or serosanguineous, may be milky. Occasionally ascites is massive and chronic in duration. High protein fluid. High concentration of amylase. Pseudocysts found in high proportion.

Amylase levels normal in tuberculous peritonitis, cirrhosis.

TUBERCULOUS PERITONITIS

	# pts.	Total protein gm/100 ml
Tavel, 1959	7	7 3.1 or greater
Burack, 1960	12	6 less than 3.0
Spak, 1960	7	4.93 ± 1.2
Johnston and Sanford 1961	6	1 - 2.4 5 - 5.0 to 7.2
Pillay, 1963 (Durban)	20	20 3.5 or greater

WBC differential on ascitic fluid rarely more than 20% polys.

Doughy abdomen is rare finding.

Tuberculin skin tests negative approx 1/3 - approx 1/2.

Chest X rays frequently normal.

Smears of fluid for acid fast bacilli usually negative. Cultures positive less than half. Guinea pig innoculation also frequently negative.

Lapa rotomy affords diagnosis in high proportion.

Hughes, H.J., Carr, D.T. and Geraci, J.E. Tuberculous peritonitis: a review of 34 cases with emphasis on the diagnostic aspects. Dis. Chest 38:42-50, 1960.

CHYLOUS ASCITES

Milky, lactescent ascites. Mechanical obstruction of the thoracic duct, cysterna chyli, intestinal lymphatics is usual common factor in pathogenesis of chylous ascites.

Wallis, R.L.M. and Schölberg, H.A. On chylous and pseudo-chylous ascites. Quart. J. Med. 4:153-204, 1911.

Tumor	81 - 56 carcinoma of abdominal organ
Infection	46 - 33 tbc
Cirrhosis	28

Nix, J.T., Albert, M., Dugas, J.E. and Wendt, D.L. Chylothorax and chylous ascites. A study of 302 selected cases. Am. J. Gastroenterology 28:40-55, 1957.

Malignant neoplasm	58	TBC	7
Benign neoplasms	4	Unknown	31
Benign lymphadenopathy	19	Trauma	
Liver disease	16	Surgical	2
		Nonpenetrating	17

Kelley, M.L. Jr., and Butt, H.R. Chylous ascites: An analysis of its etiology. Gastroenterology 39:161-170, 1960.

Lymphomas	33
Other malignancies	29

Nine adults, without malignancy - pancreatitis - 2, heart failure - 1, cirrhosis 1, thromboses portal vein - 1, post op abdomen - 1, adhesions - 1.
Eight children - 5 congenital atresia thoracic duct, 1 cirrhosis.

Protein concentration appears to be that of basic disease process.

Rovelstad et at, 1958

	Carcinoma	Cirrhosis
Total lipids > 0.35 gm/100 ml	38% (20)	0

8/20 chylous - lipids 0.59 - 1.43 gm%.
12/20 nonchylous

In carcinoma group, no correlation between gross milkiness and amount of fat.

Chylous fluid 10/116	Lipids
8 - Carcinoma	0.59-1.43
1 - Nephrotic	0.14
1 - Carcinoma + infection	0.34

CHEMISTRY and CAUSES of ASCITES

Epstein, A.A. Studies on the chemistry of serous effusions. J. Exper. Med. 20: 334-345, 1914.

Paddock, F. The diagnostic significance of serous fluids in disease. New Eng. J. Med. 223:1010, 1940.

Rovelstad, R.A., Bartholomew, L.G., Cain, J.C., McKenzie, B.F. and Soule, E.H. Ascites I. The value of examination of ascitic fluid and blood for lipids and for proteins by electrophoresis. Gastroenterology 34:436-450, 1958.

Tavel, M.E. Ascites: etiologic consideration with emphasis on the value of several laboratory findings in diagnosis. Am. J. Med. Sci. 237:727-742, 1959.

Spak, I. On the clinical value of chemical analysis of ascites. A study of the main proteins and some enzymes in ascites of differing etiology. Acta. Chir. Scand. Supp. 261, 1960.

Burack, W.R. and Hollister, R.M. Tuberculous peritonitis. Am. J. Med. 28:510-523, 1960.

Johnston, F.F. and Sanford, J.P. Tuberculous peritonitis. Ann. Int. Med. 54:1125-1133, 1961.

Berendsohn, S. Biochemical studies of the ascitic fluid in hepatic cirrhosis. Am. J. Dig. Dis. 1:160-166, 1962.

Hyman, S., Villa, F. and Steigmann, F. Mimetic aspects of ascites. J.A.M.A. 183: 651-655, 1963.

Pillay, V.K.G. Diagnostic significance of protein concentration in serous fluids. South Afr. Med. J. 37:379-381, 1963.

Cabot, R.C. The causes of ascites: a study of five thousand cases. Am. J. Med. Sci. 143:1-14, 1912.

Meigs, J.V. Fibroma of the ovary with ascites and hydrothorax. Ann. Surg. 110: 731-753, 1939.

CASE # 1, 26-38-17, J.B.: A 51-year-old unemployed male admitted with abdominal swelling of 2 weeks duration. Coexistent with this he developed aching in his upper abdomen and when swelling was considerable he became short of breath on exertion. Orthopnea and paroxysmal nocturnal dyspnea were denied. Patient denied alcohol intake, previous jaundice, fever, night sweats or weight loss. Appetite was said to have been good. Nausea and vomiting were denied - he admitted to intermittent bouts of watery diarrhea. Patient admitted to headaches since 1950 for which he took codeine. This was consumed in form of ETH with codeine, 4-6 ounces (42% alcohol) daily for past 3 to 4 years.

Px exam - thin, unkempt. No icterus. No lymphadenopathy. Moderate ascites, distended abdominal veins. Liver down 5 cm, no spleen.

Laboratory - Hemoglobin 13.1 to 14.4, WBC 4 to 7 thousand, Platelets 74,000, 120,000. Amylase < 320 Bilirubin 1.1, 1.0, Ceph. floc 3+/4+, Thymol turbidity 4.4, Alkaline phosphatase 14.9, 11.6. LAP - 270, BSP-18%, SGOT 53. Total protein 5.0, alb 3.4, glob. 1.6, then 5.8, alb 3.2, glob 2.6.

Ascites - cloudy, no clot, 153 WBC, 117 polys., protein 0.8, specific gravity 1.010. Bacterial smear neg., culture neg. Neg. for malignant cells.

X rays chest, Barium enema normal. Proctoscopy negative. Esophageal varices present. Liver scan normal.

Liver biopsy - portal (nutritional) cirrhosis with fatty metamorphosis. Ascites diminished rapidly with sodium restriction, thiazide diuretic and KCL.

Diagnosis: Laennec's Cirrhosis.
Portal hypertension with esophageal voices.
Ascites.

CASE # 2, 19-02-83, E.M.: A 40-year-old mentally retarded white female who had been institutionalized since age 13, admitted to Parkland Memorial Hospital 7/31/59 for evaluation of ascites. One year ago she began to complain of pain in the right side and developed ankle edema. Four months ago her abdomen began to swell. There was no history of jaundice, nausea or vomiting. Physical examination revealed massive ascites, dilated superficial abdominal veins, moderate edema to knees. Jaundice, spiders and hepatosplenomegaly were absent. Temperature occasionally reached 100-101°.

Laboratory - Hemoglobin 13.6, WBC 9,750, platelets 142,000. Urinalysis normal. Ascitic fluid - protein 2.6 gm%, no tumor cells, tbc culture negative. Serum amylase < 320. Tuberculin skin test - intermediate strength positive. Serology negative. No venous pressures performed. Liver biopsy - normal liver. Sinusoids appear slightly dilated in some areas.

	7/23	8/10	8/20	9/18
Bilirubin	1.4	1.4	1.4	8.8
Ceph floc		neg	2+	
Thymol turbidity	9.0	8.5	10.2	
Alkaline phtase		5.6		9.2
Total protein	6.5	6.0	7.3	
Albumin	3.8	3.2	3.7	
Globulin	2.7	2.8	3.6	
SGOT	58.6			
BSP			10%	
Glucose	108	50		34

The patient was discharged on 9/4/59 and appeared well for a week. Then ascites and leg edema increased, appetite fell off, she became progressively obtunded over ensuing week. She was readmitted on 9/17/59. The patient was unresponsive, BP 60/40, pulse 120. She was jaundiced. Marked ascites and superficial abdominal veins were present. No hepatosplenomegaly was detected. The patient died on 9/18.

Autopsy: Liver 900 grams; severe centrolobular necrosis. Numerous thrombi hepatic veins - varying ages. Large bile lakes.
Gall bladder - 2 large stones.
Spleen - 90 grams - thick adhesions at periphery.
Chronic congestion, marked increase in fibrous tissue.
Esophagus - no varices, esophageal erosions.
Duodenal ulcer.

Diagnosis: Hepatic vein thrombosis.

CASE # 3, 17-36-51, L.B.H.: 40-year-old male, heavy machine mechanic; seen first July 1958 for swelling of abdomen of 10 days duration. He had experienced intermittent leg edema for one year, fatigue, weight loss and anorexia of 7 to 8 months duration.

Px exam - ascites only abnormal finding.

Laboratory - Hgb. 15.5 Bilirubin 0.3, ceph floc 1+, thymol turbidity 13.8 BSP 17%
Total protein 5.2, albumin 2.8, globulin 2.4. Serum amylase < 320.
Chylous ascites - protein 2.0 gm%

He was discharged, and received Rx for strongyloides which had been detected in stool. His ascites disappeared spontaneously on good diet, rest and vitamins and patient remained well till Oct. 1958, when weakness returned and ascites and peripheral edema returned.

Px exam revealed evidence of some weight loss, ascites and 2+ peripheral edema.

Laboratory - November, 1958. Hemoglobin 15.5 Bilirubin 0.1 Ceph floc 4+, thymol turbidity 39 units, alkaline phosphatase 14, BSP 24%. Serum amylase < 320. PPD negative. Total protein 4.2, albumin 2.2, globulin 2.0.

Ascites - milky, did not clot, protein 1.4 gm% amylase < 320, neg for culture and for malignant cells.

X rays - obliterated left costophrenic angle. GI series negative. Liver scan normal.

Exploratory laparotomy by Dr. Tom Shires. Finely granular fibrous liver and distended lymphatics throughout course of entire small bowel were only abnormal findings. Biopsies revealed a postnecrotic type cirrhosis. No evidence of neoplasm.

Patient subsequently died, and cirrhosis without evidence of neoplasm, thoracic duct or cysterna clyli obstruction found. Acute and chronic inflammation of retroperitoneal nodes found.

Diagnosis: Cirrhosis.
Splenomegaly due to portal hypertension.
Retroperitoneal lymphadenitis.
Chylous ascites.

CASE # 4, 26-09-76, C.W.: 62-year-old woman noted abdominal swelling, fall 1961. This persisted with some increase till admission to Parkland in October, 1963. Aside from the increase in abdominal size, she was asymptomatic. Several days prior to admission, her physician removed 5000 cc of dark sanguinous fluid.

Px exam revealed abdomen markedly distended with fluid. Pelvic exam negative - including one by gyn consultant.

Laboratory - Hemoglobin 13.8, Bilirubin 1.3, 0.2 Ceph floc 3+ then 1+, thymol turbidity 4.4, Alkaline phosphatase 4.8, 3.4. SGOT 20. Total protein 5.5, albumin 3.5, globulin 2.0.
Tuberculin skin test negative. Serum amylase < 320.

Ascites - chocolate colored fluid, + guaiac, 165,000 RBC, 36,000 WBC, 99% polys. Amylase < 320, protein 2.3. Negative for malignant cells, culture negative.

Chest X ray - negative.

Laparotomy revealed a huge left ovarian cyst containing 9.5 liters of brown-mucoid fluid.

Diagnosis: - Papillary mucinous cystadenoma of left ovary, benign.

Volwiler, W. , Grindlay, J.H. and Bollman, J.L. The relation of portal vein pressure to the formation of ascites. An experimental study. Gastroenterology 14:40-55, 1950.

Nix, J.T., Mann, F.C., Bollman, J.L., Grindlay, J.H. and Flock, E.V. Alterations of protein constituents of lymph by specific injury to the liver. Am. J. Physiol. 164:119-122, 1951.

Schilling, J.A. Experimental ascites. Studies of electrolyte balance in dogs with partial and complete occlusion of the portal vein and of the vena cava above and below the liver. J. Clin. Invest. 31:702-710, 1952.

Mallet-Guy, P., Devic, G., Feroldi, J., and Desjacques, P. Etude experimentale des ascites; stenoses veineuses post-hepatiques et transposition du foie dans le thorax. Lyon Chir. 49:153, 1954.

Hyatt, R.E., Lawrence, G.H. and Smith, J.R. Observations on the origin of ascites from experimental hepatic congestion. J. of Lab. and Clin. Med. 45:274-280,1955.

Aiello, R.G., Enquist, I.F., Skezono, E. and Levowitz, B.S. An experimental study of the role of hepatic lymph in the production of ascites. S.G.O. 111:77-81, 1960.

Baggenstoss, A.H. and Cain, J.C. The hepatic hilar lymphatics of man. Their relation to ascites. New Eng. J. Med. 256:531-535, 1957.

Dumont, A.E. and Mulholland, J.H. Flow rate and composition of thoracic-duct lymph in patients with cirrhosis. New Eng. J. Med. 263:471-474, 1960.

Dumont, A.E. and Mulholland, J.H. Alterations in thoracic duct lymph flow in hepatic cirrhosis: significance in portal hypertension. Ann. Surg. 156:668-677, 1962.

Dumont, A.E. and Mulholland, J.H. Effect of thoracic duct to esophagus shunt in dogs with vena caval constriction. Am. J. Physiol. 204:289-290, 1963.

Davis, J.A., Howell, D.S. and Southworth, J.L. Mechanisms of fluid and electrolyte retention in experimental preparations in dogs. III. Effect of adrenalectomy and subsequent desoxy corticosterone acetate administration on ascites formation. Circulation Research I:260-270, 1953.

Davis, J.O., Ayers, C.R. and Carpenter, C. C.J. Renal origin of an aldosterone-stimulating hormone in dogs with thoracic caval constriction and in sodium-depleted dogs. J.C.I. 40:1466-1474, 1961.

STARLING FORCES in ASCITES FORMATION

Cherrick, G.R., Kerr, D.N., Read, A.E., and Sherlock, S. Colloid osmotic pressure and hydrostatic pressure relationships in the formation of ascites in hepatic cirrhosis. Clin. Sci. 19:361, 1960.

Atkinson, M. and Losowsky, M.S. The mechanism of ascites formation in chronic liver disease. Quart. J. Med. 30:153-166, 1961.

Atkinson, M. and Losowsky, M.S. Plasma colloid osmotic pressure in relation to the formation of ascites and oedema in liver disease. Clin. Sci. 22:383-389, 1962.

Losowsky, M.S. and Davidson, C.S. The source of ascitic fluid in cirrhosis of the liver. Arch. Int. Med. 110:279-282, 1962. (Editorial)

Losowsky, M.S. Local factors in ascites formation in chronic liver disease. Gastroenterology 45:429-432, 1963. (Editorial)

ASCITES with PORTAL VEIN THROMBOSIS

Pallette, E.C. Portal Thrombosis. California and Western Med. 45:324-327, 1936.

Ratnoff, A.D., Conley, C.L. and Berthrong, M. The differentiation between extrahepatic and intrahepatic obstruction of the portal circulation. A clinical study of the "Banti Syndrome." Bull. Johns Hopkins Hospital 87:305-327, 1950.

Eisenmenger, W.J. and Nickel, W.F. Relationship of portal hypertension to ascites in Laennec's Cirrhosis. Am. J. Med. 20:879-889, 1956.

Ekman, C. Portal hypertension. Acta Chir. Scand. Supp. 222, 1957.

Turner, M.D., Sherlock, S. and Steiner, R.E. Splenic venography and intrasplenic pressure in the clinical investigation of the portal venous system. Am. J. Med. 23:846-859, 1957.

Baggenstoss, A.H. and Wollaeger, E.E. Portal hypertension due to chronic occlusion of the extrahepatic portion of the portal vein: Its relation to ascites. Am. J. Med. 21:16-25, 1956.