Cholelithiasis in children with sickle cell anemia: Report of a case

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A case of cholelithiasis in a child with sickle cell anemia is reported to illustrate some of the controversial features in management. The apparent high prevalence of cholelithiasis in children with sickle cell anemia appears to be an indication for screening for gallbladder disease in these children. Ultrasonography is a useful modality for such purposes. Since there is great difficulty in differentiating the abdominal pain of vaso-occlusive sickle cell crisis from that of acute cholecystitis, the performance of elective cholecystectomy in the symptomatic child with sickle cell anemia may be indicated. Recent literature demonstrates low morbidity in such circumstances. This low morbidity is achieved by preoperative transfusions of hemoglobin A-containing erythrocytes and prevention of the factors that initiate vaso-occlusive crises.

Gallbladder disease is known to occur frequently in patients with chronic hemolytic anemias.[1-3] The prevalence of cholelithiasis in children with sickle cell anemia has been reported to vary between 10 and 37 percent.[3-5] With the use of ultrasound in addition to cholecystography, cholelithiasis has been detected in children as young as 4 years of age.[5] The prevalence of cholelithiasis in children with sickle cell anemia below the age of 10 years has been found to be 10 percent. Over 10 years of age, 55 percent of children with homozygous (hemoglobin SS) disease have gallstones.[5]

Several studies suggest the use of diagnostic ultrasonography as a routine screening procedure in children with sickle cell disease.[5,6] The absence of harmful ionizing radiation makes this procedure particularly attractive for screening in the pediatric age group.[5,6] Since children may not cooperate in the performance of oral cholecystography and since some gallbladders will not opacify with either oral or intravenous contrast material, gallbladder ultrasonography provides a useful, noninvasive method for the detection of gallbladder dysfunction in sickle cell disease.[4-6]

Cholecystitis and cholelithiasis are included in the differential diagnosis of causes of abdominal pain in children with sickle cell anemia, and have been known to mask an abdominal crisis.[1,3,4,6,7] There is disagreement in the pediatric literature about the routine performance of elective cholecystectomies on children with sickle cell anemia and asymptomatic cholelithiasis. The pediatric literature does support the performance of elective cholecystectomy on children with hemoglobin (HB) SS disease who have symptomatic cholelithiasis and cholecystitis.[1,3,4,7]

Report of case

A 7-year-old black boy with known HB SS disease was initially seen on a pediatric referral in November 1979, because of frequent bouts of pneumonia and marked eosinophilia with a persistent leukocytosis. He had a history of multiple vaso-occlusive crises always manifested by abdominal pain and bone pain. Medications included ampicillin and folic acid. He had never received the pneumococcal polysaccaride vaccine, nor yearly influenzal vaccinations. He had been hospitalized at least eight times for abdominal crises and pneumonia, and had received multiple blood transfusions.

His mother related that his "heart was enlarged," and that he sometimes had difficulty breathing. He had evidence of good weight gain and was occasionally enuretic.

Physical examination revealed the patient to be in no distress. He was adequately developed and nourished and his weight and height were at the seventy-fifth percentile for a 7-year-old boy. There was prominence of the frontal and zygomatic regions of the skull. The tonsils were hypertrophic and erythematous. Anterior cervical lymphadenopathy was present. A grade 2/6 systolic flow murmur was present at the upper left sternal border and

changed with position. The liver was palpable 1 cm. below the right costal margin and the spleen was palpable 1 cm. below the left costal margin. The penis was uncircumcised. The only other remarkable feature was widespread dental caries.

The patient was admitted to Fort Worth Osteopathic Hospital for evaluation of the eosinophilia. The results of hematologic evaluation are shown, in part, in Table 1. In addition, there were four nucleated erythrocytes per 100 leukocytes, with occasional sickle cell forms. A peripheral smear revealed sickle cells along with anisocytosis, macrocytes, polychromatophilia, target cells, and an apparent increase in platelets. Leukocytosis was present. Hemoglobin electrophoresis revealed 93 percent hemoglobin S, 5 percent hemoglobin F, and 2 percent hemoglobin A_2. Spinous process bone marrow aspiration revealed erythroid hyperplasia with a marked increase in the number of eosinophils and their precursors. However, no evidence of malignant characteristics was seen.

Several midstream urine cultures revealed greater than 100,000 colonies per milliliter Escherichia coli. A catheterized specimen was sterile.

An electrocardiogram was suggestive of left ventricular hypertrophy but was actually normal for the child's age. A chest x-ray film was interpreted as normal. An intravenous urogram showed possible splenic enlargement but otherwise was normal.

The glucose 6-phosphate dehydrogenase value was normal at 20 minutes. The Monospot test for infectious mononucleosis was negative. The serum IgE value was 66 units (normal). Multiple serologic specimens were sent to the Center for Disease Control in Atlanta. SGOT was 29 I.U./ml. and SGPT was 14 I.U./ml.

Tuberculosis and monilia skin tests were negative. National Institute of Health tapes detected no pinworms. Stools were negative for occult blood, ova, and parasites.

An adult hematologist agreed with plans to immunize the patient with pneumococcal polysaccharide vaccine, continue supplementary folic acid, and await the serologic results. Fluid restrictive therapy and special instructions on voiding were prescribed for enuresis. The parents were advised to have the child see a pedodontist because of the dental caries. Approximately 1 month later, the serologic test results from the Center for Disease Control revealed a Toxocara titer in a dilution greater than 1:256.

The patient was subsequently lost to pediatric follow-up, but was followed by his family physician. He appeared at Fort Worth Osteopathic Hospital in July 1980, at which time a request was made by the family physician for a pediatric consultation prior to the performance of an elective tonsillectomy and adenoidectomy. The history was unchanged except for complaints of frequent bouts of viral tonsillitis and vague upper right quadrant pain, intolerance to fried and greasy foods, flatulence, occasional diarrhea, and episodic abdominal bloating.

Physical examination revealed the patient's height to be at the fiftieth percentile for his age and weight at the sixtieth percentile. Widespread severe dental caries was present, and oral hygiene was poor. The tonsils were hypertrophic and cryptic. The heart murmur noted before was still present. Palpation revealed neither hepatosplenomegaly nor abdominal tenderness. Eosinophilia (32 percent) was still present.

Recommendations were made to cancel surgery because of the lack of a strong indication for it and the view that its performance could become life threatening. Monthly prophylaxis with benzathine penicillin was recommended, as was an ophthalmology consultation because of the history of toxocariasis. Additional recommendations included an ultrasound examination of the gallbladder, pedodontic consultation, removal of dogs and cats from the home environment, and yearly influenzal immunization. The case was subsequently turned over for pediatric management.

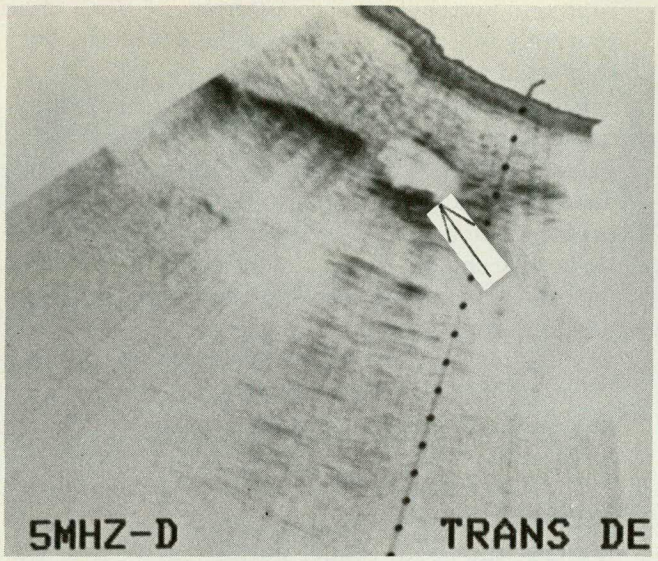
Pertinent hematologic data are shown in Table 1. An automated blood chemistry profile revealed the total bilirubin value to be 4.6 mg./dl., SGOT, 62 and, SGPT, 183 I.U./ml. A test for hepatitis B surface antigen was negative.

Oral cholecystography demonstrated nonvisualization of the gallbladder. Ultrasonography of the gallbladder revealed the presence of cholelithiasis (Figs. 1 and 2). Ophthalmologic consultation revealed no evidence of either toxocara involvement of the retina or sickle cell retinopathy.

The patient was admitted to the W. I. Cook Children's Hospital on July 28. The pediatric hematologist and a pediatric surgeon both thought that a cholecystectomy was indicated because of symptomatic cholelithiasis. The pediatric hematologist recommended hypertransfusion with hemoglobin A-containing erythrocytes to a hematocrit value of 45-50 percent, followed by a 3½ to 4-week waiting period, followed by a second hypertransfusion of hemoglobin A-containing erythrocytes to a hematocrit value of 45 to 50 percent. He thought that these two episodes of transfusions should yield a decrease of the hemoglobin S level below 30 percent, which would be satisfactory to prevent intraoperative or postoperative hypoxia and resultant sickling of the erythrocytes.

During this hospital stay a chest x-ray film revealed cardiomegaly. Double-dose oral cholecystography was normal (Fig. 3) but suggested gallstones. An electrocardiogram was normal. A peripheral smear revealed the presence of Howell-Jolly bodies. The hemoglobin and hematocrit values 6 hours after the patient received three separate transfusions of fresh, frozen, compatible, washed, sickle-negative packed erythrocytes in a dose of 10 ml./kg. are shown in Table 1.

Three weeks later the patient was readmitted to Cook Children's Hospital. The admission hematologic values are shown in Table 1. Sickled cells and target cells were also noted. The hemoglobin electrophoresis revealed 55 percent hemoglobin A, 40 percent hemoglobin S, and 5 percent hemoglobin A_2. The patient received four units of fresh, frozen, compatible, washed, sickle-negative, packed erythrocytes. Repeat hemoglobin electrophoresis after the last transfusion revealed the presence of 77 percent hemoglobin A, 19 percent hemoglobin S, and 4



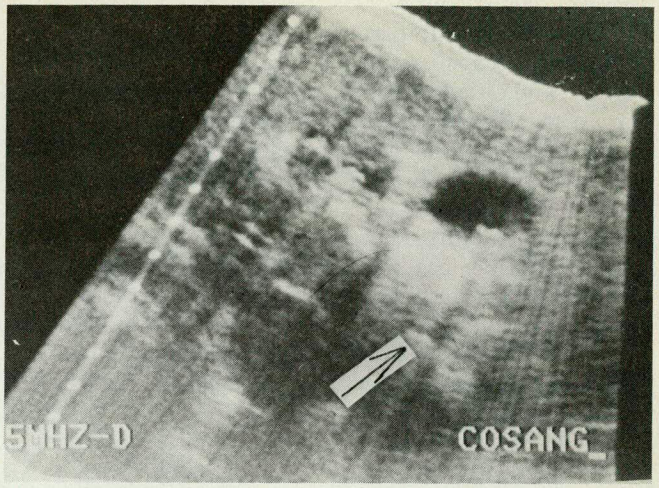
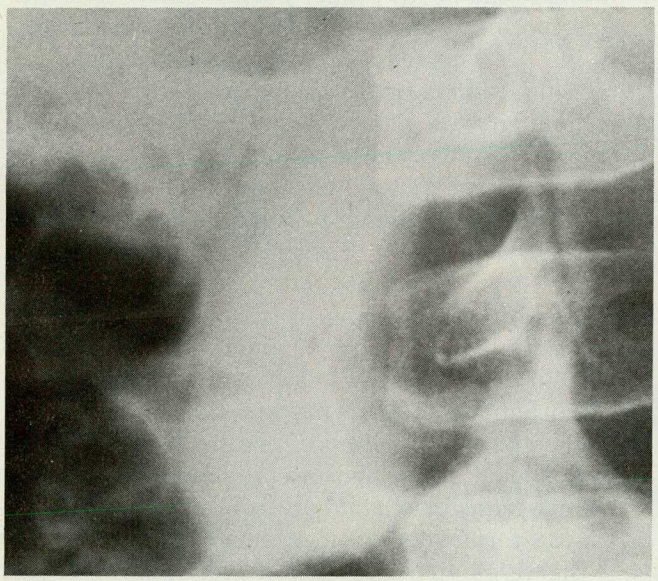


Fig. 1. *Gallstones (arrow) detected by ultrasound examination of the gallbladder.* Fig. 2. *Same as Figure 1. Note echos produced by the stones (arrow).*



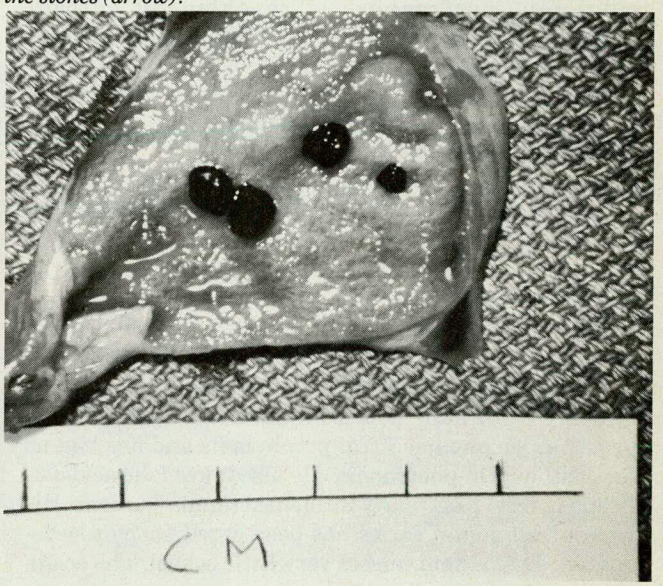


Fig. 3. *Normal oral cholecystogram.* Fig. 4. *Operative specimen showing cholelithiasis and chronic cholecystitis.*

TABLE 1. HEMATOLOGIC STUDY RESULTS.

	November 1979 admission	July 1980 pediatric consultation	July 1980 post-transfusion	August 1980 admission	September 1980 admission	September 1980 post-transfusion
Hemoglobin (grams/dl.)	8.6	7.5	14.7	7.6	9.4	15.5
Hematocrit (%)	25.6	22.6	44.7	23.1	28.7	46.7
Leukocytes	22,500	18,300		20,600	14,000	
Segmented forms	46	30		28	46	
Lymphocytes	27	34		39	26	
Monocytes		4		10	4	
Eosinophils	27	32		21	23	
Basophils				2	1	
Reticulocytes (%)	8.4					
Platelets	306,000				Normal	
Total eosinophils	7,880					
Hemoglobin A concentration	0 percent			Pretransfusion, 55% Post-transfusion, 77%		80%

percent hemoglobin A_2.

The patient's temperature began to spike after the first set of transfusions was completed. Blood, urine, throat, and nasopharyngeal cultures grew no pathologic organisms. The chest x-ray film was unchanged from before. The pediatric hematologist placed the child on a regimen of Amoxicillin 500 mg. every 8 hours orally. The patient was discharged and the surgery postponed.

He was readmitted 3 weeks later on September 6. The history and physical findings remained unchanged except for a decrease in the intensity of the heart murmur. The admission complete blood count results are shown in part in Table 1. In addition, target cells were present. The prothrombin and partial thromboplastin times and platelet count were normal.

He was transfused with two units of fresh, frozen, compatible, washed, sickle-negative, packed erythrocytes (10 ml./kg). Post-transfusion hemoglobin and hematocrit values are shown in Table 1. A preoperative hemoglobin electrophoresis revealed 80 percent hemoglobin A, 17 percent hemoglobin S, and 3 percent hemoglobin A_2.

The cholecystectomy and an incidental appendectomy were performed. Pathologic examination revealed the presence of chronic cholecystitis and cholelithiasis (Fig. 4), with a normal appendix.

The patient did well postoperatively and was discharged on the seventh postoperative day. He was to take folic acid 0.5 mg. twice daily, and see a pedodontist for further dental care.

Followup examination has revealed that the patient no longer has right upper quadrant pain nor intolerance to fatty, greasy, or fried foods. He presently receives monthly benzathine penicillin injections by the family physician as pneumococcal prophylaxis and has had no further bouts of pneumonia. He also takes folic acid daily and is receiving yearly influenzal immunizations. His widespread dental caries has been cared for by a pedodontist. The patient misses very little school. The eosinophilia has resolved.

Discussion

Some authors do not recommend the performance of elective cholecystectomy in the asymptomatic patient with sickle cell anemia.[8] Gibson and coworkers,[8] for example, believe that these patients have a shortened life expectancy and are relatively poor surgical risks. Their study population ranged in age from 8 to 48 years, with an average of 26 years. Of the 16 symptomatic patients who had elective cholecystectomies, 6 experienced complications and 1 died. Most of the literature, however, supports the view that patients with symptomatic cholelithiasis and/or cholecystitis should have elective cholecystectomies performed.[1,3,4,7] On the basis of the frequency of gallstones in patients over the age of 10 years, the likelihood of symptoms, possibly with serious complications, and the apparent ease with which the properly prepared sickle cell patient comes through surgery, cholecystography and/or cholecystosonography appears to be routinely indicated in adolescents and adults with sickle cell anemia.[4,6]

Biliary obstruction may create a serious diagnostic and therapeutic dilemma causing confusion when a patient with HB SS disease presents with abdominal pain.[1,3,4,6,7] Because it is not possible to predict which patient will experience complications with cholelithiasis, and because the manifestations of such complications may be virtually impossible to diagnose in the presence of sickle cell anemia, it seems plausible to consider elective cholecystectomy in all patients with sickle cell anemia and cholelithiasis, especially if they are symptomatic.

The collections of larger series of patients with sickle cell anemia who have had surgery seem to indicate that with proper perioperative preparation, morbidity and mortality have decreased greatly.[1,3,7,9-11] The pediatric surgical literature indicates that with competent perioperative management of children with sickle hemoglobinopathy, surgical mortality can be avoided and morbidity decreased.[9-11]

The specific method of preparation of the child with sickle cell anemia for surgery is another subject of much debate. The preparation of children with sickle cell disease for surgery by a one-time preoperative transfusion of 15-20 ml./kg. of packed erythrocytes, producing a hematocrit of at least 38 percent is rather easy to accomplish. The absence of morbidity and mortality associated with its use suggests that this kind of preoperative preparation is more desirable than previously utilized exchange transfusions. It certainly is efficacious and easy to perform in an emergency.[11]

Elective hypertransfusion over several weeks, on the other hand, affords the opportunity of monitoring the quantity of hemoglobin S available for sickling prior to surgery.[9] This alternative is less hazardous, but it is more cumbersome and also more costly. It may, however, be performed on an outpatient basis over several weeks. This method also affords time for the blood volume to stabilize, equilibration to occur, and tachycardia to subside. It essentially converts a patient with sickle cell hemoglobinopathy to one with little or no added risk. Certainly, meticulous efforts to prevent the patient from becoming acidotic, hypoxic, hypotensive, hypothermic or hyperthermic, or dehydrated also assist in the prevention of vaso-occlusive crises and their consequent ischemic infarctions.

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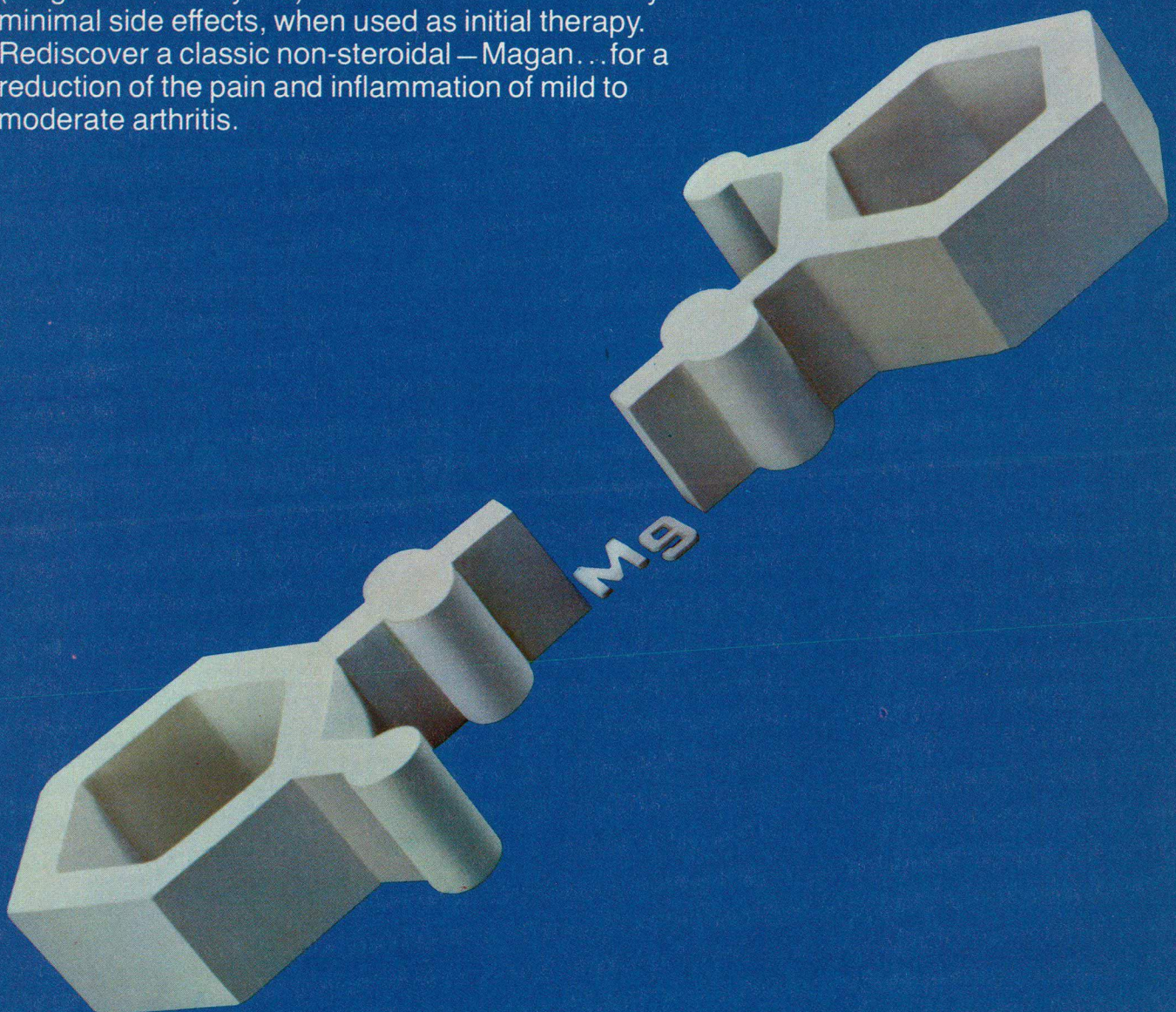

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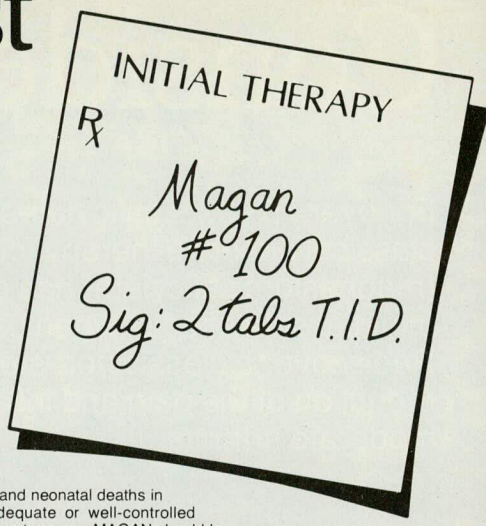

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Clinical Pharmacology: Salicylic acid is the active moiety released into the plasma by MAGAN (magnesium salicylate). Salicylic acid is enzymatically biotransformed through two pathways to salicyluric acid and salicylphenolic glucuronide and eliminated in the urine.

Oral salicylates are absorbed rapidly, partly from the stomach but mostly from the upper intestine. Salicylic acid is rapidly distributed throughout all body tissues and most transcellular fluids, mainly by pH-dependent passive processes. It can be detected in synovial, spinal and peritoneal fluid, in saliva and in milk. It readily crosses the placental barrier. From 50% to 90% of salicylic acid is bound to plasma proteins, especially albumin.

Following the ingestion of a single dose of 524 mg of magnesium salicylate, a peak concentration of 3.6 mg/dl salicylic acid is reached in 1.5 hours with a T ½ of 2 hours. The major biotransformation paths for the elimination of salicylic acid from the plasma become saturated by low doses of salicylic acid. As a result, repeated doses of MAGAN increase the plasma concentration and markedly prolong the plasma half-time. The plasma concentration of salicylic acid is increased by conditions that reduce the glomerular filtration rate or tubular secretion such as renal disease or the presence of inhibitors that compete for the transport system such as probenecid.

Therapeutic plasma concentrations of salicylic acid for an adequate anti-inflammatory effect needed for the treatment of rheumatoid arthritis range between 20-30 mg/dl. Effective analgesia is achieved at lower concentrations. Salicylates relieve pain by both a peripheral and a CNS effect. Salicylates inhibit the synthesis of prostaglandins; the importance of this mechanism in analgesia and antiinflammation has not been fully elucidated. Salicylates have an antipyretic effect in febrile patients but little in subjects with normal temperatures. This appears to be due to the inhibition of the synthesis of prostaglandins which are powerful pyrogens that affect the hypothalamus. Higher therapeutic concentrations cause reversible tinnitus and high tone hearing loss. Full therapeutic doses of salicylates increase oxygen consumption and CO_2 production. They cause an extracellular and intracellular respiratory alkalosis which is rapidly compensated. Salicylates irritate the gastric mucosa and frequently lead to blood loss in the stool; this effect is more pronounced with aspirin than magnesium salicylate. Salicylates in large doses (over 6 g/day) reduce the plasma prothrombin level. In contrast to aspirin, magnesium salicylate does not affect platelets. Salicylic acid increases the urinary excretion of urates at higher doses but may decrease excretion at lower doses.

Indications and Usage: MAGAN is indicated for the relief of the signs and symptoms of rheumatoid arthritis, osteoarthritis, bursitis and other musculoskeletal disorders.

Contraindications: MAGAN is contraindicated in patients with advanced chronic renal insufficiency. It may counteract the effect of uricosuric agents and should not be prescribed for patients on such drugs.

Warnings: As with all salicylates, MAGAN should be avoided or administered with caution to patients with liver damage, pre-existing hypoprothrombinemia, vitamin K deficiency and before surgery.

Precautions: *General*–Appropriate precautions should be taken in prescribing MAGAN for persons known to be sensitive to salicylates and in patients with erosive gastritis or peptic ulcer. If a reaction develops, the drug should be discontinued. MAGAN should be used with caution, if at all, concomitantly with anticoagulants. Appropriate precautions should be taken in administering MAGAN to patients with any impairment of renal function including discontinuing other drugs containing magnesium and monitoring serum magnesium levels if dosage levels of MAGAN are high.

Drug Interactions–Even small doses of MAGAN should not be given with uricosuric agents such as probenecid that decrease tubular reabsorption because it counteracts their effect. Large doses of MAGAN cause hypoprothrombinemia. Lower doses enhance the effects of anticoagulants such as coumadin and must be used with caution in patients receiving anticoagulants that affect the prothrombin time. Caution should also be exercised in patients concurrently treated with a sulfonylurea hypoglycemic agent or methotrexate because of the drug's capability of displacing them from the plasma protein binding sites, resulting in enhanced action of these agents. A similar displacement of barbiturates and diphenylhydantoin may occur; diphenylhydantoin intoxication has been precipitated by the consumption of aspirin. Salicylates inhibit the diuretic action of spironolactone.

Carcinogenesis, Mutagenesis, Impairment of Fertility–There have been no studies in animals or humans to evaluate the carcinogenesis, mutagenesis or impairment of fertility for magnesium salicylate. Aspirin causes testicular atrophy and inhibition of spermatogenesis in animals.

Pregnancy–Category C.

1. Teratogenic effects–Aspirin has been shown to be teratogenic in animals and to increase the incidence of still births and neonatal deaths in women. There are no adequate or well-controlled studies of MAGAN in pregnant women. MAGAN should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

2. Non-Teratogenic effects – Chronic, high dose salicylate therapy of pregnant women increases the length of gestation and the frequency of post maturity and prolongs spontaneous labor. It is recommended MAGAN be taken during the last three months of pregnancy only under the close supervision of a physician.

Nursing Mothers–Since salicylates are excreted in human milk, caution should be exercised when MAGAN is administered to a nursing woman.

Pediatric Use–Safety and effectiveness of MAGAN in children have not been established.

Adverse Reactions: Magnesium salicylate in large doses has a hypoprothrombinemic effect and should be given with caution in patients receiving anticoagulant drugs, patients with liver damage, pre-existing hypoprothrombinemia, vitamin K deficiency or before surgery.

Salicylates given in overdose produce stimulation (often manifested as tinnitus) followed by depression of the central nervous system. The dosage should be lowered at the onset of tinnitus.

Salicylates may cause gastric mucosal irritation and bleeding. However, fecal blood loss in patients taking MAGAN is significantly less than in those taking aspirin.

MAGAN should not be given to patients with severe renal damage because of the possibility of hypermagnesemia.

In moderate to high doses, salicylates lower the blood glucose in diabetics. Aspirin-induced hypoglycemia has been described in adults undergoing hemodialysis.

Unlike aspirin, magnesium salicylate is not known to affect the platelet adhesiveness involved in the clotting mechanism; and therefore, does not prolong bleeding time. MAGAN has not been associated with reactions causing asthmatic attacks in susceptible people.

Overdosage: Acute overdosage results in salicylate toxicity. Early signs and symptoms from repeated larger doses as well as a large single dose consist of headache, dizziness, tinnitus (which may be absent in children or the elderly), difficulty in hearing, dimness of vision, mental confusion, lassitude, drowsiness, sweating, thirst, hyperventilation, nausea, vomiting and occasionally diarrhea. More severe salicylate poisoning is manifested by CNS disturbances including EEG abnormalities. Hyperventilation occurs producing initial respiratory alkalosis. This is followed by severe metabolic acidosis with dehydration and loss of potassium. Restlessness, garrulity, incoherent speech, apprehension, vertigo, tremor, diplopia, maniacal delirium, hallucinations, generalized convulsions and coma may occur. Toxic symptoms may occur at serum levels greater than 20 mg/dl in patients over 60 years of age. Hyperventilation may occur at plasma salicylate levels over 35 mg/dl. Death may result from salicylate levels between 45-75 mg/dl. As with other salicylates, 10 to 30 g of the drug may be fatal. Renal or hepatic insufficiency and fever and dehydration in children enhance the acute toxicity of salicylate overdoses. Treatment of acute poisoning is a medical emergency and should be undertaken in a hospital. Serum Salicylate, Na, K, Cl, CO_2 levels, pH, BUN, blood glucose and urine pH and specific gravity should be obtained. Emesis should be induced or gastric lavage performed. Activated charcoal may be administered. Hyperthermia should be controlled with tepid water sponging. Dehydration should be treated and acid-base imbalance corrected. A high concentration of salicylic acid in the brain may be fatal. Correction of acidosis shifts salicylate from the brain to the plasma. A bicarbonate solution should be infused to maintain an alkaline diuresis. Care should be taken to avoid pulmonary edema. The blood pH, plasma Pco_2 and plasma glucose level should be monitored frequently. Ketosis and hypoglycemia may be corrected by glucose infusions and hypokalemia by potassium chloride added to the intravenous infusate. Avoid respiratory depressants. Shock may be combatted by plasma infusions. Hemorrhagic phenomena may necessitate whole blood transfusions or vitamin K_1. In severe intoxication, exchange transfusion, peritoneal dialysis, hemodialysis or hemoperfusion should be performed to remove plasma salicylic acid. Dialysis should be seriously considered if the patient's condition is worsening despite appropriate therapy.

Dosage and Administration: The dosage for MAGAN in the treatment of musculoskeletal disorders such as arthritis should be adjusted according to individual patient's needs. The recommended initial regimen is two tablets three times per day. Dosage may be increased, if necessary, to achieve the desired therapeutic effect. In adjusting the dosage, the physician should monitor the dose limiting parameters such as tinnitus and/or serum salicylate over 30 mg/dl.

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Great Britain in order to fulfill this purpose.

In general, most of the topics explored are covered only superficially. For example, the chapter on congestive heart failure contains an extensive review of the etiologies of heart failure in table format, but there is no mention of the basic pathophysiologic mechanisms. The concept of using various drugs to alter preload and afterload in congestive heart failure is also omitted. Instead, one reads about such outdated methods as rotating tourniquets and phlebotomy in this particular symptom complex.

Another fault of the book is that it is written from the perspective of a physician practicing outside of the United States. A family practitioner in this country could have difficulty relating Wharton's methods to his practice needs. Even though it is interesting to learn about these methodological differences, the material does not become any more applicable. Numerous examples of how Wharton's practice differs from accepted practices in the United States can be found in the text. Three cases that illustrate this problem include: the treatment of acute myocardial infarction at home versus hospitalization of all myocardial infarctions; the employment of drugs which are not approved for use in the United States (amiodarone, mexilitine, IV disopyramide, more numerous beta blockers); and the method for S.B.E. prophylaxis which does not follow American Heart Association guidelines.

Certain treatments that the author recommends are contraindicated in recent medical literature. One example of this situation can be found in Wharton's prescription of intravenous disopyramide for ventricular arrhythmias in individuals who have acute myocardial infarctions. Current research suggests that a practitioner should avoid using oral or intravenous disopyramide as an agent of first choice since it exhibits the greatest myocardial depressant effect when compared to other approved type I antiarrhythmics.

The book is not entirely without merit. Certain problems ranging from endocarditis to hypertension, dysrhythmias and ischemic heart disease are adequately discussed, and an excellent review of the various etiologies of chest pain is given. The chapter on ischemic heart disease provides an up-to-date report on its medical management and a short synopsis on the use of slow channel-blocking agents is included. The section on hypertension is also well done. The author covers many of its organic causes, and he outlines a "step care" form of treatment he utilizes.

I would not recommend this book to the general practitioner as a review of the problems in cardiology. A physician's time could be better spent reading more complete and pertinent texts.

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Pathologic mechanisms and human disease

By Roderick A. Cawson, Alexander W. McCracken, and Peter B. Marcus. Pp. 594, with illus. C.V. Mosby Co., 11830 Westline Industrial Dr., St. Louis 63141, 1982. $24.95 (paper).

This is a concise, clinically oriented, and abundantly illustrated book on pathology, a specialty which the authors rightfully describe as the "common ground on which all disciplines in the health care professions meet."

Exclusive of index, there are 574 pages in this book, less than half the number in Robbins and Cotran's *Pathologic basis of disease*. Histologic, electron microscopic, clinical, and diagrammatic illustrations are plentiful, cutting down further the textual content. The book covers the standard subjects of pathology, starting with a chapter on the normal cell and intercellular substance and concluding with a chapter on sexually transmitted diseases. There is also a 55-entry glossary.

This book has several merits. It succeeds in presenting pathology as a basic and a clinical science in the briefest manner possible. Of special interest to pathologists are the excellent clinical and specimen photographs from the Gordon Museum of Guy's Hospital (London) where Addison, Bright, and Hodgkin practiced medicine. Students and teachers will appreciate the many tables that organize and summarize highlights of a particular subject. The type style and format allow for easier reading. Finally, the publication of a pathology textbook in soft cover is a welcome change, certainly an answer to the worsening problem of book price and bulkiness.

But, there are deficiencies in this book. Typographical errors (page xi) and editing oversights are present. It is difficult to comprehend the following statement (page 215); "Minor degrees of heart block can only be detected on an electrocardiogram or as a slow pulse (breathing, for example, at half the atrial rate)." Also, there must be something mathematically wrong with this statement (page 34); "There are 46 chromosomes comprising one pair of sex chromosomes and 21 pairs of autosomes." Some unfortunate omissions are also evident. There is not a single diagram to illustrate the hemodynamics of congenital heart diseases, and no chapters on environmental pathology and pathology of the pediatric age group.

This book is certainly not a substitute for the Robbins and Cotran textbook. However, for purposes of introductory reading or quick review of pathology, this book is recommended to pathologists, house staff physicians, and students.

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FAOCPA, FACOS
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Diabetes: The GlucograF™ method for normalizing blood sugar

By Richard K. Berstein. Pp. 298, with illus. Crown Publishers, Inc., One Park Avenue, New York 10016, 1981. $14.95.

Surpassed only by cardiovascular diseases and cancer, diabetes is the third leading cause of death in

continued on page **439**/149

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It's going to help ruin a lot of appetites

- **Prelu-2,** as a short-term adjunct to caloric restriction, provides effective anorexia, with simple once-a-day dosage.
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Some points to discuss:

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Obesity

Timed release capsules 105 mg

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Contraindications: Advanced arteriosclerosis, symptomatic cardiovascular disease, moderate to severe hypertension, hyperthyroidism, known hypersensitivity, or idiosyncrasy to the sympathomimetic amines, glaucoma.

Agitated states.

Patients with a history of drug abuse.

During or within 14 days following the administration of monoamine oxidase inhibitors (hypertensive crises may result).

Warnings: Tolerance to the anorectic effect usually develops within a few weeks. When this occurs, the recommended dose should not be exceeded in an attempt to increase the effect; rather, the drug should be discontinued.

Phendimetrazine tartrate may impair the ability of the patient to engage in potentially hazardous activities such as operating machinery or driving a motor vehicle; the patient should therefore be cautioned accordingly.

Drug Dependence: Phendimetrazine tartrate is related chemically and pharmacologically to the amphetamines. Amphetamines and related stimulant drugs have been extensively abused, and the possibility of abuse of phendimetrazine tartrate should be kept in mind when evaluating the desirability of including a drug as part of a weight reduction program. Abuse of amphetamines and related drugs may be associated with intense psychological dependence and severe social dysfunction. There are reports of patients who have increased the dosage to many times that recommended. Abrupt cessation following prolonged high dosage administration results in extreme fatigue and mental depression; changes are also noted on the sleep EEG. Manifestations of chronic intoxication with anorectic drugs include severe dermatoses, marked insomnia, irritability, hyperactivity, and personality changes. The most severe manifestation of chronic intoxications is psychosis, often clinically indistinguishable from schizophrenia.

Usage in Pregnancy: The safety of phendimetrazine tartrate in pregnancy and lactation has not been established. Therefore phendimetrazine tartrate should not be taken by women who are or may become pregnant.

Usage in Children: Phendimetrazine tartrate is not recommended for use in children under 12 years of age.

Precautions: Caution is to be exercised in prescribing phendimetrazine tartrate for patients with even mild hypertension.

Insulin requirements in diabetes mellitus may be altered in association with the use of phendimetrazine tartrate and the concomitant dietary regimen.

Phendimetrazine tartrate may decrease the hypotensive effect of guanethidine.

The least amount feasible should be prescribed or dispensed at one time in order to minimize the possibility of overdosage.

Adverse Reactions:

Cardiovascular: Palpitation, tachycardia, elevation of blood pressure.

Central Nervous System: Overstimulation, restlessness, dizziness, insomnia, euphoria, dysphoria, tremor, headache; rarely psychotic episodes at recommended doses.

Gastrointestinal: Dryness of the mouth, unpleasant taste, diarrhea, constipation, other gastrointestinal disturbances.

Allergic: Urticaria.

Endocrine: Impotence, changes in libido.

Overdosage: Manifestations of acute overdosage with phendimetrazine tartrate include restlessness, tremor, hyperreflexia, rapid respiration, confusion, assaultiveness, hallucinations, panic states.

Fatigue and depression usually follow the central stimulation.

Cardiovascular effects include arrhythmias, hypertension or hypotension and circulatory collapse. Gastrointestinal symptoms include nausea, vomiting, diarrhea, and abdominal cramps. Fatal poisoning usually terminates in convulsions and coma. Management of acute phendimetrazine tartrate intoxication is largely symptomatic and includes lavage and sedation with a barbiturate. Experience with hemodialysis or peritoneal dialysis is inadequate to permit recommendation in this regard. Acidification of the urine increases phendimetrazine tartrate excretion. Intravenous phentolamine (Regitine) has been suggested for possible acute, severe hypertension, if this complicates phendimetrazine tartrate overdosage.

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For complete details, please see the full prescribing information.

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America. Life span from the time of onset of diabetes is decreased by 30 percent for the average patient. The life span of the insulin-dependent diabetic is considerably less. Diabetes, as the author points out, is the leading cause of new cases of blindness and a major cause of kidney disease, neurologic disorders, cardiovascular morbidity, impotence, and nonaccidental amputation of limbs, to list only some of the major complications.

Mr. Bernstein considers diabetes and its complications a tragedy that might be avoided if both the patient and the physician understood the necessity of normalizing blood glucose levels. Early in the book, he attempts to explode the popular misconception that control of the blood glucose level is not important. Himself a diabetic for 34 years, the author approaches this controversial question with missionary zeal.

Before describing the GlucograF™ method, Mr. Bernstein reviews the different types of diabetes mellitus and the roles of certain hormones in regulating the blood glucose level. The review is clear and concise; it is written in a fashion that can be understood by the diabetic patient, as well as his or her physician adviser. This is important because in the GlucograF™ method the patient is in charge of the disease. In this method, the physician's role is that of adviser, not supervisor.

A whole chapter in this book is dedicated to testimonials from patients who are living with the GlucograF™ method. These testimonials show that the method can work and may very well improve the quality of life, as well as prolong it with limited complications.

The remainder of the book is dedicated to the mechanics of the GlucograF™ method and their application. Basically, the method involves having the patient perform serial blood sugar determinations with a portable analyzer and use these results to titrate his or her own insulin dose. To do this, the patient may use several different varieties of insulin in combination. This book gives an excellent review of these different varieties and their duration of action. It also explains why it is useless to follow the urine glucose values.

This book will be of interest to the diabetic patient and the physician who treats diabetics. I intend to refer to it often.

MICHAEL R. OLDEN, D.O.
Olympia Fields, Illinois

Books received

New books received by the Andrew Taylor Still Memorial Library are acknowledged below. Those of greatest interest to readers will be reviewed later.

Noninvasive assessment of the cardiovascular system: Diagnostic principles and techniques. Edited by Edward B. Diethrich; pp. 432, with illus.; John Wright•PSG Inc, Littleton, Mass., 1982, $39.50.

Segmental anatomy: Applications to clinical medicine. (Reference guide.) By Marvin Wagner and Thomas L. Lawson; pp. 650, with illus.; Macmillan Publishing Co., Inc., New York, 1982, $95.00.

Posterior lumbar interbody fusion. (History, principles, indications, and techniques are discussed.) Edited by Paul M. Lin; pp. 308, with illus.; Charles C Thomas, Publisher, Springfield, Ill., 1982, $44.50.

Foot and ankle pain. (Functional anatomy and treatment of foot and ankle disorders.) By Rene Cailliet; pain series; ed. 2, pp. 200, with illus.; F.A. Davis Co., Philadelphia, 1982, paperbound, $11.95.

Lecture notes on clinical pharmacology. (Brief, up-to-date review. For students and practicing clinicians.) By John L. Reid, Peter C. Rubin, and Brian Whiting; pp. 365, with illus.; Blackwell Scientific Publications, distributed by Blackwell Mosby Book Distributors, St. Louis, 1982, paperbound, $14.95.

Lithium and animal behavior. (Reference source on the effects of lithium on humans through experimental studies on animals. For professionals in psychopharmacology, psychology, psychiatry, toxicology, neuropsychology, and the behavioral sciences.) By Donald F. Smith, edited by David F. Horrobin; lithium re-

search review series; vol. 2, pp. 134; Human Sciences Press, Inc., New York, 1982, $16.95.

Manual of hemostasis and thrombosis. (Pathophysiologic approach to diagnosis and management, for students, house officers, and clinicians.) By Arthur R. Thompson and Laurence A. Harker; ed. 3, pp. 219, with illus.; F.A. Davis Co., Philadelphia, 1982, paperbound, $8.95.

Current literature review in obstetrics & gynecology, 1982. (Multiple choice questions with answers based on significant articles from major obstetrics and gynecology journals in the United States, as well as a few general medical journals, during the calendar year 1981. For students and practitioners.) By Richard M. Lackritz; current literature review series; pp. 157; Appleton-Century-Crofts, Norwalk, Conn., 1982, paperbound, $18.95.

Update: Dermatology in general medicine. By Thomas B. Fitzpatrick, et al.; ed. 2, pp. 237, with illus.; McGraw-Hill Book Co., New York, 1983, $40.00.

Handbook of poisoning: Prevention, diagnosis & treatment. By Robert H. Dreisbach; ed. 9, pp. 632, with illus.; Lange Medical Publications, Los Altos, Calif., 1983, pocketsize, $11.00.

The D.O.'s: Osteopathic medicine in America. (In-depth study of the profession by a sociologist.) By Norman Gevitz; pp. 183, with illus.; The Johns Hopkins University Press, Baltimore, 1982, $18.50.

Practical gastrointestinal endoscopy. (Revised and updated methods and techniques.) By Peter B. Cotton and Christopher B. Williams; ed. 2, pp. 203, with illus., Blackwell Scientific Publications, distributed by Blackwell Mosby Book Distributors, St. Louis, 1982, $34.50.

Essential surgical practice. (Up-to-date reference on the pathophysiology, clinical features, and management of disorders encountered in surgical practice. For surgeons.) Edited by A. Cuschieri, G.R. Giles, and A.R. Moossa; pp. 1,296, with illus., John Wright•PSG Inc, Littleton, Mass., 1982, $65.00.

The foot and its disorders. (Updated and expanded reference source for clinicians, orthopedists, and surgeons.) Edited by Leslie Klenerman; ed. 2, pp. 460, with illus.; Blackwell Scientific Publications, distributed by Blackwell Mosby Book Distributors, St. Louis, 1982, $65.00.

Evaluating residency training. (For clinical teachers, administrators, and residents.) By John B. Corley; ed. 2, pp. 300, with illus.; The Collamore Press, D.C. Heath Co., Lexington, Mass., 1983, $23.95.

A synopsis of anaesthesia: General anaesthesia; regional analgesia; intensive

Currently available antianginal agents...
and how they are most appropriately employed as

BASIC THERAPY FOR THE PREVENTION OF ANGINA PECTORIS*

Report from "Step-Care Therapy for Ischemic Heart Disease"
—a special symposium preceding the annual meeting
of the American College of Cardiology, April 24, 1982.

Synopsis of the Symposium

Step 1: Nitrates

Since nitrates both reduce myocardial oxygen demand and increase myocardial oxygen supply, their use is warranted as initial therapy for the control of angina pectoris. Dosage needs often vary significantly from patient to patient, and attention should be given to ensure that an adequate dosage level has been established.

Step 2: Nitrates and beta blockers

For angina refractory to maximum-dose nitrate therapy, the complementary actions of beta blockers and nitrates provide a rational therapeutic modality. At this point a beta blocker should be added to the nitrate regimen.

Step 3: Nitrates and calcium antagonists

Calcium antagonists have special usefulness in controlling cases where the myocardial oxygen supply is diminished by coronary vasospasm; their use in combination with nitrates is recommended if Step 2 therapy is unsatisfactory (or if the patient has variant or vasospastic angina).

Consensus of the Symposium

Nitrates are basic in step-care therapy for ischemic heart disease

Step 1
Stable or unstable angina: Nitrates

Step 2
If not controlled: Nitrates + beta blockers

Step 3
If still not controlled (or in variant angina): Nitrates + calcium antagonists

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The complete Proceedings of the Symposium are available on request from the Professional Services Department of Ives Laboratories.

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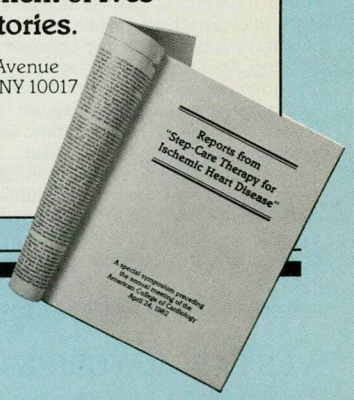

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therapy. By R.S. Atkinson, G.B. Rushman, and J. Alfred Lee; ed. 9, pp. 976, with illus.; John Wright•PSG Inc, Littleton, Mass., 1982, paperbound, $33.50.

A radiographic index. (Revised and updated brief reference guide.) By Myer Goldman and David Cope; ed. 7, pp. 99; John Wright•PSG Inc, Littleton, Mass., 1982, pocket-size, $10.00.

An introduction to neurophysiology. By J.F. Stein; pp. 386, with illus.; Blackwell Scientific Publications, distributed by Blackwell Mosby Book Distributors, St. Louis, 1982, paperbound, $24.95.

Enteral and parenteral nutrition: A clinical handbook. (A broad approach to the nutritional therapy of vulnerable patients for all health professionals concerned with patient care.) Edited by Andrew Grant and Elizabeth Todd; pp. 175, with illus.; Blackwell Scientific Publications, distributed by Blackwell Mosby Book Distributors, St. Louis, 1982, paperbound, $9.50.

Occupational therapy for physical dysfunction. (Evaluation and treatment of physical dysfunctions with theoretical bases of these treatment procedures. For students and experienced therapists.) Edited by Catherine Anne Trombly; ed. 2, pp. 512, with illus.; Williams & Wilkins Co., Baltimore, 1982, paperbound, $29.00.

Diabetes mellitus and obesity. Edited by Bernard N. Brodoff and Sheldon J. Bleicher; pp. 816, with illus.; Williams & Wilkins Co., Baltimore, 1982, $60.00.

Dictionary of rehabilitation medicine. By Herman L. Kamenetz; pp. 368; Springer Publishing Co., New York, 1982, $21.95.

Drug-induced ocular side effects and drug interactions. (Reference source for ophthalmologists and clinicians.) By F.T. Fraunfelder; ed. 2, pp. 544; Lea & Febiger, Philadelphia, 1982, $30.00.

Basic & clinical pharmacology. (Text for students.) Edited by Bertram G. Katzung; pp. 814, with illus.; Lange Medical Publications, Los Altos, Calif., 1982, paperbound, $23.50.

Epidemiology of diseases. (Concise account of important contemporary diseases.) Edited by D.L. Miller and H.D.T. Farmer, pp. 492, with illus.; Blackwell Scientific Publications, distributed by Blackwell Mosby Book Distributors, St. Louis, 1982, $57.50.

Multiple choice questions on lecture notes on clinical medicine. (Self-assessment questionnaire for students.) By David Rubenstein and David Wayne; ed. 2, pp. 70; Blackwell Scientific Publications, distributed by Blackwell Mosby Book Distributors, St. Louis, 1982, paperbound, $5.95.

How to write and publish papers in the medical sciences. (Step-by-step guide for beginners and established authors.) By Edward J. Huth; professional writing series; pp. 203; ISI Press, Philadelphia, 1982, $17.95.

applications for membership

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