

MCV/Q

MEDICAL COLLEGE OF VIRGINIA QUARTERLY
VOLUME ELEVEN • NUMBER FOUR • 1975

NEW DIMENSIONS OF PROBLEMS IN OFFICE PRACTICE

TOMPKINS-McCAW
LIBRARY

FEB 20 1976

C. U.
of VA.
23219

HOW MUCH ANXIETY

"There is a common tendency in our day, both on the part of professional psychologists and laymen, to look upon anxiety as a negative, destructive, "abnormal" experience, one which must be fought and if possible annihilated...."

O. H. Mowrer¹

Since 1950 the literature on anxiety, both professional and lay, has increased a thousandfold in the form of articles, symposia, reports and scientific exhibits. And virtually all of this output reflects a common presumption—that anxiety is a negative, nonproductive experience. This viewpoint leads naturally to a discussion of how to combat or eliminate anxiety.

But anxiety, as Mowrer implies, has its uses. It can play a positive and constructive role in human development. Without it neither an individual nor a society can grow.

Productive vs. nonproductive anxiety: a matter of degree

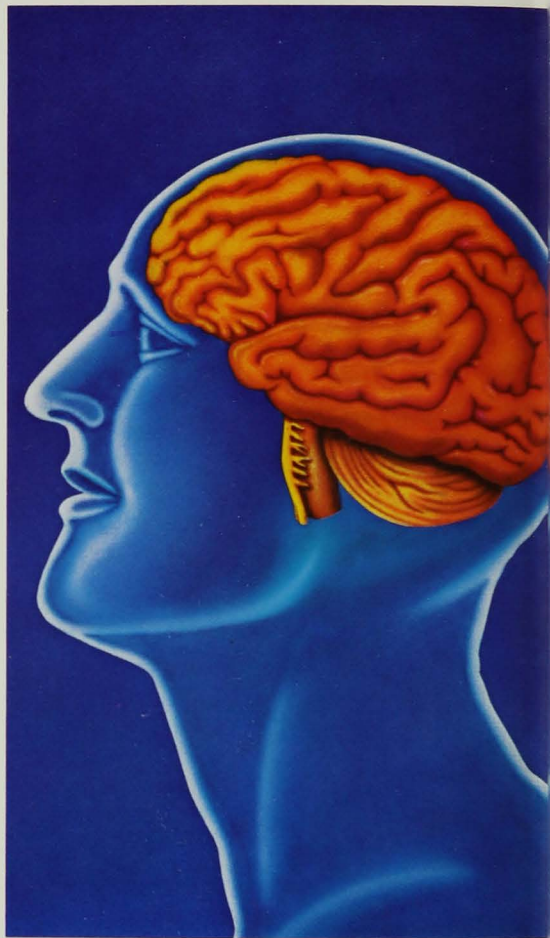
For the physician the difference is not an academic one. He *must* distinguish between productive and nonproductive anxiety. And the difference is often one of degree.

In low levels of anxiety, for example, the individual is alert and sensitive to threats and acquires an increased ability to cope. Performance is often improved.²

But at higher levels of anxiety the opposite is true.² The ability to distinguish between the dangerous and the trivial is reduced and often leads to inappropriate behavior. Apprehension becomes fear. And coping becomes difficult, if not impossible.

Crossing the anxiety threshold

The key question for the physician then becomes: Is the degree of anxiety experienced produc-



tive or nonproductive for the individual patient? And while some patients may require relatively large amounts of anxiety to perform optimally, for others lower levels of anxiety may prove unproductive.

Librium® (chlordiazepoxide HCl): to help lower the level of anxiety

When anxiety has reached levels that serious

HOW MUCH ANXIETY'S PRODUCTIVE?

"There is a common tendency in our day, both on the part of professional psychologists and laymen, to look upon anxiety as a negative, destructive, 'abnormal' experience, one which must be fought and if possible annihilated...."

O. H. Mowrer¹

Since 1950 the literature on anxiety, both professional and lay, has increased a thousandfold in the form of articles, symposia, reports and scientific exhibits. And virtually all of this output reflects a common presumption — that anxiety is a negative, nonproductive experience. This viewpoint leads naturally to a discussion of how to combat or eliminate anxiety.

But anxiety, as Mowrer implies, has its uses. It can play a positive and constructive role in human development. Without it neither an individual nor a society can grow.

Productive vs. nonproductive anxiety: a matter of degree

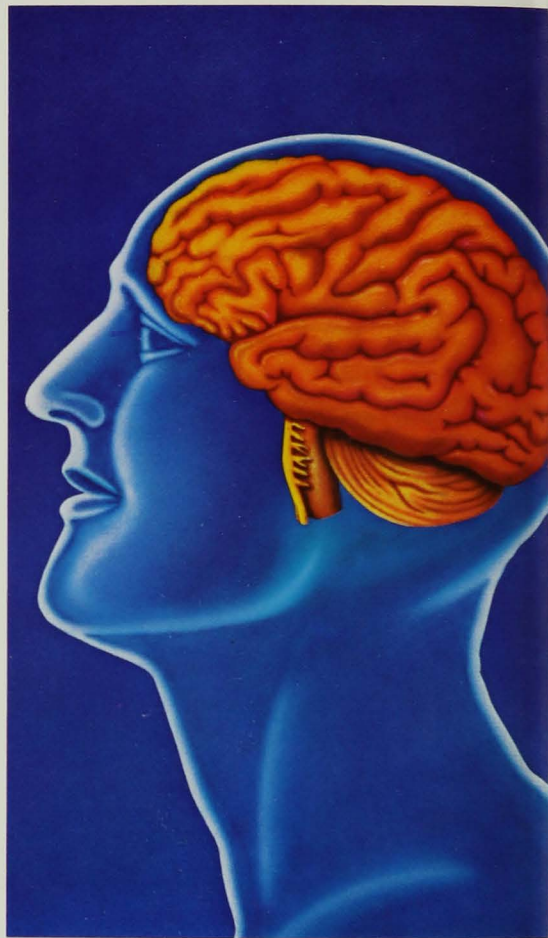
For the physician the difference is not an academic one. He must distinguish between productive and nonproductive anxiety. And the difference is often one of degree.

In low levels of anxiety, for example, the individual is alert and sensitive to threats and acquires an increased ability to cope. Performance is often improved.²

But at higher levels of anxiety the opposite is true.² The ability to distinguish between the dangerous and the trivial is reduced and often leads to inappropriate behavior. Apprehension becomes fear. And coping becomes difficult, if not impossible.

Crossing the anxiety threshold

The key question for the physician then becomes: Is the degree of anxiety experienced produc-



tive or nonproductive for the individual patient? And while some patients may require relatively large amounts of anxiety to perform optimally, for others lower levels of anxiety may prove unproductive.

Librium® (chlordiazepoxide HCl): to help lower the level of anxiety

When anxiety has reached levels that seriously

impair performance, reassurance and counseling may be sufficient for the patient. If not, adjunctive antianxiety medication may be called for.

Librium (chlordiazepoxide HCl), by quickly and effectively calming the anxious patient, helps to lower the level of anxiety. When anxiety has been reduced to manageable levels, therapy with Librium should be discontinued.

Librium® (chlordiazepoxide HCl): an uncomplicated clinical course

To be truly effective, antianxiety medication must allay anxiety without complicating the clinical course. Librium meets this criterion. Librium, when used in proper dosage, rarely interferes with mental acuity. Side effects are seldom encountered. And Librium is used concomitantly with many primary medications.

For a more detailed discussion of the side effects, precautions and warnings, please consult the brief summary of product information on this page.

References: 1. Mowrer OH, quoted in May R: *The Meaning of Anxiety*. New York, Ronald Press Co., 1950, pp. 108 ff. 2. Basowitz H et al: *Anxiety and Stress*. New York, McGraw-Hill, 1955, pp. 12 ff.

LIBRIUM®
chlordiazepoxide HCl / Roche®
5 mg, 10 mg, 25 mg capsules

BASIC IN CLINICAL ANXIETY

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Relief of anxiety and tension occurring alone or accompanying various disease states.

Contraindications: Patients with known hypersensitivity to the drug.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. As with all CNS-acting drugs, caution patients against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving). Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported. Use of any drug in pregnancy, lactation or in women of childbearing age requires that its potential benefits be weighed against its possible hazards.

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (e.g., excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido — all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

Supplied: Librium® Capsules containing 5 mg, 10 mg or 25 mg chlordiazepoxide HCl. Libritabs® Tablets containing 5 mg, 10 mg or 25 mg chlordiazepoxide.



Roche Laboratories
Division of Hoffmann-La Roche Inc.
Nutley, New Jersey 07110

S PRODUCTIVE?

impair performance, reassurance and counseling may be sufficient for the patient. If not, adjunctive antianxiety medication may be called for.

Librium (chlordiazepoxide HCl), by quickly and effectively calming the anxious patient, helps to lower the level of anxiety. When anxiety has been reduced to manageable levels, therapy with Librium should be discontinued.

Librium® (chlordiazepoxide HCl): an uncomplicated clinical course

To be truly effective, antianxiety medication must allay anxiety without complicating the clinical course. Librium meets this criterion. Librium, when used in proper dosage, rarely interferes with mental acuity. Side effects are seldom encountered. And Librium is used concomitantly with many primary medications.

For a more detailed discussion of the side effects, precautions and warnings, please consult the brief summary of product information on this page.

References: 1. Mowrer OH, quoted in May R: *The Meaning of Anxiety*. New York, Ronald Press Co., 1950, pp. 108 ff. 2. Basowitz H et al: *Anxiety and Stress*. New York, McGraw-Hill, 1955, pp. 12 ff.

LIBRIUM®
chlordiazepoxide HCl / Roche®
5 mg, 10 mg, 25 mg capsules

BASIC IN CLINICAL ANXIETY

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Relief of anxiety and tension occurring alone or accompanying various disease states.

Contraindications: Patients with known hypersensitivity to the drug.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. As with all CNS-acting drugs, caution patients against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving). Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported. Use of any drug in pregnancy, lactation or in women of childbearing age requires that its potential benefits be weighed against its possible hazards.

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (e.g., excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

Supplied: Librium® Capsules containing 5 mg, 10 mg or 25 mg chlordiazepoxide HCl. Libritab® Tablets containing 5 mg, 10 mg or 25 mg chlordiazepoxide.



Roche Laboratories
Division of Hoffmann-La Roche Inc.
Nutley, New Jersey 07110

MCV/Q

MEDICAL COLLEGE OF VIRGINIA QUARTERLY

*A Scientific Publication of the School of Medicine
Health Sciences Division of Virginia Commonwealth University*

1975 • Volume Eleven • Number Four

MEDICAL COLLEGE OF VIRGINIA QUARTERLY Published quarterly (Spring, Summer, Fall, Winter), by the Medical College of Virginia, Division of Health Sciences, Virginia Commonwealth University. The QUARTERLY publishes results of original research in basic and clinical sciences. Contributions from outside the Medical College of Virginia faculty are invited. Manuscripts, submitted in duplicate, should be prepared according to recommendations in the Style Manual for Biological Journals, Washington, D.C., American Institute of Biological Sciences, Second Edition, 1964.

Correspondence: MEDICAL COLLEGE OF VIRGINIA QUARTERLY, Medical College of Virginia, Richmond, Virginia 23298. Phone 804/770-4027.

Subscription rates for U.S.A., Canada, and Mexico: 1 year, \$6.00; 2 years, \$10.00; 3 years, \$14.00. All other countries: 1 year, \$8.00; 2 years, \$12.00; 3 years, \$15.00. Libraries and institutions: (U.S.A.) 1 year, \$12.00; 2 years, \$20.00; 3 years, \$28.00; (*foreign*) 1 year, \$13.00; 2 years, \$21.00; 3 years, \$29.00. Interns, residents, and students: 1 year, \$3.00. Single issue: \$2.00.

Third class postage paid at Richmond, Virginia.

Editorial Advisory Board

John T. Farrar
Hunter H. McGuire
M. Pinson Neal, Jr.
Kinloch Nelson
Frederick J. Spencer

Editorial Consultants

Larry F. Cavazos *Boston*
Richard G. Lester *Durham*
Sami I. Said *Dallas*
Malcolm E. Turner, Jr. *Birmingham*

Editor

Fairfield Goodale, Jr.

Managing Editor

Mary-Parke Johnson

Cover Design

Raymond A. Geary

CONTENTS

THE 28TH ANNUAL STONEBURNER LECTURE SERIES *New Dimensions of Problems in Office Practice*

Sponsored by the School of Medicine, Department of Continuing Education and the Department of Family Practice, Medical College of Virginia, Health Sciences Division of Virginia Commonwealth University.

FITZHUGH MAYO, M.D., *Guest Editor*

Introduction	154
FITZHUGH MAYO, M.D.	

Differential Diagnosis of Hypertension	155
W. ANDREW DICKINSON, JR., M.D.	

Practical Office Therapy of Hypertension	159
A. JARRELL RAPER, M.D.	

Some Practical Thoughts on Medical Malpractice	164
ROBERT L. HARRIS, J.D.	

Asthma in Adults	170
JAMES P. BAKER, M.D.	

Sprains and Strains	174
ROBERT W. WADDELL, M.D.	

SCRIPTA MEDICA

The Birth of a New Department at MCV/VCU— Human Genetics	187
PETER MAMUNES, M.D.	

TABLE OF CONTENTS FOR VOLUME ELEVEN	189
-------------------------------------	-----

INDEX TO VOLUME ELEVEN	191
------------------------	-----

INTRODUCTION

Since the awakening of the public and the medical educational establishment to the growing deficit of appropriately trained physicians to deliver primary care in the community, attention is now turned to the provision of educational programs appropriate for such physicians at the graduate and continuing education levels.

In the past, the inability to measure outcomes of patient care systems (that is, to use the scientific method in community settings) has hampered the union of the educational system with that of patient care. Many studies now are showing that outcomes are profoundly affected by communication and compliance between physician and patient. A partnership of the expertise of the medical center with the laboratory of the practice in the community equipped to measure the process of delivery, as well as outcomes, appears essential.

Such practices are now available within the MCV-VCU Family Practice residency training system, and the 1975 Stoneburner Lectures were dedicated to bringing together consultant faculty from the medical center and the community as well as faculty from the family practice centers. High-volume problems seen by primary care physicians were discussed, and the latest scientific information concerning such problems as hypertension, asthma, sprains and strains was related to the realities of patient care in community settings.

Programs such as the Stoneburner Lectures represent only a milestone in the ongoing marriage of education and patient care. Projects linking tertiary and secondary care to primary care systems will allow the natural history of early disease to be studied and preventive measures to be evaluated.

My thanks to our faculty from all levels who participated in these lectures and to the staff of the *MCV Quarterly*.

FITZHUGH MAYO, M.D.
Chairman
Department of Family Practice

Differential Diagnosis of Hypertension

W. ANDREW DICKINSON, JR., M.D.

Internal Medicine and Cardiology, Virginia Beach, Virginia

Accepting that hypertension occurs in an estimated 20% of the adult population in the United States (1) and that it is a leading risk factor in the development of vascular disease (2, 3), physicians are being challenged to assume a more pragmatic approach to the differential diagnosis of hypertension than has been promoted in the past.

Detection. The levels of blood pressure defining hypertension vary, but 140/90 in patients under age 40 and 160/95 in patients over age 40, are generally accepted (1). Such elevated recordings in the office, screening program, or upon admission to the hospital should be followed by three successive recordings made by the physician or trained nursing personnel to definitely determine that the patient has hypertension and that the original reading was not falsely elevated because of emotional factors surrounding the initial examination. Bed rest for hospitalized patients will yield normal blood pressure recordings, and an initial recording which is elevated should be followed by repeated recordings after the patient has returned to normal daily living.

Recording of the blood pressure, though simple, should be properly performed to avoid categorizing a patient in a group already challenging the system's ability to evaluate and treat those in need. With the use of two sets of stethophones connected to a single diaphragm, it is quite easy to instruct office personnel in proper auscultation of the blood pressure. When the mercury manometer is used, the meniscus should be at point 'O' when deflated, and the mercury column should fall freely with a good air flow into the top of the manometer. Aneroid manometers

should be standardized with a Y-tubing against a mercury manometer for accuracy. The bladder of the blood pressure cuff should be placed over the brachial artery, and the bell or diaphragm of the stethophone over the pulsating vessel in the antecubital fossa. The patient should be resting for at least five minutes and the blood pressure initially recorded in the supine or sitting position. The cuff should be inflated until the radial pulse can no longer be felt and then deflated at approximately 2 to 3 mm/sec. The initial sound (phase I) is recorded as the systolic pressure, with the point of disappearance (phase V) coinciding best with intra-arterial diastolic pressure (4). Where phase V is over 5 mm lower than the point of muffling (phase IV), both numbers should be recorded. Patients with bruits in their arms due to partial stenosis of the artery, as well as those with aortic insufficiency and hyperkinetic circulations from other causes, may not have disappearance of Korotkoff's sounds until near 'O'. In these instances, phase IV, or muffling, is used as the diastolic pressure.

Recordings should be made in both arms, where up to 10 mm Hg difference in pressure may be normally noted; greater differences should suggest the possibility of some intra-arterial obstruction to that extremity. In patients who are markedly obese, a thigh cuff should be used in the upper arm and may be more accurate than recording the blood pressure in the forearm. Pumping the cuff up rapidly, keeping the recording arm elevated, as well as allowing 15 seconds or more between recordings, will prevent venous engorgement, which might affect the arterial pressure.

In addition to blood pressure determinations lying and sitting, the blood pressure should be recorded

Presented by Dr. Dickinson at the 28th Annual Stoneburner Lecture Series, 10 April, 1975, at the Medical College of Virginia, Richmond.

immediately after the patient assumes the standing position and one minute after. The degree of postural drop in blood pressure may be marked in lean patients, particularly in the older age group, and greatly influences not only the patient's further evaluation but also the selection of therapy.

Etiology. In the past, an estimated 10% to 15% or more of hypertensives have been defined as having curable causes, though Gifford (5) found that less than 6% of 5,000 hypertensive patients at the Cleveland Clinic have potentially curable forms of hypertension. In the average primary medical practice, the figure for curable hypertension may be even less, perhaps 2%; that is, over 98% of patients may have hypertension which is either essential or due to renal parenchymal disease.

Essential Hypertension. In essential hypertension, the age at which hypertension was first detected, or when normal blood pressure was last documented, should be sought. Essential hypertension usually begins at the third and fourth decade of life and progresses through the years. A family history of hypertension, or vascular complications related to hypertension, is often obtained. The Systems Review should be directed at detecting evidence of organ damage such as cerebrovascular insufficiency, vision impairment, arteriosclerotic heart disease, as well as claudication. Most patients will either be entirely asymptomatic or present with manifestations of target organ dysfunction as the main complaint.

An ophthalmoscope, which can be carried in the breast pocket, facilitates funduscopy, and most patients will be found to have normal fundi or perhaps grade I changes due to arteriolosclerosis manifested by an increased light reflex ("copper wire changes"), with occasionally more advanced changes of A-V nicking and compression, all of which relate more to the duration of hypertension, rather than to its severity. Rarely, one will see retinal hemorrhages, exudates, and/or papilledema in the patient with accelerated, "malignant" hypertension. Bruits over the carotid arteries indicate the likelihood of atherosclerotic plaques in these vessels and suggest not only the degree of atherosclerosis present, but should also alert the physician to possible side effects with future drug therapy which might cause postural hypotension and neurological complications. A diffuse left ventricular impulse over the precordium suggests left ventricular hypertrophy due to hypertension, or dilatation as a result of a dyskinetic area of the left ventricle affected by ischemic heart disease.

An S-4 gallop sound is heard in patients with more severe hypertension. Palpation of the abdominal aorta for an aneurysm, as well as the pulses in the lower extremities, add further to the evaluation of the total vascular picture.

Laboratory studies should include a CBC and an SMA-12, or at least a cholesterol, 2 hr. p.c. sugar, 16 hr. fasting triglyceride level, and a urinalysis. A baseline electrocardiogram and P-A radiograph of the chest for evidence of left ventricular hypertrophy or enlargement should be obtained and used for comparison with future studies.

Chronic Renal Disease. Proteinuria, particularly when accompanying or preceding the onset of hypertension by history, or when associated with a history of glomerulonephritis or repeated pyelonephritis, suggests primary renal disease as the cause for hypertension. An abnormal urinary sediment including white blood cells, red blood cells, and/or casts further suggests such an etiology. An elevated creatinine, with intravenous pyelogram (IVP) demonstration of kidneys which are contracted, polycystic, or with typical changes of chronic infection may also serve to document this etiology.

Renal Artery Stenosis. Of the curable forms of hypertension, renal artery stenosis probably represents the larger group, though it still represents a small number of patients in the average practice. The abrupt onset of hypertension, particularly in the age groups below 40, and particularly in females, more likely suggests fibromuscular disease of a renal artery. The rather abrupt onset of hypertension in older age groups that were previously normotensive also suggests a stenotic lesion, but one due to atherosclerosis. Hypertensive patients under therapy who are increasingly difficult to control might also be suspected of having renal artery stenosis. The history, except in regard to the progression of the hypertension itself, is usually unrevealing. It should be noted that renal artery stenosis is not often seen in blacks. The presence of vascular spasm on funduscopy suggests a more acute onset of hypertension.

A rapid sequence IVP revealing delayed appearance of contrast media by one kidney suggests that it is inadequately perfused. A difference in kidney size is significant if the left kidney is more than 1.5 cm shorter than the right, or if the right kidney is 2.0 cm or more shorter than the left, the right kidney being normally shorter than the left. Aortography should be performed if pyelography suggests unilateral renal disease, and even when the IVP is normal, if the clinical

picture is strongly suggestive, since there are significant numbers of false-negative IVP's. On physical examination, systolic bruits in the epigastrium are not sufficiently reliable in the diagnosis of renal artery stenosis, though a systolic murmur with a diastolic component would be strongly suggestive. The demonstration of stenosis in a renal vessel does not mean that such a kidney is inadequately perfused, or that inadequate perfusion is the cause of the patient's hypertension, since renal artery stenosis may exist in normotensive patients.

Plasma renin vein assays may also be normal or elevated. It is, therefore, necessary to obtain renal vein renin ratios by catheterization of each of the renal veins. Following a period of salt deprivation, and three hours after 80 mg of furosemide by mouth, if the renin level from the suspected kidney is 1.5 to 2 times that of the anatomically normal kidney, and particularly if the contra-lateral kidney renin level is lower than the peripheral plasma renin, then the suspected kidney is probably contributing to the hypertension. Further confirmation is available by the administration of an analog of angiotensin II, the potent vasopressor responsible for the hypertension in renal artery stenosis, that can be used to block angiotensin II. When the analog produces a clear-cut lowering of blood pressure when given intravenously, this would further support the suspicion that renal artery stenosis is causing the hypertension (6). This analog is saralasin acetate and is still under investigation for general use.

Primary Aldosteronism. An even more rare cause of hypertension is primary aldosteronism. Such patients may present with a history of weakness, muscle cramps, and paresthesias, but will more likely be detected by the finding of hypokalemia. When such is documented on repeated determinations of the serum potassium in a patient who has not been on diuretics or a low-salt diet or who does not have other clinical causes for hypokalemia, such as diarrhea, vomiting, and so forth, then a 24-hour urine sample for total potassium should be obtained. If the patient has been off diuretics for at least three days and the potassium excretion exceeds 30 to 40 mEq in 24 hours, then excessive aldosterone effect must be considered. Such patients must be on an adequate salt intake diet before urinary measurements of potassium are made, as well as the analyses of plasma renin and aldosterone levels, which will be needed to further confirm this diagnosis. High levels of aldosterone, accompanied by abnormally low levels of renin,

are diagnostic of this disease, but do not determine whether such are due to an isolated adenoma of the adrenal cortex or to bilateral nodular hyperplasia. Visualization of the adrenal glands prior to surgery is recommended since patients with bilateral adrenal hyperplasia present a more serious problem for surgical, as well as subsequent endocrine management, and such patients usually respond quite well to medical management with spironolactone.

Pheochromocytoma. Most patients with pheochromocytoma will be symptomatic with fluctuating blood pressure due to the intermittent discharge of catecholamines. Headaches, nervousness, palpitations, and sweating are the usual symptoms. These patients are rarely overweight, and the presence of obesity would make the diagnosis unlikely. Laboratory studies are usually not necessary to exclude this diagnosis, but if suspected, a metanephrine screening test of the urine should be obtained. If this is positive, free catecholamines can be measured in a 24-hour urine sample. Drugs and dietary factors, which complicate measurement of VMA, make this test of limited value.

Cushing's Disease. The typical Cushingoid features, together with evidence of carbohydrate intolerance, make this diagnosis one to be considered, though the overwhelming majority of such patients suspected on this basis, will turn out not to have the disease. Measurement of plasma cortisol on a 7 AM specimen, after administration of 1.0 mg dexamethasone at bedtime the night before, if suppressed below normal values, all but excludes completely such a diagnosis.

Coarctation of the Aorta. Simple palpation of the pulses in the lower extremities and comparing their volume and time of onset with that in the radial or carotid pulse is usually all that is necessary to lead one to consider the possibility of coarctation. If there is a pulse lag and diminished or absent pulses in the legs, then the blood pressure should be recorded, if possible, in the arm and thigh. Normally, the thigh blood pressure exceeds the arm blood pressure and if the opposite is evident, then the diagnosis of coarctation will need to be pursued further.

"The Pill." The use of oral contraceptives may cause reversible hypertension and is probably one of the more common causes of curable hypertension among the others mentioned, in the average population. Discontinuing oral contraceptives will result in the return of blood pressure to normal within several months.

Classification of Hypertensive Patients by Renin

Assay. Renin is produced and released by the juxtaglomerular apparatus in the kidney in response to the pressure in the afferent arteriole and by the sodium level in the macula densa of the kidney. Normal physiological factors which lower renal artery pressure cause increased renin release, whereas factors which increase renal artery pressure cause a decrease in renin levels. Similarly, diminished sodium effect, such as seen in salt depletion, results in increased renin release, whereas increased sodium effect on the blood pressure is associated with suppressed renin levels (7). The compensatory mechanisms for restoration of normal pressure and sodium effect on the blood pressure come about through renin influence on the production of more, or less, angiotensin II, the potent vasopressor circulating agent. Angiotensin II further influences the release of aldosterone from the adrenal cortex which when increased results in increased sodium and fluid retention as a result of its effect on the renal tubule (8).

Plasma renin assay (PURA) is actually a measurement of the amount of angiotensin I (the inactive precursor of angiotensin II) formed from a substrate over a given period of incubation time. Renin levels are expressed as ng/cc/hr of incubation and are commercially available by radioimmunoassay techniques. Such plasma renin activity is compared with those determined to be within the low, normal, or elevated levels for the particular laboratory. Reliable PURA requires that the sodium-volume status of the patient not be disrupted by outside influences such as diets, and so forth, and most of the commonly used hypertensive drugs, as well as oral contraceptives, must be omitted temporarily to obtain diagnostic and unprovoked results. Approximately 25% to 30% of patients with essential hypertension will have low renin levels, whereas a somewhat smaller number will have high renin levels, with a slight majority having normal renin levels (9). Determination of renin levels in patients with essential hypertension has been advanced by some who have studies to indicate that renin may be toxic to the vascular system and should be treated with renin-lowering drugs (10, 11) such as propranolol or methyldopa. Other studies do not confirm this (9). Others have proposed a diagnostic trial of antihypertensive drugs with known effect on renin activity, as a better approach than using the renin levels per se; that is, a propranolol or methyldopa response suggesting increased renin hypertension, whereas diuretic re-

DICKINSON: DIAGNOSIS OF HYPERTENSION

sponsiveness would suggest low or normal renin levels.

Conclusion. The overwhelming majority of hypertensive patients have essential hypertension, which is a potent risk factor in producing vascular disease. Overall, it must be stated that the differential diagnosis of hypertension is a relatively minor factor in approaching the tremendous challenge of detecting and adequately treating the overwhelming majority of patients who are now undetected, untreated, or inadequately treated.

REFERENCES

1. Guidelines for the detection, diagnosis and management of hypertensive populations. Report of Inter-Society Commission for Heart Disease Resources. *Circulation* 44:A263-A272, 1971.
2. Effects of treatment on morbidity in hypertension: Results in patients with diastolic blood pressures averaging 90 through 129 mm/hg. Veterans Administration Cooperative Study Group on Antihypertensive Agents. *JAMA* 202 (pt 2):1028-1034, 1967.
3. Effects of treatment on morbidity in hypertension: II. Results in patients with diastolic blood pressure averaging 90 through 114 mm/hg. Veterans Administration Cooperative Study Group on Antihypertensive Agents *JAMA* 213 (pt 2):1143-1152, 1970.
4. GEDDES LA: *The Direct and Indirect Measurement of Blood Pressure*. Chicago, Yearbook Medical Publishers, 1970.
5. GIFFORD RW JR: Evaluation of the hypertensive patient with emphasis on detecting curable causes. *Milbank MEM Fund Q* 47:170-186, 1969.
6. STREIFEN DHP, ANDERSON GH, FREIBERG JM, ET AL: Angiotensin antagonist in diagnosing angiotensinogenic hypertension. *N Engl J Med* 292:657-662, 1975.
7. DAVIS JO: The control of renin release. *Am J Med* 55:333-350, 1973.
8. LARAGH JH, ANGERS M, KELLEY WG, ET AL: Hypotensive agents and pressor substances: The effect of epinephrine, norepinephrine, angiotensin II, and others on the secretory rate of aldosterone in man. *JAMA* 174:234-240, 1960.
9. KAPLAN NM: The prognostic implications of plasma renin in essential hypertension. *JAMA* 231:167-170, 1975.
10. LARAGH JH: Vasoconstriction-volume analysis for understanding and treating hypertension: The use of renin and aldosterone profiles. *Am J Med* 55:261-274, 1973.
11. GEISE J: Renin, angiotensin and hypertensive vascular damage, A review. *Am J Med* 55:315-332, 1973.

Practical Office Therapy of Hypertension

A. JARRELL RAPER, M.D.

*Associate Professor of Medicine, Medical College of Virginia, Health Sciences
Division of Virginia Commonwealth University, Richmond, Virginia.*

Because of the sheer number of patients and because of their location, the community practitioner is in a position to deal with the vast majority of problems that occur in hypertension. The trend in hypertension therapy is toward individualizing the work-up of the hypertensive patient, which with many patients can be very simple.

When emphasizing aspects of practical office care of the hypertensive, one of the first questions that comes to mind is: what exactly is the blood pressure?

There is not any one universally accepted definition of hypertension. Until such a one is adopted, we have put together a Medical College of Virginia definition, using the opinions of several of us in the Department of Medicine (Fig 1). We use the following criteria:

1. A diastolic of 95 mm Hg. Anything greater than this is hypertension.
2. The seated posture.
3. The diastolic pressure only; not the systolic (for reasons which will be discussed later).
4. Disappearance of all sound (or phase V of Korotkoff's sounds).

Thus, hypertension is defined as any measurement that is above 95 mm Hg diastolic, in the seated posture. *Sustained hypertension* in our definition is three such consecutive readings, or three out of four, on an outpatient basis. This works out well in a practical way, because the first reading can be the "screening" blood pressure, the second one can be obtained

during the first evaluation of the patient, and the third one is taken when the lab work has come back, and the physician is ready to make a decision about the course of treatment. *Transient hypertension* is hypertension that comes and goes, with normal blood pressures in between.

There are certain simple things that should be done, short of long-term therapy, and these will sometimes suffice. For instance, if a patient who is grossly obese can be induced to lose 20% or 25% body weight, often there will be a drop in the blood pressure. We have had very little success with this, unless the patient can be induced to join a group where there is high morale and a real motivation toward lowering body weight. Such a group is far superior to drug therapy. Another problem to be considered when dealing with a new hypertensive is the "cultural salt eater." There are some groups in the population that eat very salty, rich foods as a family and cultural tradition. Certain types of dishes extremely rich in salt can increase the intake to 15 to 20 gm/day. This can usually be discovered just by talking to the patient. It is not necessary to collect a 24-hour specimen for sodium excretion, though this would be a way to document it. Some of these people will have a mild drop in blood pressure when they are put on an average salt diet. This has been one of the few instances where altering the salt content of the diet has seemed useful to us in the management of uncomplicated hypertension. Rarely, a patient with severe hypertension can benefit from salt restriction. Sometimes, of course, it is necessary because of renal, hepatic, or cardiac disease.

A few comments about contraceptive pills are in order. There is an average increase in the blood pres-

Presented by Dr. Raper at the 28th Annual Stoneburner Lecture Series, 10 April, 1975, at the Medical College of Virginia, Richmond.

HYPERTENSION: Seated diastolic blood pressure greater than 95 mm Hg in either arm. Use phase V (disappearance of Korotkoff's sounds), if IV and V differ by 4 mm, record both.

SUSTAINED HYPERTENSION: Seated blood pressure greater than 95 mm Hg diastolic on three outpatient occasions out of four at least one week apart. The pressures used may include the "screening" (or initial) pressure.

TRANSIENT HYPERTENSION: Any seated blood pressure greater than 95 mm Hg diastolic. People with transient hypertension should be treated only if appropriate, or scheduled for return appointment for B.P. check in six months.

TREATABLE HYPERTENSION: That hypertension which in the judgment of the physician carries, untreated, a greater risk than that of treatment. Near universal agreement now exists that seated diastolic B.P. greater than 104 mm Hg (sustained) requires treatment in males, perhaps females too; and that greater than 95 mm Hg diastolic (sustained) requires treatment if evidence of cardiovascular damage is present (Grade II, III, or IV fundi, left ventricular hypertrophy, evidence of cardiac decompensation, evidence of coronary artery disease). Impaired renal function, proteinuria, or hematuria are not ordinarily the result of modest elevation of blood pressure. Intermittent hypertension should probably also be treated if there is any detectable cardiovascular damage reasonably attributable to the blood pressure or if documented acute rises seem dangerous.

Fig 1—Definition of hypertension, as used at MCV/VCU.

sure of young women when they take these pills, even in what we would call the normal population (1). The order of this increase is about 14 mm Hg systolic and 8 to 9 mm Hg diastolic. It is sufficient to elevate the blood pressure into what we would call hypertension in a certain percentage of young women who take these drugs. Such patients should be advised to use other means of contraception. This is an unusual type of hypertension; it builds up slowly in weeks to months, and it lasts from four to six months after the pill is discontinued. The physician cannot simply check the patient in two to three weeks post-pill to see if the high blood pressure has gone away. There is a subtle biochemical change in the bloodstream, in which the substrate for renin is actually increased. Renin itself may rise at first, then later fall. Of course, some patients are going to have to be treated, and then have this treatment withdrawn after four to six months to see if the problem has disappeared.

Another aspect in the care of the hypertensive is the "treatment decision." This is the point at which the physician has to decide whether or not to put the patient on (usually) lifetime therapy. It is an important decision because of its implications for the patient, having to do with self-image, the expense of repeated office visits, and trouble for the doctor. Therefore, a decision is best made with all available data: several blood pressures, a history, physical examination, and whatever lab work is appropriate for that particular patient.

Figure 2 shows factors of importance in this decision. We feel that a patient should be treated on the basis of the blood pressure per se if the seated diastolic blood pressure is over 104 mm Hg. This value is

from the Veterans Administration Cooperative Study (2). We should recognize that their study involved middle-aged men only. There were no females. The evidence for treatment is not quite as strong for females, but most authorities in this field feel that women ought to be included in this group, and that treatment is indicated (on blood pressure alone) above 104 mm Hg diastolic pressure. This may change as larger and larger populations are studied in the future.

Below 104 mm Hg but above 95 mm Hg, we feel that an individualized decision is best. Various ways of arriving at this have been advanced. Some physi-

TREATMENT DECISION	
1. Worth careful consideration.	
a. Risk of hypertensive complications.	
b. Cost and bother of pills and office visits; adverse reactions.	
2. Seated diastolic BP > 104 mm Hg (either sex), on three successive, or three out of four, office visits.	
3. Seated diastolic BP 95 to 104 mm HG	
	PLUS
a. Detectable CV damage.	
	●R
b. Risk factors: male sex, age under 50, black race, family history, high cholesterol, diabetes, cigarettes, all readings above 95.	

Fig 2—Factors of importance in deciding whether or not the patient needs treatment.

cians even have a "point system" that adds up "points" for the various factors. Such things considered are age (young), sex (male), race (black), family history (early occurrence of vascular diseases or hypertension), increased cholesterol, and cigarette smoking. If cardiovascular damage can be detected during evaluation of the patient, the likelihood of further damage from hypertension is increased. Such damage might show up as retinal findings, on the electrocardiogram (left ventricular hypertrophy), and so forth. In short, *any* evidence of cardiovascular damage due to hypertension is thought to increase the patient's risk for uncontrolled hypertension and would be sufficient to put him in a "treatment" category, if the diastolic is above 95 mm Hg.

As to plasma renin, the postulate has been advanced that "low renin hypertension" carries a low risk. A few have even gone so far as to speculate that perhaps these patients should not be treated at all. We are not using renin for this purpose. The finding of "low risk" associated with "low renin hypertension" has been contradicted in a number of studies, and the matter is, frankly, controversial (3).

The therapeutic aim of office management of chronic hypertension is reduction of the blood pressure to normal. This tests the staying power of the doctor and patient alike as usually it involves a lifetime of therapy.

The problem of the "missing" patient (non-compliance) is a very difficult one and there is no ideal answer. We send out a lot of letters (saying, "where are you?") and an appointment card requesting the patient to come back in. A one-to-one relationship with the physician helps, and the interest the doctor shows in treating the blood pressure, and his interest in the patient, is most important. It has also been shown that the one-to-one relationship need not be with a physician (4). In large complex clinics, where there is a shifting physician population, it has been shown that a well-trained nurse (or paramedical personnel) can take over this role, with the physician then supervising the medical decisions to make sure that these are appropriate. In most circumstances, however, it is the physician who has to build this one-to-one relationship with the patient.

The patient ought to know the names of the drugs being used for treatment, their chief side effects, and the bottles ought to be labeled. This procedure is useful for most patients, although there will be some obvious exceptions. In terms of patient education, there are recent efforts to produce audiovisual

or tape cassette aids for the patient. I have not yet heard of one that I thought was good enough for general use. Some are under development in the American Heart Association and will be available through local chapters in late 1975. A physician who sees a large number of hypertensive patients may want to tape a brief discussion of the condition for the benefit of new hypertensives to avoid constant repetition.

A number of drugs are available to treat high blood pressure. Figure 3 is an attempt to subdivide hypertensives into levels of blood pressure severity, and to indicate the initial appropriate therapeutic regimen. We have labeled "mildest hypertension" as being in the range of 100 mm Hg diastolic. For this, a thiazide diuretic is probably the best (5). The thiazide diuretic dehydrates the patient very slightly, but the blood volume and total body water come back to normal after about two weeks. The patient does not stay dehydrated, but his blood pressure stays down. Fifty mg/day or twice a day or even 25 mg/day in a few patients can suffice (hydrochlorothiazide). There are some side effects that can be troublesome; hypokalemia, particularly in a patient who is taking digitalis. A rise in the serum uric acid may occur. We ignore this unless it gets very high or unless a patient has a history of gout, in which case it is lowered with allopurinol. Impairment of glucose tolerance occurs in a certain number of patients. This may be ameliorated by correction of hypokalemia. Thiazides do not seem to cause diabetes *de novo*. Other diuretics have

MILDEST HYPERTENSION: Diastolic 100 mm Hg
Diuretic—thiazide is best—HCT 50 mg/day
or b.i.d.

MODERATELY HYPERTENSIVE: Diastolic 110 mm Hg
Reserpine 0.1 to 0.2 mg/day
Thiazide daily or b.i.d.
Add hydralazine 25 to 50 mg t.i.d., if needed

MODERATELY SEVERE: Diastolic 120 mm Hg
Methyldopa 250 mg b.i.d. and up
Thiazide daily or b.i.d.
Hydralazine 25 to 50 mg t.i.d., if needed

SEVERELY HYPERTENSIVE: Diastolic 130 mm Hg
Thiazide daily
Guanethidine 25 mg (or *much more*) q.a.m.
(titrate individual dose by B.P. response)
(Later perhaps add hydralazine)

Fig 3—Drugs useful in treatment of several "grades" of hypertension.

hypotensive effects, but they are not as strong as the thiazide molecule. It does not matter which thiazide is used. There are about 15 of them on the market; it is a matter of dosage size, convenience, duration, and other factors. We use hydrochlorothiazide at MCV.

For moderately hypertensive patients (diastolic in the range of 110), something else is usually used in addition to the thiazide. The "something else" until recently was reserpine. We have taken all of our women hypertensives off reserpine. The reason is that several large surveys done in New York, and repeated in London, have shown association between breast cancer and reserpine therapy. This is a bombshell; nobody expected it. Several people at MCV have been over the data and say that the studies are well done. We are taking it seriously, but the final resolution of this problem awaits more data. The public health hospitals and VA hospitals have not seen fit to interfere with use of reserpine for their hypertensive patients, but have contented themselves with sending out a letter to all their physicians warning them about this data. We use small doses of methyl dopa instead of the reserpine.

If something further is needed in addition to the small dose of reserpine or a small dose of methyl dopa, hydralazine is added. Hydralazine is a very useful drug marketed under the name of Apresoline®. It is an old drug and has been around for a long time. It fell into disuse because of worry about a lupus erythematosus-like syndrome, which was quite serious in some patients. Studies have shown, however, that this will not occur if less than 200 mg/day is given to an average-size patient. Our schedule works out to a maximum dose of 150 mg/day. We use a lot of hydralazine and we have seen very little difficulty with it.

Moderately severe hypertensives, with diastolic hypertension around 120, require methyl dopa instead of the reserpine. The dosage has to be titrated until the desired response is achieved. About 3.5 gm/day for the average-size patient is the largest effective dosage in our experience. There have been some recommendations in the literature for a four-times-a-day dosage, which is difficult for the patient to comply with. We have come to the conclusion that a twice-a-day dosage is acceptable, which is important in terms of compliance.

Severely hypertensive patients require our heavy artillery, guanethidine, marketed as Ismelin® (6). This is an annoying and troublesome drug to deal with. It interferes with sympathetic transmission in

the periphery and, therefore, interferes with postural reflexes. We have a lot of patients walking the tight-rope between symptomatic postural hypotension and control of their blood pressure with guanethidine. The drug takes several days to achieve its full effect, once given, and it takes several days to ease off once the dosage is decreased. It interferes with sexual function in males; this can be a limiting factor. Some men simply will not take the drug once they have had this experience. A physician can sometimes get by with less guanethidine and addition of a somewhat similar drug that does not have the same side effects, such as methyl dopa. The MCV schedule says 25 mg "or much more." The dose of guanethidine is the level achieved in the individual patient after titration. The dosage can be as much as 250 to 300 mg/day in a few patients. Hydralazine can be added to this program also and will sometimes help in the control without adding additional symptoms. Gastrointestinal distress is a problem with guanethidine. It does not seem to produce any irreversible reaction in the G.I. tract, but it can certainly produce a symptomatic diarrhea that is very annoying. Lomotil® (diphenoxylate) is useful for this. Guanethidine can also cause exercise hypotension, a problem with laborers. We try to get by, if we can, without using large doses of guanethidine. There are, however, some patients who require it.

Another drug just recently on the market is clonidine or Catapres®. This drug has a mechanism of action similar to methyl dopa, probably a central (brain) action that "turns down" the sympathetic system so that it is less active. Clonidine is used similarly to methyl dopa. We have not used much of it at MCV because of a pronounced "overshoot" of blood pressure when it is stopped.

A word about drug combinations. It is obvious that more than one drug is often used. Drug combinations are useful and important because they are cheaper and because they simplify compliance to therapy. They may present some difficulties, however. A well-known triple combination contains reserpine 0.1 mg, hydralazine 25 mg, and hydrochlorothiazide 15 mg. If this is given twice a day, an appropriate dose of reserpine is being given but not enough hydralazine or enough hydrochlorothiazide. If the frequency is increased to three times a day, the dosage contains a reasonable amount of hydralazine, but still not quite enough thiazide and already too much reserpine. If the frequency is increased yet to six times a day, entirely too much reserpine is being given. Most

patients will feel badly if they are taking that much reserpine. The hydralazine and hydrochlorothiazide are acceptable. The best way to handle combinations is to start the patient on individual components, see what he needs, and then look up the combinations to see if you can switch him to something that is cheaper and involves one less pill, in which case it is worth doing.

There are a few patients who are not going to respond to standard therapy. A vasodilator of sufficient power would decrease the blood pressure by increasing the size of peripheral arteries, and allowing the blood to circulate faster (7). When this is done, an increase in cardiac output occurs with an annoying heavy palpitation and heavy heart beat. In some instances, angina pectoris occurs where it did not exist before. (This can be a side effect to hydralazine, for instance, in a patient with coronary disease). Such a vasodilator may also cause systemic edema, in some instances even heart failure, and weight gain. These undesirable effects can be blocked. Propranolol, (Inderal®) will block the tachycardia. A diuretic (thiazide or furosemide) will prevent the weight gain and edema. This is called "triple therapy." Vasodilators that are more powerful than hydralazine will come on the market within a year or so, allowing better use of this triple therapy. The name of one is minoxidil. Propranolol must be used cautiously as it does interfere with stroke volume, and it presents a significant danger to people with heart disease, particularly heart disease that has been of sufficient magnitude to have ever caused heart failure. This is regarded as a contraindication under most circumstances. Asthma is also a contraindication. We give propranolol in such a manner as to not drive the heart rate down too low. We like to have the heart rate remain at 60 or above, seated.

In summary, in office management of hypertension, patient education is very important; patient motivation and a strong doctor-patient relationship are needed. The interest and enthusiasm of the physician in treating blood pressure will often be the determining factor in whether or not the patient complies. Very simple therapy suffices for most patients. Control is possible even in most severe cases, but with these, considerable individualization will be needed and frequent office visits.

Figures 1 and 2 are reproduced with permission from the *Virginia Medical Monthly* (101:942-947, 1974).

REFERENCES

1. KUNIN CM, McCORMACK RC, ABERNATHY JR: Oral contraceptives and blood pressure. *Arch Intern Med* 123:362-365, 1969.
2. Effects of treatment on morbidity in hypertension: II. Results in patients with diastolic blood pressure averaging 90 through 114 mm/hg. Veterans Administration Cooperative Study Group on Antihypertensive Agents. *JAMA* 213(pt 2):1143-1152, 1970.
3. OPARIL S, HABER E: The renin-angiotensin system. *N Engl J Med* 29:389-401, 1974.
4. FINNERTY FA: Critical considerations in the treatment of arterial hypertension. *Mod Conc CV Dis* 42:37-40, 1973.
5. HARTMAN F, HEIMSOOTH V: Disturbances in carbohydrate and uric acid metabolism during diuretic treatment. In Gross F (Ed): *Antihypertensive Therapy*. New York, Springer-Verlag, 1966, pp. 436-447.
6. RICHARDSON DW, WYSO EM, MAGEE JH, ET AL: Circulatory effects of guanethidine. *Circulation* 22:184-190, 1960.
7. FREIS ED: The treatment of hypertension. Laragh (Ed): *Hypertension Manual*. New York, Dun-Donnelley, 1973, pp. 743-758.

Some Practical Thoughts on Medical Malpractice

ROBERT L. HARRIS, J.D.

Member of the firm of Harris, Tuck, Freasier and Johnson, Richmond, Virginia

Today more malpractice suits are being filed than ever before. Not only are more suits being filed but the amount of settlements and verdicts has significantly increased. Perhaps one of the primary reasons for this phenomenon is the consumerist movements. Obviously, the more consumer groups advocate consumer protection, and the more that juries render favorable verdicts, the better educated and aware of verdicts the consumer becomes. The result of this is that people become more litigation conscious. In Virginia, medical malpractice litigation appears to be just beginning to flourish in relation to other large urban areas in the North and Far West. Notwithstanding this, malpractice litigation in Virginia is increasing rapidly. For example, over the past twenty years the number of medical malpractice claims in Virginia has increased from approximately 47 in 1955¹ to 272 so far in 1975², which represents an increase of almost 600%. Concurrently, the average cost of concluding a medical malpractice claim has increased from approximately \$4,900 in 1969, to \$10,600 in 1974³, representing over a 100% increase in the past five years.

In order to make this presentation as practical as

possible, I am going to assume that none of you have been a defendant in a malpractice claim and that you know nothing about the law of medical malpractice. The problem is real and must be faced. Senator Abraham Ribicoff in 1969 had the Subcommittee on Executive Reorganization investigate the medical malpractice problem.⁴ One conclusion reached by the subcommittee in its report is that most claims are justifiable,⁵ therefore, I will focus my attention primarily on two questions. First, assuming that there is a bona fide claim, what can a physician do to reduce the insurance company's cost, thereby reducing his insurance premium? Second, what can a doctor do to attempt to avoid a malpractice claim? Before considering these questions, I feel that it would be helpful to briefly discuss the law of medical malpractice, so that a physician will know what the law expects of him.

The Law of Medical Malpractice. The law of medical malpractice is simply another form of what the law classifies as a tort. Very simply, a tort is a private or civil wrong or injury.⁶ For a tort to exist, the following three elements must be present.

1. There must be a legal duty owing from the defendant to the plaintiff.
2. The defendant must fail to discharge this duty.
3. As a proximate result of the breach of this duty the plaintiff must suffer some harm.⁷

Adapted from a presentation made by Mr. Harris at the 28th Annual Stoneburner Lecture Series, 10 April, 1975, at the Medical College of Virginia, Richmond.

¹ Shepherd, *The Law of Medical Malpractice in Virginia*, 21 WASH. & LEE L. REV. 212, 213, n.4 (1964).

² These statistics were furnished to the writer by the St. Paul Insurance Company which insures 85% to 90% of the practicing physicians in Virginia, and only represents the claims against that company. At the present time, the State Bureau of Insurance is compiling data, but these statistics are not currently available.

³ These statistics were furnished to the writer by the St. Paul Insurance Company.

⁴ SUBCOMMITTEE ON EXECUTIVE REORGANIZATION, 91st CONG., 1st SESS., MEDICAL MALPRACTICE: THE PATIENT VERSUS THE PHYSICIAN (Comm. Print 1969).

⁵ *Id.* at 1.

⁶ BLACK'S LAW DICTIONARY 1660 (Rev. 4th Ed. 1968).

⁷ *Id.*

Therefore, the logical starting point is to examine the legal duty a physician owes to his patient.

A physician is not required to exercise the highest degree of skill and diligence possible in the treatment of an injury, unless he has by some special contract agreed to do so. In the absence of such special contract, he is only required to exercise such reasonable and ordinary skill and diligence as are ordinarily exercised by the average of the members of the profession in good standing in similar localities and in the same general line of practice with regard being had to the state of medical science at the time.⁸

This standard does not make the doctor an insurer or guarantor of the results,⁹ in the absence of promising a certain result such as by saying: "I can take care of that and you will have no problem at all." Obviously, a prudent physician would not do this because sometimes favorable results do not always follow treatment, even without fault on the part of the physician.

Within this standard are various duties which a physician must discharge. A physician is not under a legal obligation to exercise the highest degree of care. All that is required is that he exercise that degree of care, skill, or knowledge offered by the *average* reputable physician.¹⁰ This standard is also relative to several other considerations. The law does not test a physician by the standard of other physicians who are not in the same practice or specialty. Therefore, a general practitioner is not held to the same standard as a specialist. A specialist is required to have and exercise that degree of skill and knowledge which is ordinarily possessed by other physicians in that specialty.¹¹ Also, in certain areas of medicine there may be two theories or schools for the treatment of a particular injury or disease. If the physician aspires to one particular school, he must measure up to the proper standard of practice for that school.¹² The physician's standard of care is also relative to the locality in which he practices. For example, a physician in a logging camp in western Virginia would not be expected to exercise the same professional knowledge and skill as a professor at the Medical College of Virginia. What might be malpractice at the Medical College of Virginia might be ac-

ceptable medical care at the logging camp.¹³ Finally, the law imposes on the physician a duty to keep reasonably abreast of the state of medical science at any given time.¹⁴ Procedures and techniques used three years ago, today might constitute medical malpractice if used.

The final element in establishing a *prima facie* case of medical malpractice is causation. The injury or harm which the patient complains of must have been proximately caused by the physician's breach of one of the above duties. This requires the patient/plaintiff to prove a causal connection between the alleged negligence of the physician and the resulting injury. The test actually encompasses two elements. First, the "but for" test is used which establishes a logical causal connection. But for what the physician did, the injury would not have occurred. In addition, the law requires in order to establish proximate or legal causation, that the resulting harm must have been reasonably foreseeable.

From what has been said, it is easy to understand why the law as a general rule requires another physician to testify. This is really the only way that a Court can determine what standard to apply. There is, however, a major exception to this general rule. This is the doctrine of *res ipsa loquitur*. *Res ipsa loquitur* means very simply that "the thing speaks for itself."¹⁵ For this doctrine to be applicable it must be shown that the means or instrumentality which caused the injury was in the exclusive possession and control of the physician charged with negligence; that the physician has or should have had exclusive knowledge of the manner in which the instrumentality was used; and that the injury would not ordinarily occur in the absence of the means or instrumentality being used improperly.¹⁶ The traditional case in which this doctrine has been applied is one in which the physician inadvertently leaves a laparotomy sponge, forceps, or surgical pad in the patient.¹⁷

Assuming that all of the elements exist for a medical malpractice claim, the Statute of Limitations in Virginia for maintaining a claim is two years.¹⁸

⁸ *Alexander v. Hill*, 174 Va. 248, 252, 6 S.E.2d 661, 663 (1940).

⁹ *Ropp v. Stevens*, 155 Va. 304, 308, 154 S.E. 553, 554 (1930).

¹⁰ *Id.*

¹¹ *Fox v. Mason*, 139 Va. 667, 670, 124 S.E. 405, 406 (1924).

¹² *Reed v. Church*, 175 Va. 284, 8 S.E.2d 285 (1940).

¹³ *Fox v. Mason*, 139 Va. 667, 671, 124 S.E. 405, 406 (1924).

¹⁴ *Reed v. Church*, 175 Va. 284, 293, 8 S.E.2d 285, 288 (1940).

¹⁵ *BLACK'S LAW DICTIONARY* 1470 (Rev. 4th Ed. 1968).

¹⁶ *Easterling v. Walton*, 208 Va. 214, 216-17, 156 S.E.2d 787, 789-80 (1967).

¹⁷ See, e.g., *Easterling v. Walton*, 208 Va. 214, 156 S.E.2d 787 (1967).

¹⁸ VA. CODE ANN. §8-24 (Cum. Supp. 1975).

if there has been no fraud or concealment on the part of the physician to prevent the patient from discovering the injury.¹⁹ In certain cases the injury may not manifest itself until some time after the operation or treatment, which raises the question of when the Statute of Limitations begins to run: From the time of the treatment or operation, or from the time that the injury is discovered? Some states have taken the position that the Statute of Limitations does not begin to run until the injury is discovered,²⁰ but Virginia holds that it begins at the time of the wrong and not upon discovery of the injury.²¹

One final consideration before leaving this brief discussion of the law of medical malpractice is the so-called "Good Samaritan Rule." Virginia has passed the following statute which protects a doctor who renders assistance in an emergency situation.

§ 54-276.9 Persons rendering emergency care exempt from liability.

(a) Any person who, in good faith, renders emergency care or assistance, without compensation, to any injured person at the scene of an accident, fire, or any life-threatening emergency, or en route therefrom to any hospital, medical clinic or doctor's office, shall not be liable for any civil damages for acts or omissions resulting from the rendering of such care or assistance.

(b) Any emergency medical care attendant or technician possessing a valid certificate issued by authority of the State Board of Health who in good faith renders emergency care or assistance, without compensation, to any injured or ill person, whether at the scene of an accident, fire or any other place, or while transporting such injured or ill person to, from or between any hospital, medical facility, medical clinic, doctor's office or other similar or related medical facility, shall not be liable for any civil damages for acts or omissions resulting from the rendering of such emergency care, treatment or assistance.

(c) Any person having attended and successfully completed a course in cardiopulmonary resuscitation, which has been approved by the Board of Health, who in good faith and without compensation renders or administers emergency cardiopulmonary resuscitation, cardiac defibrillation or other emergency life-sustaining or resuscitative treatments or procedures which have been approved by the State Board of Health to any sick or injured person, whether at the scene of a fire, an accident or any other place, or while transporting such person to or from any hospital, clinic, doctor's office or other medical facility, shall be deemed qualified to administer

such emergency treatments and procedures; and such individual shall not be liable for acts or omissions resulting from the rendering of such emergency resuscitative treatments or procedures.

(d) Nothing contained in this section shall be construed to provide immunity from liability arising out of the operation of a motor vehicle.²²

With a basic understanding of the law of medical malpractice in mind, I will offer suggestions which a physician may follow in order to prevent a malpractice claim, or if suit is filed or a claim made, how the physician can reduce the cost to the insurance company, and thereby reduce insurance premiums.

Preventing a Medical Malpractice Claim. Various authors and surveys recognize that a decline in the personal physician-patient relationship, lack of rapport, and lack of sympathy are significant contributing factors in explaining the increase in medical malpractice cases.²³ While the obvious cause is the fact that today's society has an abundance of patients and a shortage of physicians, the number of suits filed increases, notwithstanding fault. Crawford Morris, a Cleveland attorney who has defended physicians and hospitals in medical malpractice litigation for many years, wrote the following to the Subcommittee on Executive Reorganization.

It is common knowledge today that almost all doctors are making enormous amounts of money, refuse to make housecalls, play golf on Wednesdays, drive expensive cars, own yachts, hunting lodges and apartment houses.

The doctor's image is sadly tarnished.

Once thought of as "the old country doctor driving through the rain all night to sit beside a sick patient," they are now thought of as "supersuccessful businessmen." This, perhaps subconscious, attitude makes patients more willing to sue their doctors and makes patients on juries more willing to return a verdict and one of considerable size against doctors.²⁴

My parents never would have considered suing their doctor, because he was a friend who they saw at

¹⁹ VA. CODE ANN. §8-33 (Rep. Vol. 1957).

²⁰ See, e.g. *Morgan v. Grace Hospital, Inc.*, 149 W. Va. 783, 144 S.E.2d 156 (1965).

²¹ *Hawks v. DeHart, Adm'x*, 206 Va. 810, 146 S.E.2d 187 (1966).

²² VA. CODE ANN. §54-276.9 (Cum Supp. 1975).

²³ SUBCOMMITTEE ON EXECUTIVE REORGANIZATION 91ST CONG., 1ST SESS., MEDICAL MALPRACTICE: THE PATIENT VERSUS THE PHYSICIAN 3-4 (Comm. Print 1969); U. S. News & World Report, Jan. 20, 1975, p.54; U. S. News & World Report, June 16, 1975, p.50-51.

²⁴ SUBCOMMITTEE ON EXECUTIVE REORGANIZATION, 91ST CONG., 1ST SESS., MEDICAL MALPRACTICE: THE PATIENT VERSUS THE PHYSICIAN 3 (Comm. Print. 1969).

church, at the movies, or at a picnic. Today, we live in an urban society and doctors are simply too busy to spend the time with patients to become their "friends." If a physician will recognize this situation and accept it from the outset, I believe certain steps can be taken which will perhaps help remedy it.

On numerous occasions I have had the following stereotype of doctors presented to me by clients and acquaintances. Most of the following comments are not based on my own experience and obviously are not applicable to every doctor. First, when the patient goes to a doctor, he or she finds a receptionist who is extremely busy and has no time to give the patient her personal attention. The patient waits sometimes an hour or more past the appointment time and is then ushered into a small examining room where an assistant comes in and takes the patient's temperature and blood pressure. These people do not really see the patient as a person. Finally, the doctor comes in. The doctor may have twenty patients scheduled for that hour and can afford to spend only from three to five minutes with the patient. How can one expect such a patient to feel that he or she should refrain from suing that doctor if an unexpected injury results from the treatment. This is often the patients' view, and much of this feeling is caused by the tremendous demand placed on that physician's time. I submit, however, that if the physician would pat the patient on the hand and take just a few minutes to explain the situation in language that can be understood, perhaps 50% or more of the medical malpractice claims would not be filed. Patients do not sue doctors for whom they have a warm feeling, unless of course, it is an obvious case of negligence.

Another thing a physician should always do is keep detailed, legible, and dated notes. These are more helpful to the insurance company than anything else, and this is particularly true in cases involving informed consent. Informed consent means more than just telling the patient that there is a possibility of complications. The physician is under a legal duty to disclose to the patient risks incident to medical diagnosis and treatment. A physician who fails to make such disclosure may be legally liable for adverse consequences even though the physician was not negligent in his treatment.²⁵ The rationale behind this rule is that every adult of sound mind has the legal choice to determine what shall be done with his or her own body. This requires that the patient consent

to an operation or treatment, and for the consent to be meaningful, it must be intelligent in the sense that the patient is aware of all pertinent facts.

A good example of an informed consent case is the Texas case of *Wilson v. Scott*.²⁶ In this case, the patient had diminished hearing in one ear and the doctor decided that a stapedectomy would probably improve his hearing. According to the doctor's testimony, the patient was informed that there was a 90% chance for hearing improvement and a 10% chance that hearing would not improve. However, the doctor failed to inform the patient that there was a 1% chance of hearing loss. As a result of the operation, the patient lost all hearing, experienced vertigo, instability, and tinnitus. On the basis of expert testimony, the Court found that it was standard practice to inform the patient that there was a 1% chance of hearing loss and accordingly rendered a verdict in favor of the patient. Even though there was no evidence that the doctor was negligent in the performance of the stapedectomy, the patient, if he had been advised of this possibility, might have decided that he would prefer to have diminished hearing rather than take a chance on an operation which could result in a complete loss of hearing.

This appears to be the type of case which is presenting itself more frequently today than any other. In order to prevent this, the physician should not only inform the patient of possible adverse consequences, but also note in the medical records that the patient has been so informed. If the physician does not put this in the medical records, then it becomes the physician's word against the patient's. Many physicians say that they informed the patient in a particular case because they always inform their patients. It is submitted that doctors are extremely busy and it is not impossible for them to forget to inform the patient in such a case, because at the time they were perhaps interrupted or thought they had told the patient the last time they saw him or her.

For the above reasons, it is submitted that if the doctor would prepare a written form advising the patient about the operation or treatment and have the patient read and sign it, three goals would be accomplished. First, if the patient says that he or she was not informed of this, or that, the doctor can go back to the records, pull the consent form, and see exactly what the patient was informed of. Second, such a procedure will require the doctor to think

²⁵ Dietze v. King, 184 F. Supp 944 (E.D. Va. 1960).

²⁶ 412 S.W.2d 299 (Tex. 1967).

about what the adverse consequences of an operation are rather than inform the patient somewhat rotely. Finally, if a form is used prior to every operation, it will prevent the doctor from forgetting for some reason or another to inform the patient.

Below are some of the basic facts that should be included in the consent form and presented to the patient prior to an operation. This list is not intended to be exhaustive and additional information may be deemed necessary under certain circumstances.

1. Have the patient authorize the performance of the operation.
2. Inform the patient of the nature of the procedure necessary to treat him.
3. Inform the patient of the risks associated with the particular operation.
4. Inform the patient of the consequences which are normal in the procedure.
5. Inform the patient of the risks inherent in the performance of any surgery.
6. Inform the patient of reasonable alternative treatment, if it exists.

One obvious question a physician will ask is, do I have to tell the patient of every conceivable adverse consequence? The answer is no, because in order for the doctor to be liable, he must have fallen below the standard of the reasonably prudent physician. One author has suggested the following four factors for the physician to consider in determining what risks he should inform the patient of:²⁷

1. The nature or degree of the risks, harm, or adverse result;
2. The frequency or percentage of cases that such risk, harm, or adverse result occurs;
3. The probable effect of the procedure or treatment on the patient's health or well being;
4. The probable effect of disclosure of the risks on the patient's mental health or well being.

The Anatomy of a Medical Malpractice Case.

If the foregoing preventive measures fail, there are still certain others which can be taken. Therefore, I am going through the anatomy of a medical malpractice claim, step by step, and explain what you as doctors can do for self-protection and also save the insurance premium dollar. To better understand how some of these suggestions can save the insurance premium dollar, it would be helpful to see where your premium dollar goes. For every dollar spent for insurance, approximately 30% goes to the injured patient; approximately 15% goes to the plaintiff's attorney; and

the balance, approximately 55%, goes to the defense attorney and defense investigation costs.²⁸ It is obvious that if a claim is concluded at an early date, much of the defense costs can be eliminated.

If the physician has an idea that a claim is going to be made, he should notify his insurance carrier as soon as possible. Upon notification an adjuster can go out and talk to the claimant right away and perhaps conclude the matter promptly, and prior to an attorney becoming involved. This would avoid much of the defense and litigation expense, consequently lowering the insurance premium. I know that most professionals, myself included, are very reluctant to admit error, but by the same token "stone walling" is not the answer. If a patient has a legitimate claim, it is to the physician's advantage, monetarily as well as emotionally, to conclude the matter as expeditiously as possible. If the claim is not valid, it still cannot be ignored. It is the insurance company's responsibility to investigate and dispose of the claim. They are trained in these areas and are not going to settle a claim that is not valid, or pay any more than the claim is worth.

If the claim cannot be settled initially, let me explain what an experienced, competent attorney will do when a client comes in. One article analyzed what it cost for an attorney to handle a medical malpractice case.²⁹ This article used metropolitan New York as its setting. The result was that for an attorney to *net* \$30,000 per year, he must produce \$62 per billable hour at a minimum. Further analysis revealed that the average malpractice case required the attorney to spend 67 hours prior to trial. If this is computed, it amounts to over \$4,000 of the attorney's time. It should, therefore, be obvious that it would be economic suicide for an attorney to take a meritless malpractice case. If, however, the case has merit and the injury is considerable, the lawyer has an ethical and moral responsibility to prosecute the claim, just as a doctor has to treat the patient who is sick or injured.

Finally, there is the case with marginal liability, and the harm is not great. A lawyer will evaluate the

²⁸ These statistics were furnished the writer by St. Paul Insurance Company. See also SUBCOMMITTEE ON EXECUTIVE REORGANIZATION, 91st CONG., 1st SESS., MEDICAL MALPRACTICE: THE PATIENT VERSUS THE PHYSICIAN 10 (Comm. Print 1969). While it is true that most medical malpractice cases are handled on a 33 1/3% contingent basis, attorneys do not become involved in all claims.

²⁹ Shayne, *Meritless Malpractice Cases: A Fragile Dilemma*, in MEDICAL MALPRACTICE 309 (Practicing Law Institute 1975) (Reprinted with permission from Trial, May-June 1975).

²⁷ 11 HOUSTON L. REV. 1075, 1076, (1974).

case and the physician involved. If the lawyer knows that the doctor has done everything possible for the patient and has tried to keep a good rapport with the legal profession, he will probably discourage the case. Plaintiff's attorneys who handle automobile liability cases have considerable contact with doctors. Occasionally, when these attorneys try to get medical reports from doctors they have to beg for them. Also, when some doctors write a report, they attempt to minimize the patient's injury for some reason, even when the injury is legitimate. Another situation which arises is where the attorney needs the doctor to testify in an automobile liability case. The attorney calls the physician for a pre-trial interview to explain what questions will be asked from both sides. The appointment may be arranged for 4:00 PM, and the doctor finally may see him at 7:00 PM. If an attorney has experienced this situation with the doctor, he is very unlikely to discourage litigation in a marginal medical malpractice case. I submit that if doctors would treat the attorneys the way they want to be treated, the attorneys would inevitably discourage a marginal medical malpractice case.

Once the attorney determines that the patient's claim is legitimate, he will file a Motion for Judgment.³⁰ In this the attorney will put every possible basis for a malpractice action he hopes to prove. When the physician is served with this, he will think, "I sound like the worst person in the world." Also, the attorney will sue for the upper limits which he hopes to recover. If you are served with the paper, do not get upset or attempt to ignore it. You should go immediately to the insurance company so that they can start working on the case. Perhaps when they investigate the case, they can bring it to a conclusion even before the responsive pleadings are filed. If they can, it will save a lot of the defense dollars and also save you a lot of mental anguish.

If it cannot be settled at this point, the insurance company's lawyer will file responsive pleadings, and the issues will be joined, and the case matured. The next step is the discovery process. This is where your medical records become invaluable. Often lawyers would not file a lawsuit if they saw the medical records and had a chance to analyze them. If, however, the medical records are not legible or do not exist, these facts can be used against you.

At this stage of the litigation, the plaintiff's attorney will probably subpoena your records, file in-

terrogatories, and take depositions. Interrogatories are written questions which must be answered under oath. Take your time with your attorney and give him complete and accurate answers. Next, depositions, which are oral questions under oath before a court reporter, will probably be taken. I recommend that you take the time to appear, even if you are not going to be questioned. The reason for this is that if a witness is inclined to stretch the truth, he or she will be less likely to do so if you are present. If you are questioned, give complete and accurate answers, because you are bound by these at trial. Obviously, if there are any inconsistencies, the other attorney will capitalize on them.

After discovery is completed by both sides, an experienced attorney is usually able to anticipate what result will be obtained at trial. If the patient's attorney determines at this point that he cannot prove the case, he will usually non-suit or dismiss the case. On the other hand, if the doctor's lawyer thinks that the case can be proved, he owes it to the doctor to approach the plaintiff's attorney with a settlement offer. Again, if the case is settled at this stage, a lot of the premium dollar can be saved.

If no settlement or agreement is made, the next step is the trial. You want to appear to the jury as the nicest fellow in the world and that you could not do anything except serve mankind. You want to appear friendly and have a pleasant expression on your face. If someone gets on the witness stand and begins to stretch the truth, or says something that you do not agree with, do not start grimacing, because the jury might think that you are bitter and perhaps punish you for that. When you testify, the key point is for you to have read the medical records and know exactly what is in them. When answering questions, face the jury. They are the ones you have to convince. Look at the jury and be candid, but whatever you do, do not get mad. Sometimes, the other attorney will use this as a trial tactic so that you cannot think properly and as a result the jury will be unimpressed with your testimony. Therefore, remain calm and think before you answer his questions.

The foregoing is not intended to be a comprehensive examination of the medical malpractice crisis. It is submitted however, that if some of the suggestions herein are followed, many of the potential medical malpractice claims would not be filed, or if filed, terminated at an early date, thereby saving the medical malpractice insurance carriers substantial sums, and consequently reduce the premiums for physicians.

³⁰ In Virginia a lawsuit is initiated by filing a Motion for Judgment. SUP. CT. OF VA. R. 3:3. In the federal courts and in other state courts it may be called a complaint.

Asthma in Adults

JAMES P. BAKER, M.D.

*Pulmonary Division, Eastern Virginia Medical School, Norfolk General Hospital,
Norfolk, Virginia*

Asthma, or acute reversible obstructive airways disease (ROAD), is a major respiratory disease affecting approximately eight million people in the United States. The disease is characterized by attacks of wheezing dyspnea with coughing and sputum production. Although most adult patients who have the disease demonstrate either bronchospastic episodes during childhood or will have repeated episodes of respiratory infection, upper and lower or both during early years, some patients do begin only in later life, that is, adult onset asthma. All of these patients usually have no symptoms or signs during interval periods, but with proper stimuli will demonstrate airways obstruction.

The pathogenesis and pathology of asthma are located in the airways where there is bronchospasm from contraction of the circumairway smooth muscle, mucosal edema, and excessive mucous production, all leading to narrowing of the airway lumen. These processes are initiated by the release of chemical mediators from pulmonary mast cells by a variety of stimuli. The abnormal response of the asthmatic bronchus may be caused by an immune reaction involving IgE, by infection, by certain physical factors including exercise and cold temperature, as well as by a combination of these factors and probably other unknown factors. Patients with asthma respond to minimal stimuli with increase in airways resistance indicating the presence of increased airway tone or ease of provocation—"twitchy airways." This is indicated by the smaller dose of known airway

constrictors necessary to produce bronchoconstriction in an asthmatic than in a normal person. This has led to the theory of partial beta blockade. The sympathetic beta two receptors located in the airways are bronchodilating. Inhibition or blockade of these receptors results in bronchoconstriction, thus suggesting a partial beta blockade in patients with asthma. Alpha sympathetic stimulation also leads to bronchoconstriction. This knowledge has led to the better understanding of the biochemical mechanisms of asthma and to drug therapy. Superficially, bronchodilation is medicated primarily by increases in 3, 5 cyclic AMP. Cyclic AMP production is increased by beta agonists, for example, ephedrine, isoproterenol, isoetharine, and others. Its breakdown is inhibited by methylxanthines, for example, aminophylline. Therefore, it is very rational to treat asthmatics with both of these classes of drugs at the same time (1-4).

The diagnosis of ROAD is not difficult in the usual case. However, particularly in older adults, difficulty in separating other chronic airways disease, especially chronic bronchitis, becomes a problem. These two conditions frequently occur together and should be separated, according to the purist. From a practical therapeutic standpoint, it makes little difference, except from a prognostic standpoint, since chronic bronchitis tends to be a progressive disease and asthma may not be.

The management of ROAD can be separated into three phases: 1) Outpatient, 2) Emergency Room, and 3) Inpatient. The objective of therapy should be to avoid, if at all possible, phases two and three.

Outpatient management should include a diligent search for inciting causes of episodes,

Presented by Dr. Baker at the 28th Annual Stoneburner Lecture Series, 11 April, 1975, at the Medical College of Virginia, Richmond.

particularly those avoidable irritants which must be isolated and removed from the patient's environment. This must include a thorough history to evaluate the problems of allergy, for example, previous history of allergic reactions, eczema as a child, recurring rhinitis, hayfever, and other factors. On physical examination, the presence of congested, bluish, nasal mucous membranes or nasal polyps suggests allergy. Eosinophilia in nasal or sputum smears or peripheral blood smears, additionally, suggests allergy and should be evaluated in all patients with ROAD. Since many parasitic diseases will have pulmonary involvement, the stools of patients with peripheral eosinophilia should be evaluated for ova and parasites. Most people spend more time in their bedroom than in any other room, thus attempts should be made to keep this room as dust- and antigen-free as possible. Therefore, nonallergic bed covers and pillows should be used, overstuffed furniture, wool rugs, and draperies should be removed. The room should be cleaned in the morning to allow dust to settle before night. No animals should be allowed in the bedroom. I believe that all patients who have a suggestion of allergy should be evaluated by an allergist and desensitized if indicated.

The drug therapy of ROAD should include both sympathomimetics, (epinephrine, ephedrine, terbutaline, metaproterenol, and others) plus xanthines (aminophylline) for the above mentioned reasons. Depending upon the severity of the difficulty, the dosage of these drugs will need to be varied. Some patients will require drugs intermittently, some continuously. Since much of the problem is caused by excessive mucous plugging of airways (7), and antihistamines have a drying effect on mucous, tending to make its removal more difficult, I prefer not to use these drugs. Instead, these patients should be encouraged to force hydrate themselves to keep secretions in as loose a state as possible. The use of antibiotics is predicated on the presence of infection which is best assessed by evaluation of the sputum, both in wet preparation and gram stains, to look for polys and pathogenic bacteria as well as eosinophils, Charcot-Leyden crystals, and Curschmann's spirals. Adrenal steroids should be used as the last drug when other drugs have failed or in patients who have been known to require steroids previously. Another drug which has outpatient application is cromolyn sodium. This drug has no effect in the acute attack and should be used only to prevent attacks. It should be tried in patients who have frequent bronchospastic

episodes or who require continuous steroids to avoid attacks. It must be administered four times daily and should be given for several weeks to assess its therapeutic value. The combination of the above modalities should be adequate to control 80% to 90% of all patients with ROAD.

Most patients who arrive in the Emergency Room because the above regimen has failed or because they have not had proper medical supervision will respond to parenteral bronchodilators. However, arterial blood gases should be measured to evaluate the need for oxygen therapy. Eighty percent or more of these patients will have mild hypoxemia and will be hyperventilating, (low PaCO_2) (5). If the pCO_2 is normal or elevated, this indicates a more severe state and such an individual should be considered under phase three (6). Either subcutaneous epinephrine 0.3 cc, 1:1000 aqueous epinephrine or terbutaline 0.25 mg should be given initially and may be repeated in 15 to 30 minutes if needed. Obviously, patients with cardiac disease (arrhythmia, angina, hypertension, and others) are at great risk under these conditions, particularly if very hypoxemic, and must be monitored carefully. If this is ineffective, intravenous aminophylline should be used, 5 to 6 mg/kg body weight, no more rapidly than 50 mg/min (10).

Aerosolized sympathomimetics may be used also, or instead of parenteral drug. Again, the cardiac rate, rhythm, and blood pressure must be monitored since there is a significant systemic effect from most of these drugs.

Most of these patients will be quite anxious and the temptation to sedate them is great. They are anxious because they are hypoxemic and dyspneic. Additionally, most bronchodilators have central effects which are stimulatory, increasing anxiety. Although some patients can trigger bronchospastic episodes by anxiety, the use of tranquilizers or sedatives is exceedingly dangerous, thus these drugs should not be used. Patients with ROAD do not thrive in the average emergency room environment. They should be kept in a quiet area under constant supervision and frequently reassured that they are receiving adequate care. If this, plus the above therapy is administered, most of these patients will not require admission. Several series have demonstrated that only 2% to 5% of all patients will require admission.

The inpatient management of ROAD, phase three, covers only a very small percentage of the total population of patients at risk; however, if improperly treated during this phase, the mortality is

significant. The question is frequently asked as to which patients should be admitted. The following guidelines are useful:

1. All patients with high normal or elevated PaCO_2 's or low pH's, that is, less than 7.30, need admission.
2. Patients with forced expiratory volume in one second (FEV_1) of less than 0.5 liters. FEV_1 's are sometimes difficult to measure and equipment is not always readily available, therefore, alternate mechanisms to assess function are needed. McFadden (9) demonstrated that intercostal retraction related well to severe mechanical obstruction and usually disappeared with FEV_1 's greater than one liter. Rebeck (8) has shown that pulsus paradoxus of greater than 10 to 12 mm Hg systolic blood pressure correlated well with severe reduction in FEV_1 . Thus, the presence of retractions and paradox should indicate admission.
3. Patients with prolonged attacks, that is, greater than 12 hours.
4. Patients demonstrating physical exhaustion.
5. Patients who fail to respond to phase two measures within four to six hours.
6. Patients with falling PaO_2 and rising PaCO_2 with emergency room management.

Once the decision to admit has been made, the severity of the situation will dictate the best area of the hospital for therapy. Generally, the ICU should be considered as the desired location. The scope of intensive care management is beyond this paper, but several points should be made (2, 4, 6):

1. No sedatives.
2. Continuous O_2 therapy (best given by nasal cannula) to maintain PaO_2 , 60 to 70 mm Hg.
3. Frequent arterial gases until the patient is clearly better.
4. Continuous aminophylline I.V. (0.9 mg/kg/hr) after an initial loading dose (5.6 mg/kg/20 min).
5. Inhaled or subcutaneous sympathomimetic drugs, or both, using monitoring of cardiac rate, rhythm, and blood pressure as the controlling factors.
6. ECG monitoring.
7. Adequate hydration, since these patients are almost invariably dehydrated, which causes difficulty in clearing trachea-bronchial secretions. This initially will have to be intraven-

ous. Various estimates of fluid deficit have been made; however, good data is not available. Inpatients with normal cardiovascular-renal mechanisms should have fluid therapy until their secretions are thin and easily cleared.

8. Humidity therapy with warm moist air, if tolerated, will help to loosen secretions.
9. Corticosteroid therapy is a major portion of the therapy and must be given in pharmacologic doses. Methylprednisolone, 250 mg, I.V. Q4h, has been very valuable in this area. It is preferred because of less mineralocorticoid effect, plus a longer half life than hydrocortisone. There are other less well defined reasons for the use of this drug.

The other frequently asked question is, when should these patients be placed on continuous ventilator support? There are several guidelines which are used by most physicians caring for numbers of these patients, and these guidelines are based primarily on arterial blood gas studies. If the PaCO_2 begins to rise above the normal range (greater than 45 mm Hg) or the pH falls below 7.30 with the above program, these patients are at great risk for respiratory and cardiac arrest, therefore, we electively intubate and assist these patients. Marked depression in the level of consciousness from severe fatigue indicates the need for ventilatory assistance. Continued unremitting bronchospasm with optimal therapy should also be considered an indication for ventilatory assistance.

In summary, reversible obstructive airways disease (ROAD) is a common respiratory problem. Proper education and outpatient management will almost always provide adequate care. However, some patients require more active therapy in emergency rooms or hospitals. Since almost all patients, properly treated, will survive to return to productive life, a well-planned, aggressive therapeutic approach is needed as outlined in this paper.

REFERENCES

1. MCCOMBS RP: I. Diseases due to immunologic reactions in the lungs. *N Engl J Med* 286:1186-1194, 1972.
2. WILLIAMS MH JR: Pathophysiology and treatment of severe asthma. *NY State J Med* 73:2446-2451, 1973.
3. GOLDBERG ND: Cyclic nucleotides and cell function. *Hosp Pract* 127-142 (May), 1974.

4. PETTY TL: Bronchial asthma: New information leads to better therapy. *Mod Med* 17-22 (July), 1974.
5. MCFADDEN ER JR, LYONS HA: Arterial-blood gas tension in asthma. *N Engl J Med* 278:1027-1032, 1968.
6. FRANKLIN W: Treatment of severe asthma. *N Engl J Med* 290:1469-1472, 1974.
7. TERR AI: Bronchial asthma, in Baum GL (ed): *Textbook of Pulmonary Diseases*, ed. 2. Boston, Little Brown, 1974, pp 421-443.
8. REBUCK AS, READ J: Assessment and management of severe asthma. *Am J Med* 51:788-798, 1971.
9. MCFADDEN ER JR, KISER R, DEGROOT WJ: Acute bronchial asthma: Relations between clinical and physiologic manifestations. *N Engl J Med* 288:221-225, 1973.
10. MITENKO PA, OGILVIE RI: Rational intravenous doses of theophylline. *N Engl J Med* 289:600-603, 1973.

Sprains and Strains

ROBERT W. WADDELL, M.D.

Virginia Beach Orthopedic Associates, Inc., Virginia Beach, Virginia, and Instructor, Orthopedic Surgery, Eastern Virginia Medical School, Norfolk, Virginia

There is a tendency for many physicians to lump sprains and strains into one category and to consider all of them minor injuries. This is far from true and it is hoped that this discussion will result in a better understanding of the difference between the two, more accurate diagnosis, and more effective treatment.

A strain is damage to a muscle tendon unit. A first-degree, or mild, strain is one in which there is no disruption of fibers. There is usually some well-localized edema. Treatment need only be that of rest and protection against stress until the patient becomes more comfortable. A second-degree, or moderate, strain is one in which there is damage to the fibers of the muscle tendon unit. There may be some loss of strength in addition to localized edema. Treatment usually consists of rest, ice, elevation, and occasionally antispasmodics and immobilization. A third-degree strain, or severe strain, is one in which there is complete rupture of the muscle tendon unit with marked loss of strength and extensive edema. Early diagnosis is essential in order to institute the proper treatment as soon as possible. Surgical repair will often be necessary in third-degree injuries.

A sprain is a ligamentous injury from overstress. A first-degree, or mild, sprain is one in which a few of the fibers are torn. There will be localized edema and hematoma formation, but no apparent loss of strength can be detected. Treatment is symptomatic. A second-degree, or moderate, sprain is one in which there is partial tearing of the ligament with some loss of strength. A second-degree sprain should be immobilized and complete recovery can usually be ex-

pected. A third-degree, or severe, sprain is one in which there is complete disruption of the ligamentous structure (Fig 1C). Often there will be a palpable defect, and marked edema will be present. Abnormal motion of the joint can usually be detected, especially if one injects the joint with a local anesthetic prior to examination. X-rays should always be made and of particular importance are stress x-rays. These will prove to be of great value in deciding whether or not the injury is second or third degree. Again, injection of an anesthetic into the joint makes it much easier to carry out an accurate stress x-ray. This type of injury is always treated by rigid immobilization and occasionally surgical repair.

The acromioclavicular joint is retained by the coracoacromial, the acromioclavicular, and the coracoclavicular ligaments (Fig 2). First-degree sprains of this joint are treated symptomatically. Second-degree with partial tearing usually results in slight superior displacement of the distal clavicle. This can be very adequately treated with an acromioclavicular strap fashioned in the office by placing a felt pad under the elbow, then applying an adhesive strap from the elbow to the AC joint while at the same time exerting some downward pressure across the distal end of the clavicle. The arm is then fastened to the body in a Velpeau fashion. Commercial acromioclavicular harnesses are also available. This type of treatment usually is very effective, but it should be carried out for a period of six weeks and the joint protected from additional stress for several weeks longer. Third-degree acromioclavicular sprains result in complete disruption of the joint with gross displacement superiorly of the distal clavicle (Fig 3). There will always be marked hematoma formation, point tenderness, and the patient will have significant functional loss. Ab-

This is an edited transcription of a lecture given by Dr. Waddell at the 28th Annual Stoneburner Lecture Series, 11 April, 1975, at the Medical College of Virginia, Richmond.

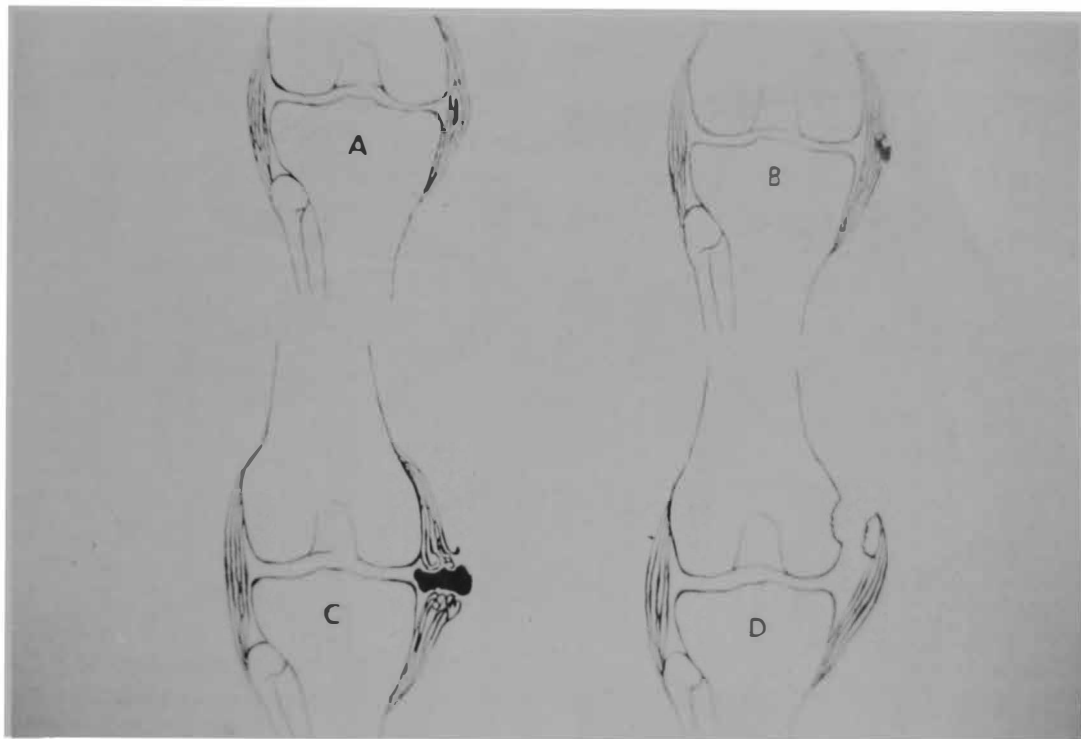


Fig 1—Sequence showing degrees of sprain. A. First-degree sprain with minimal bleeding; B. Second-degree sprain with more fibers torn; C. Third-degree sprain with complete disruption; D. Third-degree injury with avulsion fracture. (From O'Donoghue, *Treatment of Injuries to Athletes*, p 75, 1970, with permission from author and W. B. Saunders Company.)

normal motion can be demonstrated by grasping the clavicle at the middle third and then moving it anteriorly and posteriorly. The pathologic motion at the distal end of the clavicle can readily be perceived. Stress x-rays are sometimes helpful and are made by having the patient stand upright and hold heavy weights in both hands. This will usually demonstrate the separation. In younger people, it is advisable to surgically repair the acromioclavicular ligaments and stabilize the joint until healing has taken place. The results from this type of treatment are uniformly good. In an older patient, an acromioclavicular strap would be adequate management.

The glenohumeral joint is one of our most unstable joints and as a result is frequently injured. It is reinforced by a strong musculotendinous structure called the rotator cuff. This is composed of the subscapularis, supraspinous, infraspinous, and teres minor muscles. First-degree strains of this joint are

treated symptomatically. Second-degree strains are treated by immobilization for a period of four weeks. A third-degree injury with dislocation is treated for a period of six weeks in a shoulder immobilizer. Anterior subluxations frequently occur as a result of abduction and external rotation stress. Capsular tearing results and the head rides over the glenoid rim and then relocates spontaneously. The history is extremely important in diagnosing this particular injury. When this injury takes place, it results in tearing of the anterior portion of the shoulder capsule, but the force stops short of causing complete dislocation. Immobilization for a period of six weeks is in order to allow for capsular healing.

Anterior dislocation of the shoulder is one of the most commonly seen injuries. There will often be a palpable as well as a visible defect. Moderate tenderness and swelling will be apparent. The physician should always evaluate the neurovascular status both

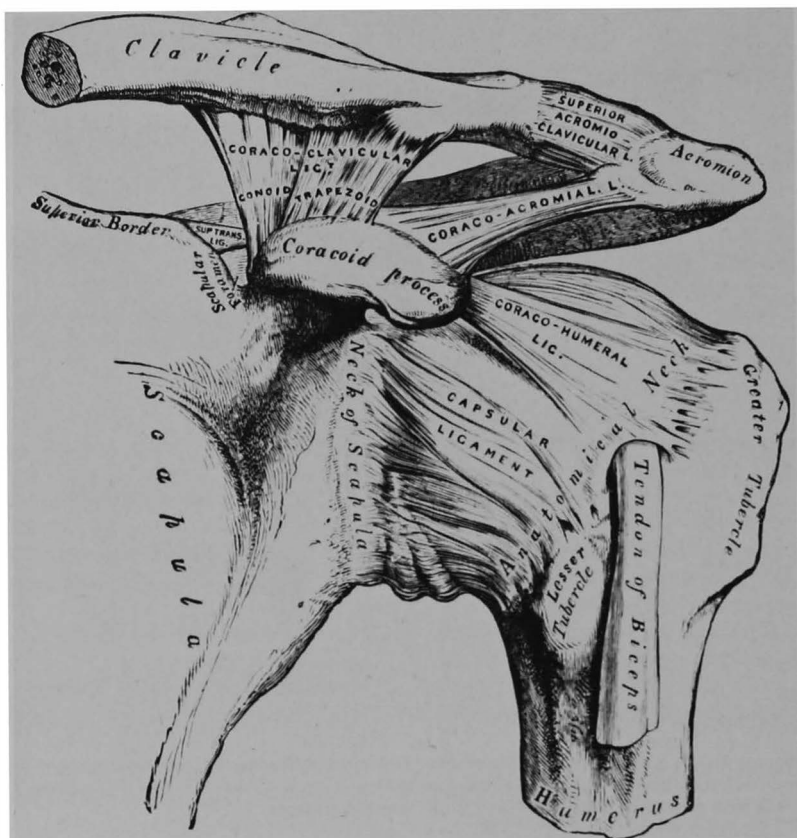


Fig 2—Detail of clavicle. (Note strong reinforcing ligaments of the acromioclavicular joint.)

prior to and after reducing the dislocation, and x-rays should be made before and after manipulation. Avulsion fractures of the greater tuberosity are frequently associated with shoulder dislocations and usually do not influence treatment.

Posterior dislocations of the shoulder are extremely rare and a routine anteroposterior x-ray may be misinterpreted as showing a normal glenohumeral relationship. A transthoracic lateral x-ray is essential to make this diagnosis. The use of intravenous Robaxin® and Demerol® can be extremely helpful when reducing a dislocated shoulder. Longitudinal traction with gentle abduction usually results in relocation. In the event that this fails to accomplish relocation, the Kocher maneuver can be carried out.

Strains of the arm do occur. First- and second-degree types present no particular problems in diag-

nosis or treatment. Third-degree strains involving complete disruption are major injuries and in general should be treated by surgical repair. Rupture of the biceps muscle is one of the most common types of third-degree strains about the arm.

Elbow injuries often present difficulty in diagnosis in children because of the numerous open epiphyses. First- and second-degree sprains are treated symptomatically and immobilization when necessary is not needed longer than three weeks. With children it is often wise to x-ray the opposite elbow for comparison. A physician will occasionally see a positive fat pad sign, indicating bleeding into the elbow joint (Fig 4). This is frequently the result of fracture which may or may not be visible on the initial x-rays. Since these patients usually have a very irritable elbow joint, it is wise to immobilize the el-

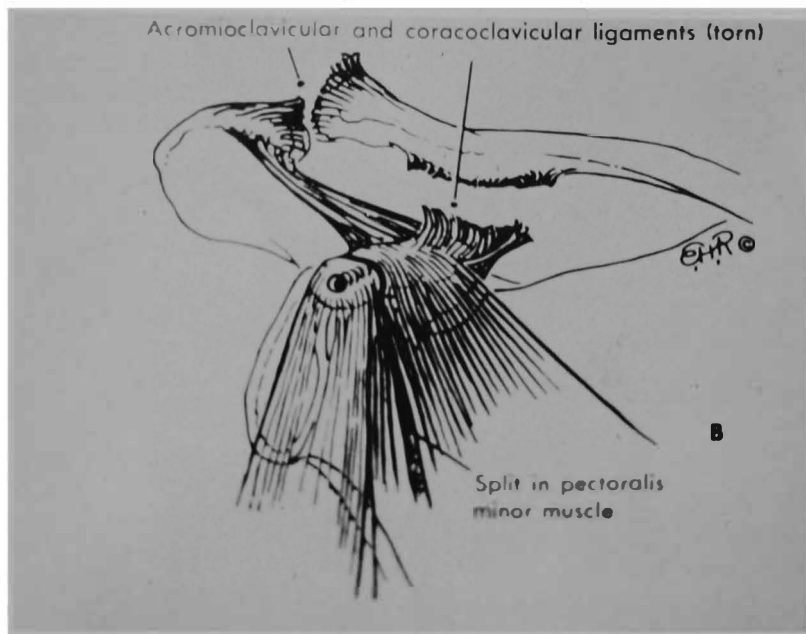


Fig 3—Complete dislocation of the acromioclavicular joint.

bow and re-x-ray the joint in 10 to 14 days. Many times a linear supracondylar elbow fracture will be apparent on the second series of x-rays.

A nursemaid's elbow is frequently encountered in the Emergency Room in the toddler. This occurs because the radial head is small at this stage of development and longitudinal stress results in partial subluxation of the radial head from beneath the annular ligament. The child presents with a painful elbow and will not move the arm. X-rays are negative. Relocation is simple and can be accomplished by placing the thumb directly over the radial head and supinating the forearm as it is flexed at the elbow. A pop or click can usually be felt beneath the thumb, indicating that the radial head has slipped back under the annular ligament. The child should then be immobilized in a cuff and collar for a period of seven days.

A third-degree elbow sprain with dislocation results in damage to the collateral and capsular ligaments. Fractures are frequently associated and neurovascular damage may be seen. This is truly a surgical emergency and should receive priority over many other types of injuries. Reduction can usually be accomplished without general anesthesia. The patient is given a narcotic and the joint is occasionally injected with a local anesthetic. Longitudinal traction is ap-

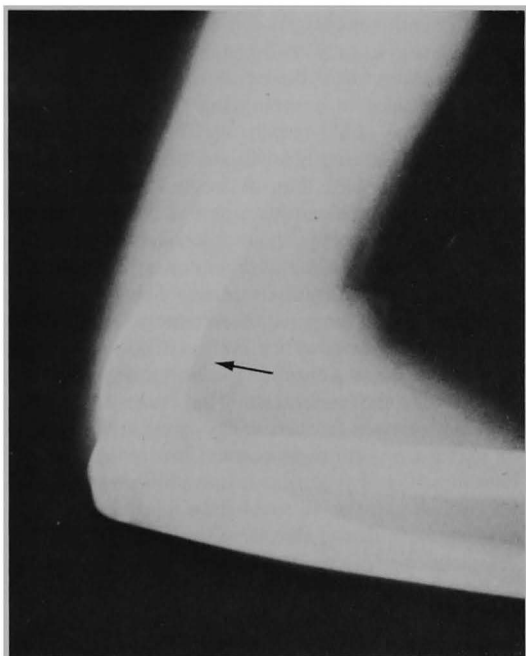


Fig 4—X-ray of elbow injury. (Note darkened shadow anterior to humerus, indicating bleeding within the joint.)

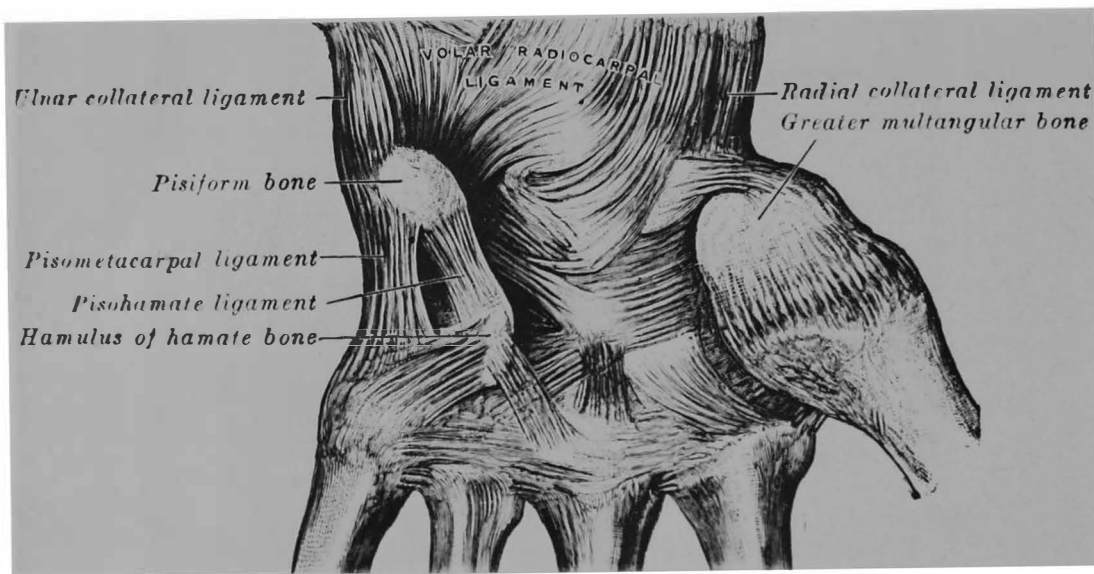


Fig 5—Detail of ligamentous structure of the wrist.

plied until the coronoid process of the ulna slips over the humeral condyle. When this is accomplished, gentle flexion usually results in relocation. Swelling is often marked with these injuries and great care should be used in immobilizing the extremity with anything that might impair circulation. A posterior plaster splint is usually adequate for initial immobilization and is safer than a circular cast. The joint should be immobilized for a period of three weeks.

Sprains of the wrist are infrequent but are frequently diagnosed. Most are tendon or bone injuries and are not ligamentous injuries. The ligamentous structure about the wrist is extremely dense and is stronger than the bony structures (Fig 5). The physician should always examine the anatomical snuff box for swelling or tenderness. Many times, even with negative x-rays, a fracture of the carpal navicular will be present. Should there be snuff box tenderness, the hand, wrist, and forearm are immobilized in a gauntlet type of cast for 10 to 14 days, and additional x-rays are obtained at that time to definitely establish the diagnosis (Fig 6). Subluxations and dislocations about the wrist do occur. If there is no associated fracture, four weeks of immobilization after reduction is usually adequate.

The carpometacarpal joints are stabilized mainly by their ligamentous structure. First-degree sprains

should be treated symptomatically. Second-degree injuries should be immobilized for a period of three weeks and third-degree injuries should be immobilized for six weeks. Some of these may require open reduction. Metacarpophalangeal joint injuries are extremely common and there will often be damage to the volar capsule as well as the collateral ligaments. The thumb is extremely vulnerable due to its position on the hand. The mechanism of injury is important in establishing an accurate diagnosis. Localization of tenderness and swelling is helpful in determining which part of the joint has been injured. Routine x-rays should always be obtained and stress x-rays should be made in the event there is any question as to the extent of the injury (Fig 7). Complete ruptures of the ulnar or radial collateral ligaments of the thumb are usually treated by surgical repair. Improper treatment of these types of injuries results in capsular redundancy, recurrent dislocation, and, later, degenerative arthritis. All sprains of these joints should be immobilized for two to three weeks. Third-degree sprains with dislocation should be splinted in 30° of flexion for a period of three weeks following reduction. Occasionally, general anesthesia is necessary to achieve reduction. It is also possible for mechanical entrapment to occur necessitating an open reduction. In the reduction of metacarpophalangeal joint

**A****B**

Fig 6—X-ray of wrist. *A*. Initial x-ray thought to be negative. *B*. X-ray 14 days later showing a fracture of the carpal navicular. (From O'Donoghue, *Treatment of Injuries to Athletes*, p 203, 1970, with permission from author and W. B. Saunders Company.)

dislocations, the force should not be applied longitudinally. Instead, it should be applied in an upward direction with the physician's finger pushing distally at the base of the proximal phalanx. The flexion is then carried out and relocation usually occurs.

The proximal interphalangeal joints are most often injured by hyperextension, causing volar plate damage, or abduction and adduction injuries, causing collateral ligament damage. All sprains of this joint should be splinted. Abnormal motion should be determined and in the event that this is in question stress films again will be found to be helpful. Surgical repair of third-degree injuries is sometimes indicated. The distal interphalangeal joint is most often injured by a football or baseball striking the end of the finger and causing acute flexion. The extensor tendon is avulsed from its insertion at the base of the distal phalanx. The patient will be unable to completely extend the distal phalanx and may develop a swan neck type of mechanical derangement. X-rays may show an avulsion fracture. This injury can be treated

conservatively with a malleable aluminum splint bent in the position seen in Figure 8. The splint should be changed weekly and should remain snugly applied to the finger for a period of six weeks.

Sprains about the hip are uncommon because of the great ligamentous strength. Strains, however, are quite common and occur most often at the ischial tuberosity, which is the site of origin of the biceps and semitendinosus tendons; the pubic ramus, where the adductor longus and gracilis tendons originate; the lesser trochanter, which is the insertion of the iliopsoas; and the greater trochanter, which is the insertion of the gluteus medius. Knowing the mechanism of injury can be extremely helpful in making the diagnosis. The physician should carefully palpate and pay particular attention to the point of maximum tenderness. X-rays should always be made since avulsion fractures may be seen in these areas. Treatment consists of rest and analgesics, although severe avulsions might require surgical repair. Activity should be avoided until healing is complete. Complications



Fig 7—Positive stress x-ray showing rupture of the ulnar collateral ligament of the thumb. (From O'Donoghue, *Treatment of Injuries to Athletes*, p 228, 1970, with permission from author and W. B. Saunders Company.)

Was there giving away, popping, or locking of the joint, and was it associated with immediate or delayed swelling? In examining the knee joint, the physician should carefully note any deformities, the condition of the skin, and the point of maximum tenderness. The extent and location of swelling and the presence of an effusion should also be determined. Manipulation is then carried out to check the range of motion, determine whether or not any motions are painful, and to note abnormal motion in the medial, lateral, anterior, posterior, and rotary planes. The knee should first be examined in extension. One hand stabilizes the medial femoral condyle, while the other exerts pressure against the lower tibial area (Fig 9). This maneuver checks the medial capsule, the medial collateral, and to some extent the anterior cruciate ligaments for stability. The joint is then flexed 30° and the same maneuver is carried out. Instability in flexion indicates a more isolated injury to the medial collateral ligament. Reverse the procedure to test the lateral collateral ligament and capsule. With the patient on his or her back, and the knees flexed to 90° , the foot is then fixed under the examiner's leg in a neutral position and both hands

such as chronic bursitis, excessive calcification, and nonunion of the avulsed fragment can occur. First- and second-degree strains about the thigh are relatively common and are usually not significant as long as they are properly diagnosed and treated protectively. Third-degree strains, or complete ruptures of the quadriceps or hamstring muscles, usually require surgical repair.

The knee is one of our most vulnerable joints. Anatomically, it is unstable and receives its stability from the anterior and posterior cruciate ligaments, the joint capsule, and the superficial and deep medial and lateral collateral ligaments. Again, knowing the mechanism of injury will prove to be invaluable in making an accurate diagnosis. The physician also wants to know how severe the injury appeared to be initially. Was the injured person able to bear his weight? Did he note a deformity at the time of injury?



Fig 8—Malleable finger splint shaped and applied for the treatment of "mallet finger."



Fig 9—Checking the stability of the medial knee joint in extension.

are placed over the medial and lateral tibia, just below the knee joint. Force is then exerted anteriorly and posteriorly. Abnormal anterior laxity indicates anterior cruciate instability while abnormal posterior laxity indicates posterior cruciate instability. In addition, the foot should be externally rotated and stabilized against the table by the examiner's leg and, again, the anterior-posterior force should be applied. Rotary or anterolateral instability is indicative of medial capsular disruption, although tears of the anterior cruciate and medial collateral ligaments increase the rotary instability (Fig 10). The uninjured knee should always be compared with the injured side, since many individuals have loose knee joints that are not considered to be pathologic. Routine x-rays should be obtained and occasionally a tunnel view will be important in determining whether or not an osteochondral fracture has occurred. Stress x-rays are extremely helpful in deciding whether or not third-degree tearing has occurred (Fig 11).

In first-degree knee injuries, good results are usu-

ally obtained by treating the joint with rest, compressive dressings, ice packs, and elevation. Second-degree sprains initially are treated by aspirating the hemarthrosis, if one exists, occasionally injecting an anesthetic and hyaluronidase, and then applying compressive dressings and ice packs. Later the knee can be immobilized in a plaster cast. Restoration of muscular tone should never be overlooked during the latter phases of treatment. Third-degree injuries are best treated by surgical repair if the joint opens more than ten degrees with stress. Neglecting such an extensive injury causes extreme disability to the patient and will invariably result in the premature development of traumatic osteoarthritic changes as well as severe functional weakness.

Minor sprains of the patella are not common. Should these occur, symptomatic treatment will usually suffice. Third-degree injuries result in capsular tearing and dislocation either medially or laterally of the patella. Most often, this occurs to the lateral side with rupture of the medial capsule. On examination,



Fig 10—The slocum test for rotary instability (see text).

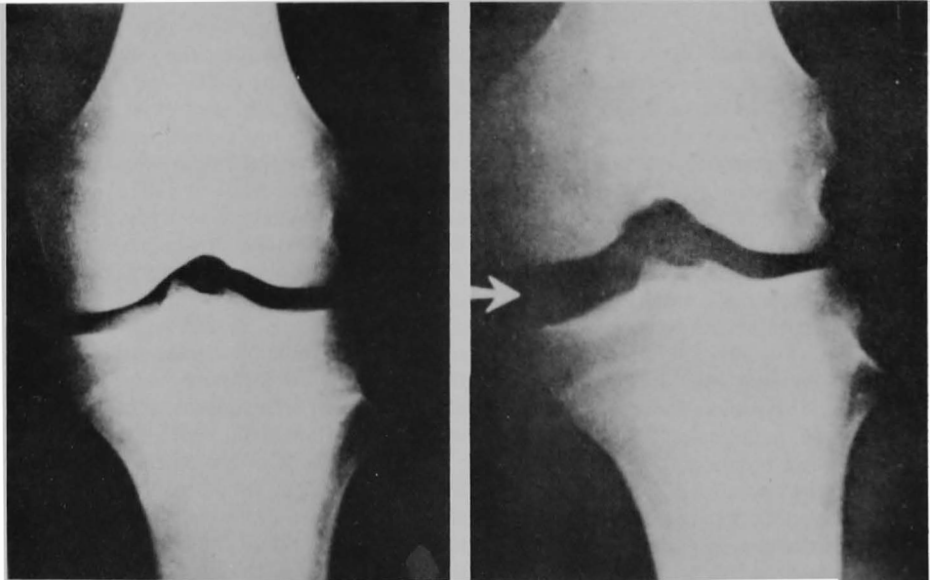


Fig 11—Positive stress x-ray indicating third-degree tearing of the medial collateral ligament (arrow). (From O'Donoghue, *Treatment of Injuries to Athletes*, p 385, 1970, with permission from author and W. B. Sanders Company.)

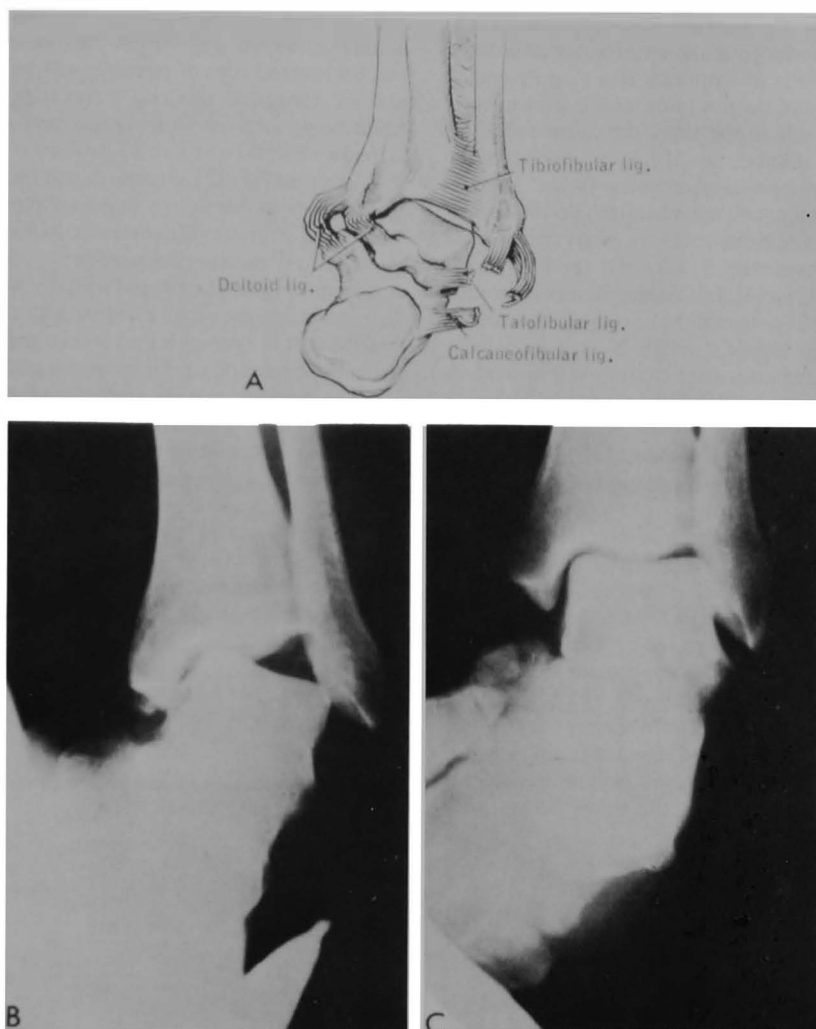


Fig 12—A. Diagrammatic illustration of the deranged ligaments of the lateral ankle joint; B and C. Comparable x-ray appearance with stress. (From O'Donoghue. *Treatment of Injuries to Athletes*, p 466, 1970, with permission from author and W. B. Sanders Company.)

the physician cannot help but note the gross deformity about the knee joint if the patella has not been reduced. After reduction, the physician should pay particular attention to the presence of tenderness and swelling about the medial aspect of the joint. This is suggestive of tearing of the medial capsule. Immobilization in a plaster cylinder cast for six weeks is the treatment of choice. Inadequate treatment often results in poor capsular healing and recurrent dislocations. Should the dislocation become recurrent,

little can be gained by continued immobilization after the second or third dislocations.

Strains of the leg are common and present no particular problem in management unless they happen to be third degree, such as rupture of the Achilles tendon. Surgical repair is then indicated.

Sprains of the ankle are one of the most common injuries seen. The severity of the injury is often not appreciated and, as a result, treatment, if any, is often inadequate. It is helpful to appreciate some of the

anatomy of the ligamentous structures about the ankle joint in order to make an accurate diagnosis. On the medial side, the primary retaining ligaments are the deltoid and the posterior talotibial ligaments. On the lateral side of the joint, the calcaneofibular, the anterior talofibular, and the anterior tibiofibular ligaments are the primary retaining forces. Posteriorly, the tibiofibular and the posterior talofibular ligaments serve as additional anchors. Inversion sprains make up approximately 85% of the sprain-type injuries to the ankle joint. In first-degree ankle sprains, the physician will note minimal swelling, tenderness, and no apparent loss of strength. X-rays are usually negative. Symptomatic treatment only is indicated. A second-degree injury with partial tearing will present as a rather painful joint associated with moderate swelling, which should be well localized, and loss of strength. Again, x-rays are usually negative. Treatment should be vigorously pursued and if a hemarthrosis is present, aspiration may be indicated. Occasionally, hyaluronidase can be of value when injected into the joint or hematoma. Compressive dressings, ice, elevation, and protection from weight bearing is suggested as the initial phase of treatment. Subsequently, a cast should be applied for a period of four weeks and the ankle should be protected by strapping for an additional two weeks. A third-degree ankle sprain is a serious and significant injury and if not properly managed will result in considerable disability for the patient. When this injury occurs, pain

and swelling will be marked. The patient will be unable to tolerate any weight bearing and on examination marked loss of strength will be apparent as well as abnormal motion. Stress x-rays, with the ankle being stressed in inversion and eversion, are extremely helpful and will be positive if the injury is third degree (Fig. 12). Fractures will occasionally be associated with this injury. In the younger individual, many third-degree ankle sprains are treated by surgical repair. If nonsurgical care is in order, a third-degree sprain may be treated initially with the same regimen as for the second degree with the exception that the cast is applied for six weeks and the ankle is protected by strapping for an additional two weeks.

The management of sprains and strains is not difficult. A history of the mechanism of injury is invaluable in determining what structures might have been injured. Careful examination with particular attention being paid to abnormal motion is essential. Stress x-rays should always be made if there is a question as to whether or not the injury is severe enough to be classified as a third-degree injury. Once the injury has been graded, management presents no particular problem.

Figures 2 and 5 are reproduced with permission from Gray's *Anatomy of the Human Body*, 29th edition, 1973, p 321 and p 333, respectively.

Figure 3 is reproduced with permission from the *Journal of Bone and Joint Surgery* (47-B:32-35, 1965).

SCRIPTA MEDICA



WALTER E. NANCE, M.D., PH.D.

*Professor of Medicine and Pediatrics
Professor and Chairman
Department of Human Genetics
Medical College of Virginia
Virginia Commonwealth University*

The Birth of a New Department at MCV/VCU—Human Genetics

PETER MAMUNES, M.D.

Associate Professor of Pediatrics and Human Genetics, Co-director, Clinical Genetics Center, Medical College of Virginia, Health Sciences Division of Virginia Commonwealth University, Richmond, Virginia

In recognition of the central role of genetics in the health professions, the MCV/VCU School of Basic Sciences and Graduate Studies substantially enlarged its commitment to this discipline by creating a new Department of Human Genetics as of September 1, 1975, under the chairmanship of Walter E. Nance, M.D., Ph.D., an internationally renowned medical geneticist. Dr. Nance comes to Richmond from Indianapolis where he was Professor of Medicine and Medical Genetics at the Indiana University School of Medicine. Here at MCV/VCU, Dr. Nance will also have joint appointments in both the Departments of Medicine and Pediatrics.

This event is the culmination of a successful 37-year effort to develop a genetics program at the Medical College of Virginia. One can be quite certain that when Dr. Roscoe D. Hughes first assumed the chairmanship of the newly formed one-man Department of Biology and Genetics in the School of Pharmacy in 1938, he could not have foreseen the explosion of knowledge in genetics that would occur during the next four decades. Through his efforts alone, genetics was first taught to medical students in 1947 and then to dental students in 1950. The faculty expanded to two in 1948. By 1965, the Department had grown to five members to meet the ever-increasing demand for instruction in genetics, and research programs had begun dealing with *Drosophila*, laboratory mice and human twins. The first advanced degree in genetics was awarded in 1953, and since that time, more than a dozen master and doctoral degrees in genetics have been granted, with research being directed by faculty members Richard Cribbs in biochemical genetics, Ives Townsend in population genetics, and

Frederick J. Grundbacher in immunogenetics, as well as Dr. Hughes.

At the time of Dr. Hughes' retirement as chairman in 1970, the Department of Biology and Genetics was administratively reorganized into a Program of Human Genetics because of the increasing demand for instruction in human genetics by all health professions in the School of Basic Sciences and Graduate Studies. Doctor Richard Cribbs served as chairman of the new program and changed its emphasis by recruiting a faculty member with special expertise in human cytogenetics and by encouraging active collaboration with faculty members in the Departments of Biology, Biophysics, Microbiology, Orthodontics, and Pediatrics. To fulfill an important need for physicians and residents of the State, a Genetics Counseling Clinic was initiated in February, 1971, in collaboration with the Department of Pediatrics. Because of an increasing case load the frequency of the clinic sessions was subsequently increased from once to twice monthly in 1973, and financial support was sought and obtained from the Virginia Capital Area Chapter of the National Foundation-March of Dimes. The burgeoning of other research and service programs in human genetics in the MCV/VCU complex including the Tay-Sachs carrier detection program in the Departments of Pediatrics and Pathology, the sickle-cell screening program in the Department of Medicine, the transplantation genetics program in the Department of Surgery, the metabolic and cytogenetic services in the Department of Pediatrics, and the amniocentesis program in the Department of Obstetrics and Gynecology over the past few years has clearly demon-

strated the need for a more coordinated effort in human genetics, and provided further impetus for the expansion of the Human Genetics program to a full departmental status.

Dr. Nance was previously the principal investigator of the Indiana University Human Genetics Center grant, one of ten such awards made by the National Institute of General Medical Science to support research in genetics. At Indiana, Dr. Nance directed a broad range of research in human genetics including studies of human twins and their families, clinical and population studies of hereditary deafness, and studies dealing with the delineation of new genetic syndromes, genetic linkage, and human biochemical genetics. A graduate of the University of the South and Harvard Medical School, Dr. Nance became interested in genetics during his medical

housestaff training at Vanderbilt, and subsequently obtained a Ph.D. in Genetics in 1968 from the University of Wisconsin, under the direction of Dr. Oliver Smithies.

Initially, Dr. Nance plans to resume and expand the pioneering twin studies begun by Dr. Hughes two decades ago. Other immediate concerns include strengthening and coordinating the cytogenetics and prenatal diagnosis programs and expansion of the genetic counseling program. The Departmental offices and laboratories are housed in 5000 square feet on the 11th floor of Sanger Hall, while outpatient activities are conducted in Randolph Minor Hall, which has recently been renovated with support from the local chapter of the March of Dimes. Additional faculty members are being recruited to help achieve the expanded goals of the new Department.

Volume Eleven

TABLE OF CONTENTS

1975 • Number One

<i>Advances in Obstetrics and Gynecology</i> Introduction LEO J. DUNN, M.D.	4
---	---

Modern Management of Diabetic Pregnancies ELSIE R. CARRINGTON, M.D.	5
--	---

The Endocrinologic Evaluation of Amenorrhea CHARLES B. HAMMOND, M.D. R. HERBERT WIEBE, M.D. LEE TYREY, PH.D. DAVID W. SCHOMBERG, PH.D.	8
--	---

Amenorrhea Due to Defects in Steroid Biosynthesis H. OLIVER WILLIAMSON, M.D. RAJESH S. MATHUR, PH.D.	15
--	----

Treatment of Amenorrhea: When Pregnancy Is and Is Not Desired JOHN A. BOARD, M.D.	33
--	----

Patient Factors Limiting Success of Cancer Detection DEAN R. GOPLERUD, MD.	37
---	----

Radical Hysterectomy for Carcinoma of the Cervix JOHN J. MIKUTA, M.D.	41
--	----

Abstracts of Papers Presented at the 46th Annual McGuire Lecture Series	45
---	----

1975 • Number Two

Nuclear Medicine—Part I

Introduction KLAUS RANNIGER, M.D.	56
--------------------------------------	----

Radiopharmaceutical Production and Quality Control JERRY I. HIRSCH, PHARM. D.	57
--	----

Interpretation of Cerebral Dynamic Perfusion Studies FRANK H. DELAND, M.D.	70
---	----

Diagnostic Use of Radionuclides in Diseases of the Thyroid ALTON R. SHARPE, JR., M.D.	75
--	----

Evaluation of Thyroid Nodules—Hot and Cold MELVIN J. FRATKIN, M.D.	78
---	----

The Diagnosis and Treatment of Carcinoma of the Thyroid RICHARD H. KIRKLAND, M.D.	84
--	----

1975 • Number Three

Nuclear Medicine—Part II

Introduction KLAUS RANNIGER, M.D.	100
--------------------------------------	-----

Scintiphography in Evaluation of Renal Transplants HALCOTT T. HADEN, M.D.	101
--	-----

Use of Bone Scanning and Skeletal Radiography in the Diagnosis of Bone Metastasis JAY C. MALL, M.D. PAUL B. HOFFER, M.D.	108
--	-----

Radionuclide Imaging Evaluation of the Patient with Trauma LETTY G. LUTZKER, M.D. LEONARD M. FREEMAN, M.D.	116
--	-----

Radioimmunologic Methods for Diagnosis of Malignant Diseases PAUL B. HOFFER, M.D.	128
--	-----

Pitfalls and Artifacts in Nuclear Imaging Studies LEONARD M. FREEMAN, M.D.	131
---	-----

1975 • Number Four

New Dimensions of Problems in Office Practice

Introduction FITZHUUGH MAYO, M.D.	154
--------------------------------------	-----

Differential Diagnosis of Hypertension W. Andrew Dickinson, Jr., M.D.	155
--	-----

Practical Office Therapy of Hypertension 159
A. JARRELL RAPER, M.D.

Some Practical Thoughts on Medical Malpractice 164
ROBERT L. HARRIS, J.D.

Asthma in Adults 170
JAMES P. BAKER, M.D.

Sprains and Strains 174
ROBERT W. WADDELL, M.D.

Scripta Medica

The Birth of a New Department at MCV/VCU 187
Human Genetics
PETER MAMUNES, M.D.

TABLE OF CONTENTS FOR VOLUME ELEVEN 189

INDEX TO VOLUME ELEVEN 191

Volume Eleven

SUBJECT INDEX

- ADRENAL HYPERPLASIA**
amenorrhea due to defects in steroid biosynthesis, 15
- ANGIOGRAPHY**
see Nuclear Imaging
- APYROGENICITY**
radiopharmaceutical production and quality control, 57
- ASTHMA**
in adults, 170
- BLOOD PRESSURE**
differential diagnosis of hypertension, 155
- BONE IMAGING**
see Nuclear Imaging
- BONE NEOPLASMS**
use of bone scanning and skeletal radiography in the diagnosis of bone metastasis, 108
- BRAIN IMAGING**
see Nuclear Imaging
- BRAIN TRAUMA**
see Trauma
- CANCER**
see Neoplasms under specific subject
- CARCINOEMBRYONIC ANTIGEN ASSAY**
see Radioimmunoassay
- CARCINOMA**
see Neoplasms under specific subject
- CERVIX NEOPLASMS**
diagnosis and management of cervical intraepithelial neoplasia (abstract), 45
radical hysterectomy for carcinoma of the cervix, 41
- CHEST TRAUMA**
see Trauma
- CONTAMINATION, RADIOCHEMICAL**
radiopharmaceutical production and quality control, 57
- DIABETES**
modern management of diabetic pregnancies, 5
- DRUG THERAPY**
asthma in adults, 170
practical office therapy of hypertension, 159
- ENDOMETRIAL NEOPLASMS**
control of endometrial cancer (abstract), 45
- ENDOCRINOPATHY**
endocrinologic evaluation of amenorrhea, 8
- ESTRIOL, URINE**
modern management of diabetic pregnancies, 5
- FEAR**
patient factors limiting success of cancer detection, 37
- GENITOURINARY TRAUMA**
see Trauma
- GOITER**
see Thyroid Disease
- HORMONE THERAPY**
amenorrhea due to defects in steroid biosynthesis, 15
diagnosis and treatment of carcinoma of the thyroid, 84
endocrinologic evaluation of amenorrhea, 8
evaluation of thyroid nodules—hot and cold, 78
treatment of amenorrhea: when pregnancy is and is not desired, 33
- HYPERTENSION**
differential diagnosis of , 155
practical office therapy of , 159
- HYPERTHYROIDISM**
see Thyroid Disease
- HYPOTHYROIDISM**
see Thyroid Disease
- HYSTERECTOMY**
radical for carcinoma of the cervix, 41
- INSULIN THERAPY**
modern management of diabetic pregnancies, 5
- KIDNEY IMAGING**
see Nuclear Imaging
- LIGAMENTOUS INJURY**
sprains and strains, 174
- LIVER TRAUMA**
see Trauma
- MEDICAL MALPRACTICE**
some practical thoughts on , 164
- MUSCULOTENDINOUS INJURY**
sprains and strains, 174
- NEOPLASM DETECTION**
patient factors limiting success of cancer detection, 37
- NODULES**
see Thyroid Disease
- NUCLEAR IMAGING**
evaluation of thyroid nodules—hot and cold, 78
interpretation of cerebral dynamic perfusion studies, 70
pitfalls and artifacts in nuclear imaging studies, 131
radionuclide imaging evaluation of trauma, 116
scintiphotography in evaluation of renal transplants, 101
use of bone scanning and skeletal radiography in the diagnosis of bone metastasis, 108
- NUCLEAR REGULATORY COMMISSION**
radiopharmaceutical production and quality control, 57
- OLIGOMENORRHEA**
endocrinologic evaluation of amenorrhea, 8
- PAPANICOLAOU CYTOSMEAR**
diagnosis and management of cervical intraepithelial neoplasia (abstract), 45
- PATIENT EDUCATION**
lack of in factors limiting success of cancer detection, 37
- PHYSICIAN-PATIENT RELATIONS**
some practical thoughts on medical malpractice, 164
- PREGNANCY**
modern management of diabetic pregnancies, 5
treatment of amenorrhea: when pregnancy is and is not desired, 33
- RADIOCHEMICAL PURITY**
radiopharmaceutical production and quality control, 57

- RADIOGRAPHY, SKELETAL**
use of bone scanning and skeletal radiography in the diagnosis of bone metastasis, 108
- RADIOIMMUNOASSAY**
diagnostic use of radionuclides in diseases of the thyroid, 75
radioimmunologic methods for diagnosis of malignant diseases, 128
- RADIOISOTOPES**
diagnosis and treatment of carcinoma of the thyroid, 84
diagnostic use of radionuclides in diseases of the thyroid, 75
evaluation of thyroid nodules—hot and cold, 78
pitfalls and artifacts in nuclear imaging studies, 131
radionuclide image evaluation of the patient with trauma, 116
scintiphotography in evaluation of renal transplants, 101
use of bone scanning and skeletal radiography in the diagnosis of bone metastasis, 108
- RADIONUCLIDES**
see Radioisotopes
- RADIATION THERAPY**
diagnosis and management of cervical intraepithelial neoplasia (abstract), 45
- diagnosis and treatment of carcinoma of the thyroid, 84**
radical hysterectomy for carcinoma of the cervix, 41
- THYROID DISEASE**
diagnostic use of radionuclides in diseases of the thyroid, 75
evaluation of thyroid nodules—hot and cold, 78
- THYROID IMAGING**
see Nuclear Imaging
- THYROID NEOPLASMS**
diagnosis and treatment of carcinoma of the thyroid, 84
evaluation of thyroid nodules—hot and cold, 78
- THYROIDITIS**
see Thyroid Disease
- TRAUMA**
radionuclide imaging evaluation of the patient with . . . , 116
- ULTRASONICS**
diagnosis and management of cervical intraepithelial neoplasia (abstract), 45
evaluation of thyroid nodules—hot and cold, 78
- VASCULAR TRAUMA**
see Trauma
- SCANNING**
see Nuclear Imaging
- SCINTIPHOTOGRAPHY**
see Nuclear Imaging
- SKELETAL TRAUMA**
see Trauma
- SPLIEN TRAUMA**
see Trauma
- SPRAINS**
and strains, 174
- STERIOD DEFECTS**
amenorrhea due to defects in steroid biosynthesis, 15
- STRAINS**
sprains and . . . , 174
- SURGERY**
diagnosis and management of cervical intraepithelial neoplasia (abstract), 45

Volume Eleven

AUTHOR INDEX

- BAKER, JAMES P., M.D., 170
BOARD, JOHN A., M.D., 33
- CARRINGTON, ELSIE R., M.D., 5
- DELAND, FRANK H., M.D., 70
DICKINSON, W. ANDREW, JR., M.D., 155
DUNN, LEO J., M.D., 4
- FRATKIN, MELVIN J., M.D., 78
FREEMAN, LEONARD M., M.D., 116, 131
- GOPLERUD, DEAN R., M.D., 37
GUSBERG, SAUL B., M.D., 45
- HADEN, HALCOTT T., M.D., 101
HAMMOND, CHARLES B., M.D., 8
HARRIS, ROBERT L., 164
HIRSCH, JERRY I., PHARM. D., 57
HOFFER, PAUL B., M.D., 108, 128
- KIRKLAND, RICHARD H., M.D., 84
- LUTZKER, LETTY G., M.D., 116
- MALL, JAY C., M.D., 108
MAMUNES, PETER, M.D., 187
MATHUR, RAJESH S., PH.D., 15
MAYO, FITZHUGH, M.D., 154
MIKUTA, JOHN J., M.D., 41
- RANNIGER, KLAUS, M.D., 56, 100
RAPER, A. JARRELL, M.D., 159
- SCHOMBERG, DAVID W., PH.D., 8
SHARPE, ALTON R., JR., M.D., 75
- TYREY, LEE, PH.D., 8
- UPTON, RICHARD T., M.D., 45
- WADDELL, ROBERT W., M.D., 174
WIEBE, R. HERBERT, M.D., 8
WILLIAMSON, H. OLIVER, M.D., 15



A service to medical education from A. H. Robins:

Excerpted from Volume 2

of the
G.I. Series

on physical examination
of the abdomen:

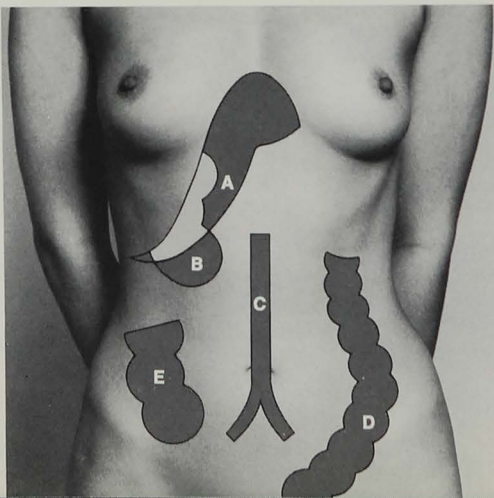
The A. H. Robins G.I. Series consists of six booklets, designed to provide a quick, yet comprehensive review of basic procedures and practices in G.I. medicine—with particular emphasis on the physical examination as performed in the office or at bedside. If you have teaching responsibilities, limited quantities are available: Part 1—*Inspection*, Part 2—*Palpation*, Part 3—*Percussion*, Part 4—*Auscultation*, Part 5—*Abdominal Pain* and Part 6—*Differential Diagnosis of Abdominal Disorders*. Write to: The Medical Department, A. H. Robins Company, 1407 Cummings Drive, Richmond, Virginia 23220.



Normally palpable organs:

the edge of the liver descending, on inspiration, below the costal margin (A); the lower pole of the right kidney (B); the abdominal aorta (C); the descending colon and the sigmoid (D); the ascending colon (E); and occasionally the bladder (though rising of this organ beyond the pubis does not necessarily indicate disease).

Impossible to outline, unless diseased, distended or enlarged: the gallbladder, pancreas, stomach, small intestine, transverse colon and spleen.





A service to medical education from A. H. Robins:

Excerpted from Volume 2
of the

G.I. Series

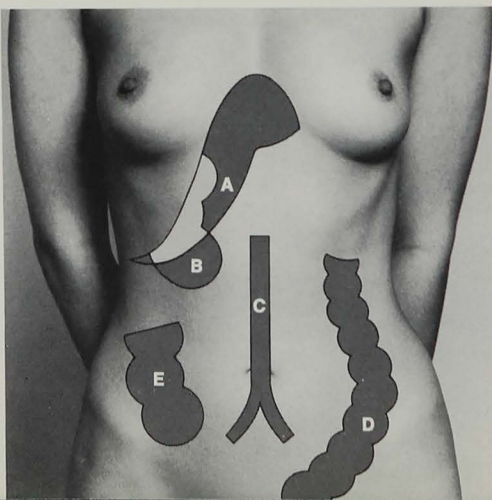
on physical examination
of the abdomen:

The A. H. Robins G.I. Series consists of six booklets, designed to provide a quick, yet comprehensive review of basic procedures and practices in G.I. medicine—with particular emphasis on the physical examination as performed in the office or at bedside. If you have teaching responsibilities, limited quantities are available: Part 1—*Inspection*, Part 2—*Palpation*, Part 3—*Percussion*, Part 4—*Auscultation*, Part 5—*Abdominal Pain* and Part 6—*Differential Diagnosis of Abdominal Disorders*. Write to: The Medical Department, A. H. Robins Company, 1407 Cummings Drive, Richmond, Virginia 23220.



Normally palpable organs:

the edge of the liver descending, on inspiration, below the costal margin (A); the lower pole of the right kidney (B); the abdominal aorta (C); the descending colon and the sigmoid (D); the ascending colon (E); and occasionally the bladder (though rising of this organ beyond the pubis does not necessarily indicate disease).



Impossible to outline, unless diseased, distended or enlarged: the gallbladder, pancreas, stomach, small intestine, transverse colon and spleen.

Spasm reactor? Donnatal!

	each tablet, capsule or 5 cc. teaspoonful of elixir (23% alcohol)	each Donnatal No. 2	each Extentab
psycamine sulfate	0.1037 mg.	0.1037 mg.	0.3111 mg.
opine sulfate	0.0194 mg.	0.0194 mg.	0.0582 mg.
psicine hydrobromide	0.0065 mg.	0.0065 mg.	0.0195 mg.
enobarbital	(1/4 gr.) 16.2 mg.	(1/2 gr.) 32.4 mg.	(3/4 gr.) 48.6 mg.

(irritating; may be habit forming)

Brief summary. Adverse Reactions: Blurring of vision, dry mouth, difficult urination, and flushing or dryness of the skin may occur on higher dosage levels, rarely on usual dosage. Contraindications: Glaucoma; renal or hepatic disease; obstructive uropathy (for example, bladder neck obstruction due to prostatic hypertrophy); or hypersensitivity to any of the ingredients.

A-H-ROBINS A. H. Robins Company, Richmond, Virginia 23220



Spasm reactor? Donnatal!®

	each tablet, capsule or 5 cc. teaspoonful of elixir (23% alcohol)	each Donnatal No. 2	each Extentab
scopolamine sulfate	0.1037 mg.	0.1037 mg.	0.3111 mg.
atropine sulfate	0.0194 mg.	0.0194 mg.	0.0582 mg.
phenobarbital	0.0065 mg.	0.0065 mg.	0.0195 mg.
phenobarbital	(1/4 gr.) 16.2 mg.	(1/2 gr.) 32.4 mg.	(3/4 gr.) 48.6 mg.
Warning: may be habit forming)			

Brief summary. Adverse Reactions: Blurring of vision, dry mouth, difficult urination, and flushing or dryness of the skin may occur on higher dosage levels, rarely on usual dosage. Contraindications: Glaucoma; renal or hepatic disease; obstructive uropathy (for example, bladder neck obstruction due to prostatic hypertrophy); or hypersensitivity to any of the ingredients

A-H-ROBINS

A. H. Robins Company, Richmond, Virginia 23220

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Tension and anxiety states, somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology, spasticity caused by upper motor neuron disorders, athetosis, stiff-man syndrome, convulsive disorders (not for sole therapy).

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence. In pregnancy, lactation or women of childbearing age, weigh potential benefit against possible hazard.

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

If there's good reason to prescribe for psychic tension.



When, for example, despite counseling
tension and anxiety continue to produce
distressing somatic symptoms

Prompt action is a good reason to consider Valium® (diazepam)

2-mg, 5-mg, 10-mg tablets

ROCHE

Roche Laboratories
Division of Hoffmann-La Roche Inc.
Nutley, N.J. 07110