An In-Training Examination for Residents in Family Practice

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An in-training examination for family practice residents has been developed and used in a regional network residency program over the past three years. The most striking result has been the strong preference expressed by residents for question-specific feedback in order to facilitate learning after taking the examination. A well-designed in-training examination has the potential to meet both individual resident and program goals as an additional measure of resident performance and growth, as well as of the effectiveness of teaching in the various curricular areas. In-training examinations for residents are in use by 12 other specialties in medicine, and have been well-accepted by program directors and residents. A nationally-sponsored in-training examination for family practice residents is needed which includes maximal teaching capability through comprehensive and specific feedback.

All who have been involved with family practice residency training in recent years have recognized the need for improved methods of measuring growth in resident performance as well as evaluating the quality of specific areas of resident training within a program. One approach to measurement is an in-training examination. Such an examination has been developed for residents in 12 other specialty disciplines,[1-10] but an in-training examination for family practice residents has not yet been developed by national organizations in this specialty. In response to this deficit, efforts have been directed during the last three years at the University of California at Davis to develop and test such an examination in the context of a regional network residency program in central and northern California. This paper will report our experience, both positive and negative, with the development and use of an in-training examination, summarize the experience of other specialties with in-training examinations for residents, and suggest future directions for a more effective national approach in family practice.

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Methods

Our goals were to develop and test an in-training examination for family practice residents which would provide confidential feedback on individual performance to each resident, sample cognitive skills across the breadth of family practice, provide residents with an opportunity to focus learning in potential deficit areas, and provide program directors with feedback of *group data* as an indirect measure of teaching effectiveness in various parts of the program.

Over 5,000 examination questions were screened from a variety of sources, primarily published test booklets in family practice and other specialties. A number of 35 mm slides were gathered from audiovisual programs obtained for the developing Network Teaching Bank, which has been described in a previous paper.[11]

As work on test item identification proceeded, a criterion group including faculty and practicing family physicians in equal numbers was organized. This group met to rate and determine those questions which were considered critical to the knowledge base of every family physician. By this process an examination was constructed in two parts: the first part containing 105 items using multiple-choice question formats, and a second part of 24 pictorial questions similar to those used by the American Board of Family Practice examination. The examination was designed for a two-hour testing period, which could be readily scheduled in each program on an annual basis. Patient management problems using branching answers and erasure or invisible ink methods were

not used due to the cost and time of development and the minimal dividend for profiling purposes in terms of time spent by the examinee on such questions.

The in-training examination comprised five major categories: general medicine, pediatrics, obstetrics-gynecology, surgery, and behavioral sciences. The number of questions in each area ranged from 15 to 53, with the greatest emphasis placed on medicine and the least in the behavioral sciences. It was possible to further classify each question in a major area into one or more of the following minor categories: (1) allergy; (2) cardiology; (3) clinical pharmacology; (4) dermatology; (5) emergency medicine; (6) geriatrics; (7) gynecology; (8) infectious diseases; (9) metabolism and endocrinology; (10) neurology; (11) obstetrics; (12) ophthalmology; (13) orthopedics; (14) otolaryngology; (15) preventive medicine; and (16) urology. The minimum number of test questions in any minor category was set at five for profiling and feedback purposes, with some minor categories having up to 15 test questions.

The examination was first administered in the summer of 1974 to a total of 37 residents from three component programs within the residency network. During the same summer the examination was also given to 81 family physicians attending the department's annual Family Practice Refresher Course. Confidential feedback was provided to each examinee for his or her main score on Parts One and Two, a total score, and scores for each major category and all minor categories. Each examinee was able to compare his or her performance against that of all other residents and practicing family physicians taking the examination. No individual scores were made available to program directors, who were given grouped data only.

The examination was revised by deleting non-discriminatory items using a Chi-square method suggested by Wilson et al,[12] and new questions were added. A new Part Two was developed and the total examination was again reviewed by the criterion group before its use in the summer of 1975, when it was administered to 37 residents and 52 family physicians attending the 1975 Family Practice Refresher Course. The revised examination consisted of 100 multiple-choice questions and 20 slide questions. Since residents had expressed their desire for additional feedback beyond their individual profiles, specific questions were discussed at the end of the testing session based on requests covering specific items raised by residents. About 20 percent of the questions were reviewed in this manner.

The examination has been further revised for 1976, and this year includes a formal method of feedback for each question missed. This includes a specific statement of the content of the test question and a reference to a readily available text or audiovisual program defending its answer (Table 1). This approach is being taken in response to a continuing desire by residents for more specific and helpful feedback.

Results

The two in-training examinations allowed for comparative analysis on several parameters. First, the reliability of the tests could be evaluated using the Kuder-Richardson formula 21.[13] This formula is used to determine the internal consistency of each question within the examination. It is a measure of how each item is representative of the total examination. In the first year, a KR21 of .82 was achieved for the total test, which indicates an acceptable level of internal consistency. In the second revised test, an even higher KR21 of .86 was achieved, probably due to improving item performance using Chi-square as discussed above.

A second analysis concerned residents as compared with practicing family physician performance. Table 2 shows the results of these compared performances for the first testing year. A significant difference at the .05 level or beyond using a t-test was not noted for any of the groups. However, an absolute progression of higher scores between first, second, and third-year residents was noted. Residents as a group scored higher than family physicians attending the 1974 Family Practice Refresher Course.

Table 3 shows the results of these compared performances for the second year. A significant difference at the .05 level or beyond using the t-test was noted when all residents were compared against family physicians who attended the course and completed the examination. Interestingly, second and third-year residents improved greatly in the second year of testing over first-year residents and practicing family physicians. It is possible that residents in the second and third year scored higher because of experience with the test in the previous year. The few points of difference in family physician and first-year resident scores between 1973-1974 and 1974-1975 can probably be accounted for by the standard error of the examination, as neither group had prior experience with the examination.

In terms of both individual and program goals of the in-training examination for family practice residents involved in this pilot project, our experience has been mixed. On the positive side, we have demonstrated that a reliable and valid examination can be prepared within a single institution if sufficient resources are directed to the effort. Perhaps our most important outcome, however, has been the strong desire expressed loud and clear by the residents that feedback be more specific and constructive to learning. On the negative side, we have been impressed with the generally low priority assigned to learning by examination on the part of many residents compared with their pressing responsibilities in patient care which they may view as more real learning opportunities. Even though we were successful in making available a fairly reliable examination, valid in its content using a criterion group, short in duration, and assuring total confidentiality for each examinee, the residents have generally internalized their feedback and it is uncertain how much subsequent learning has taken place in areas of identified weakness. Follow-up by residents in areas of identified needs would probably be greater if program directors were to review examination results on a one-to-one basis with each resident. It is also anticipated that specific feedback by the content of each missed question including related references will generate sufficient interest by many residents to actively pursue specific learning. Our overwhelming experience to date indicates that an in-training examination will

only be successful to the extent that it *teaches.*

In-Training Examinations in Other Specialties

A recent study by the American Board of Medical Specialties explored the use of in-training examinations by all specialties in medicine.[10] It was found that 12 specialties currently use such examinations. The first resident in-training examination was established in 1961 in orthopedic surgery. Two other specialties (neurological surgery and dermatology) established in-training examinations during the 1960s, and nine more have initiated examinations since 1970. All 12 specialties which have started to use in-training examinations have continued to use them. Table 4 summarizes the major features of in-training examinations by specialty.

We have followed up with residency program directors in each of these specialties in order to obtain further information about their use of in-training examinations. A thumbnail sketch by each specialty shows both commonalities and individual variations.

Orthopedic Surgery – This examination was initially developed by the American Board of Orthopedics and in 1972 was turned over to the sponsorship of the American Academy of Orthopedic Surgeons. The examination has been offered annually for the past 15 years. Increasingly used by residents and programs over the years, it became required in 1975 for all residents on an annual basis. Examination results are returned to program directors, and are reported by percentile and subscores, including comparisons with the national group. A taxonomy has been developed for profiling of results in a number of areas, such as recall, problem-solving, and overall competence. Program directors discuss examination results on an individual basis with residents.

Neurosurgery – This in-training examination was established in 1966 by the American Board of Neurosurgery, working in conjunction with the National Board of Medical Examiners. It usually is required in postgraduate years 2, 3, 4, 5, and 6 for those programs with five-year programs. Feedback of examination results, as in orthopedic surgery, is directed to the program director, who shares the individual results with each resident. Results are broken down into percentiles, compared with national results by year, and include subcategories. Since 1974, the examination has served as Part I for Board certification.

Dermatology – This in-training examination is prepared and administered by the American Board of Dermatology. The examination itself is the previous year's Board examination, and is given every other year on an elective basis by program. It can be taken by residents in postgraduate years 2 and 4, or in postgraduate year 3, depending on the year the resident entered the program. Individual residents cannot take the examination electively since it must be administered through a program. Feedback of results includes subscore percentiles with comparison with group results. Program directors share results with individual residents at their option.

Obstetrics-Gynecology – This examination is sponsored by the Council on Residency Education of Obstetrics and Gynecology (CREOG), working in conjunction with the National Board of Medical Examiners. It is elective by program, and can be taken in postgraduate years 1, 2, 3, and 4. Feedback of results is both to program directors and to residents who took the examination. Results are tabulated in much the same way as for other in-training examinations except that each printout also includes content areas for missed questions (eg, "low forceps").

Ophthalmology – This examination is sponsored by the American Academy of Ophthalmology and Otolaryngology through the College Assessment Program. The examination is elective by program for postgraduate years 2, 3, and 4, but there is increasing interest in requiring all residents to take it. Feedback is both to program directors and to residents, and includes subscore percentiles and national group results. It is anticipated that senior-resident in-training examinations will qualify as Part I of the Board examination starting in 1977.

Otolaryngology – This examination is also sponsored by the American Academy of Ophthalmology and Otolaryngology and is prepared by the Committee on Continuing Education in Otolaryngology. The examination is elective by program and is given in postgraduate years 3, 4, 5, 6, and 7. Feedback methods are similar to those for Ophthalmology.

Pediatrics – This examination is sponsored by the American Board of Pediatrics working in conjunction with the National Board of Medical Examiners. It is elective by program for postgraduate years 2 and 3. Feedback is both to program directors and to residents and also includes subscores and comparisons to national results. This is presently the only specialty requiring a passing grade before the examinee is Board-eligible.

Physical Medicine and Rehabilitation – This examination is sponsored by the American Academy of Physical Medicine and Rehabilitation. It is elective by program for postgraduate years 2, 3, and 4. Feedback is direct to residents taking the examination, and may be returned to program directors if the residents so choose by signing a permission slip. Results for postgraduate years 2 and 3 are grouped together, while postgraduate year 4 results are profiled separately. Results are otherwise reported in much the same manner as in other specialties, including subscores and national comparisons.

Plastic Surgery – The sponsor of this in-training examination is the Educational Foundation of the American Society of Plastic and Reconstructive Surgery, working in conjunction with the National Board of Medical Examiners. The examination is elective by program, and is offered every other year for residents in postgraduate years 4, 5, and 6. Results include subscores and national comparisons and are returned to program directors who usually discuss results with residents on an individual basis.

Anesthesiology – This examination is jointly sponsored by the American Society of Anesthesiologists and the American Board of Anesthesiology, working in conjunction with the National Board of Medical Examiners. It is elective by program. The examination is sometimes taken only once by the resident, usually in either postgraduate year 2 or 3, although some programs are requiring it on an annual basis. Feedback is to residents, who may give permission for results to be returned to program directors. Break-

Table 1. Question-Specific Feedback for In-Training Examination

Question No.	Content Area	References
6	Immunization procedures in children (esp. use of rubeola vaccine)	Krupp MA, Chatten MJ: Current Medical Diagnosis and Treatment. Los Altos, Calif, Lange Medical Publications, 1974, p 959
19	Treatment of cavernous hemangioma in infant	Sauer GC: Manual of Skin Diseases. Philadelphia, JB Lippincott, 1973, pp 272-274
20	Diagnosis of aspirated foreign body in lung	Conn HF, Rakel RE, Johnson TW: Family Practice. Philadelphia, WB Saunders, 1973, pp 836-838
30	Side effects of reserpine	Physicians Desk Reference, ed 29. Oradell, NJ, Medical Economics Co, 1975, pp 1042-1043
71	Diagnosis of perennial vasomotor rhinitis	Ryan RE, et al: Synopsis of Ear, Nose and Throat Diseases. St. Louis, Mo, CV Mosby, 1970, pp 157-159
99	Interpretation of T4 column tests	Wallach J: Interpretation of of Diagnostic Tests. Boston, Mass, Little, Brown and Company, 1974, pp 316-317

Table 2
Comparative Scores of Residents and Family Physicians – 1974 Examination

Group	Number	Mean Score
Physicians at course	81	74
All residents	36	78
First-year residents	13	74
Second-year residents	14	76
Third-year residents	8	81

down of results is similar to most other in-training examinations. Starting in 1977, the examination will be Part I of the American Board of Anesthesiology as the first step to Board certification.

Surgery – This examination is sponsored by the American College of Surgeons, working in conjunction with the National Board of Medical Examiners. It is elective by program, and is given in postgraduate years 3, 4, and 5. Feedback is both to program directors and to residents, and is otherwise similar to most other specialties.

Urology – This examination is prepared and administered by an independent group on an elective basis by program. The American Board of Urology is currently evaluating this approach. It is normally offered in postgraduate years 2, 3, and 4. Results are reported individually by percentile with a comparison by year of residency with all other residents in the country who took the examination. Feedback is to program directors, who may discuss results with individual residents.

Discussion

In connection with his work with a self-assessment examination for residents in psychiatry, Woods views the "ideal" examination for residents in training in this way:[8]

> An ideal examination for residents in training would accomplish both individual and program goals. Knowledge should be assessed relative to peers (those within a resident's program and perhaps a national pool) and to the educational goals and teaching activities of the residency program. An ideal examination would not only demonstrate general areas of strength and weakness, but would aid in the establishment of priorities. Since the best examination is one that teaches, the ideal examination would result in specific new learning as well as the unlearning of erroneous data, concepts or beliefs. . . . In the best of circumstances, the examination would foster an attitude of self-motivated desire for periodic review and repair that would persist throughout professional life.

Review of the experience of the 12 other specialties currently using in-training examinations for residents appears to validate the capability of these examinations to address both individual and program goals. It is impressive that all specialties who have become involved with in-training examinations have continued to use them, and that there is a trend toward increasing use in many specialties and even required use by some specialties. Many program directors feel that examination results provide an additional useful measure of resident performance as well as the capability of their programs to teach in specific areas of resident training. Residents generally welcome the opportunity to measure their progress, compare their performance with national results, and better prepare for Board certification.

Our own experience within the

Table 3
Comparative Scores of Residents and Family Physicians — 1975 Examination

Group	Number	Mean Score
Physicians at course	52	75
All residents	37	83
First-year residents	11	78
Second-year residents	13	85
Third-year residents	13	85

network residency program in the University of California Davis Affiliated Hospitals for Family Practice has demonstrated the strong interest by residents in question-specific feedback in addition to categorical breakdown of results and comparisons with group results. It is our feeling that question-specific feedback along the lines illustrated in Table 1 can facilitate increased learning by residents without compromising the future use of examination questions.

We are aware that an in-training examination for family practice is currently under development by a committee of the American Academy of Family Physicians. Based on our experience with this pilot project and that of other specialties using in-training examinations during the past 15 years, we strongly support the desirability and necessity of such a national approach. It should be possible to meet both resident and program goals through such an examination, with feedback both to program directors and to residents. Particular attention should be directed to developing an examination with maximal teaching capabilities through specific feedback. The greatest value of an in-training examination may lie in its capacity to teach and motivate toward learning in specific content areas. It is incumbent upon faculty and program directors to set a high priority on the in-training examination if it is to achieve its potential value. It would probably be wise to obtain further experience with a nationally sponsored in-training examination in family practice before considering other uses of such an examination, such as its possible role in eligibility for the Board examination itself.

Table 4. Use of Resident In-Training Examinations by Specialty

Specialty	Sponsor	Year Begun	Frequency	Comment
Anesthesiology	Board & Society	1975	Annual	Elective
Dermatology	Board	1968	Biennial	Elective
Neurological Surgery	Board	1966	Annual	Required
Obstetrics-Gynecology	Council	1970	Annual	Elective
Ophthalmology	Academy	1970	Annual	Elective
Orthopedic Surgery	Academy	1961	Annual	Required
Otolaryngology	Academy	1971	Annual	Elective
Pediatrics	Board	1971	Annual	Required for Board-eligibility
Physical Medicine and Rehabilitation	Academy	1972	Annual	Elective
Plastic Surgery	Society	1972	Biennial	Elective
Surgery	Board	1975	Annual	Elective
Urology	Independent group	1975	Annual	Elective

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Marital and Sexual Counseling in Medical Practice (2nd Edition). *D. Wilfred Abse, Ethel M. Nash, and Lois M. R. Louden. Harper and Row, Publishers, Hagerstown, Maryland, 1974, 612 pp., $14.95.*

This book is intended to improve the work of all health professionals engaged in marital and sexual counseling. Studying it and using it as a reference guide will benefit particularly those family and other primary care physicians who desire to prevent or minimize the causes of psychophysiological disturbances as well as treat the symptoms.

The book is well written by 48 qualified authorities and organized for easy readability. Many illustrative case histories are given, references are listed following each chapter, and 29 pages of double-column index are included. Appendix A is 13 pages of a practical "Marital Information Form."

The 40 chapters in the book could have been divided into seven sections under the following headings: (number of chapters in each in parentheses)

1. Understanding and treating marital and sexual disorders (12)
2. The essential functions of the physician before marriage and during early marriage (4)
3. Ages, stages, and identities in marital and sexual situations faced by the physician (6)
4. Marital and sexual components of specific illnesses or conditions (9)
5. Sexual problems of the unmarried, widowed, or divorced (3)
6. Contributions to the physician about marriage and sex from other disciplines (3)
7. The physician: his education and his personality in relation to sex and marriage (3).

A unique feature of this compendium of articles is a section entitled "Afterword" written by a professor of theology and personality. This discussion in itself constitutes a review of the book. The writer notes that 23 of the 48 contributors are psychiatrists and only one physician is identified with family practice. One portion of this section is entitled "Where is Family Medicine?" and the author goes on to state: "On the face of it, family medicine would appear to be at least one of the white hopes to achieve the objectives aimed at by the editors and authors of this book." Later, he suggests that in future editions the space now given to psychiatrist-writers should be shared equally with family physician specialists.

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Emergency Treatment and Management (5th Edition). *Thomas Flint, Jr. and Harvey D. Cain. W. B. Saunders Company, Philadelphia, 1975, 794 pp., $13.75.*

Every family physician, family practice resident, and medical student involved in emergency care should consider including this excellent reference in his or her library. It is organized in a format which expidites rapid reference through presentation of problems, complaints, principles, and procedures in emergency medicine. In addition, an extensive index provides a ready cross-reference. These features assist one in quickly developing a differential diagnosis and formulating a plan of action. As the authors state in their preface to the first edition, they have "endeavored to outline in a rapidly available form portal-to-portal care in emergency situations."

The only suggestion this reviewer would have is that a larger number of illustrations, particularly color photographs of dermatological conditions, would be of value. Those that are included are of excellent quality and complement the text. I have already used this book on several occasions and have found the format excellent. Each condition is briefly reviewed including signs and symptoms. Then, various diagnostic studies are recommended when needed. Finally, treatment plans and recommendations are made. Because this volume is designed for rapid use in emergency situations it lacks depth, and alternate methods of treatment are often not mentioned. However, one must recognize that it is meant to complement rather than replace the standard text, and in most instances these omissions will cause no problems.

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Immediate Care of the Acutely Ill and Injured. *Edited by Hugh E. Stephenson, Jr. C. V. Mosby Company, St. Louis, 1974, 266 pp., $8.50.*

This paperback publication presents an in-depth and comprehensive study of crisis medicine. Using a narrative form based on class presentations and interspersed with frequent anecdotes and case histories, this volume covers such crisis areas as immediate care priorities, cardiopulmonary emergencies, mechanical trauma, poisoning, psychiatric emergencies, and selected specialty problems. There are also some specific comments about preventive medicine.

The vast amount of material presented is well organized and comprehensive. Since much of it is taken from class lectures, however, the presentations are too wordy and often rambling, thus making it difficult for a reader to extract specific data or information quickly and concisely. Some unique chapters, such as "Rescue and Extrication," and "Esoterica: Hiccups, Fish Hooks, Rings and Such Things," further illustrate the total comprehensiveness of this publication.

The main objective is to present crisis medicine, an area often neglected in medical education, to the medical student. This book definitely fulfills this objective and will provide excellent reading and resource material for the third or fourth-year medical student. It could also serve as a good review for the busy practicing family physician and would be an extremely valuable reference in hospital Emergency Rooms or outpatient receiving centers.

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Leg Ulcers: Medical and Surgical Management. *Henry H. Roenigk, Jr., and Jess R. Young. Harper and Row, Publishers, Hagerstown, Maryland, 1975, 265 pp., $27.50.*

The authors' purpose in writing *Leg Ulcers* was to provide an overview of the problem and a quick reference for proper diagnosis and management. They accomplished this task in a very readable, 250-page text which is well organized, thorough, and succinct.

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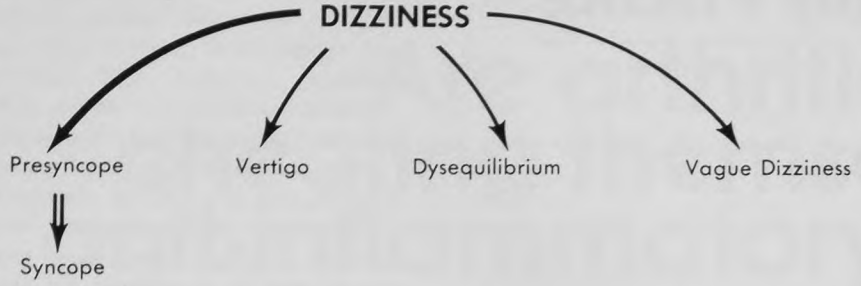

Figure 1. The four primary types of dizziness.

Table 1. Questions to Ask the Patient and Observers

1. Questions for the Patient

What were you doing during the hours and minutes preceding the blackout?
What was your situation regarding loss of sleep, ingestion of food and alcohol, and use of drugs or medications prior to the blackout?
What was your body position or posture?
What was the first thing you noticed to be wrong?
What symptoms did you experience next, in what order, and for how long?
Do you remember slumping or striking the floor?
What was the next thing you remember and what position were you in when you regained awareness?
Did you hurt yourself in the fall, did you injure your tongue or mouth, did your back or muscles ache, did you have a headache, did you lose control of your bladder or bowels?
How did you feel on awakening and how long did it take for you to feel entirely normal again? Seconds, minutes, or longer?

2. Questions for the Observers

Ask the observers to answer the above questions when appropriate.
Was there any turning of the eyes or head?
Was there any twitching or jerking of the face or extremities?
Was the skin sweaty, pale, flushed or blue?
Did the patient respond to observers in any way during the apparent unconsciousness?

position or posture, what was the first thing that appeared to be wrong, and other details summarized in Table 1.

Pathophysiology of Syncope

Cerebral blood flow is determined largely by arterial blood pressure and cerebral vascular resistance. Cerebral blood flow autoregulation is the phenomenon by which cerebral vessels automatically constrict or dilate in response to rising or falling systemic blood pressure changes. This intrinsic control mechanism maintains a virtually constant cerebral blood flow in the face of significant physiologic or pathologic fluctuations in arterial blood pressure. In healthy young adults in the upright position, the systolic blood pressure may fall to 50-60 mmHg with no significant cerebral ischemia. Below that, cerebral resistance vessels become maximally dilated and further decreases in blood pressure result in progressive decreases in cerebral blood flow. Presyncopal symptoms result and if the blood pressure continues to fall, total loss of consciousness finally occurs. In older patients with diminished capacity for cerebral vasodilation in response to relative hypotension, there may be significant cerebral ischemia with much smaller reductions in arterial blood pressure. Syncope, then, whatever its specific etiology, is due to a relatively sudden fall in cerebral blood flow to very low levels. This decrease in flow can be due, at least in part, to increased cerebral vascular resistance (eg, hyperventilation with its fall in $apCO_2$ induces [H+] changes in the interstitial space around precapillary arterioles that mediate a vasoconstriction of these resistance vessels). But usually syncope is caused by a fall in systemic blood pressure either from a loss of peripheral and/or splanchnic vascular resistance or from a decrease in cardiac output. With most causes of syncope, blood pressure rises back to the critical cerebral perfusion level as soon as the recumbent position is assumed.

If the physician can visualize the precise clinical picture at the completion of the history taking, he will usually be capable of considerable confidence in his diagnosis of syncope, or its exclusion, and often the setting will give major clues to the etiology of the spells. He can then proceed with determining the specific cause of the syncope if it is not already clear. (See Table 2.)

Neurogenic Syncope

In each of these conditions there are inadequate vasoconstrictor mechanisms.

Vasovagal (Vasodepressor, Vasomotor) Syncope

This is the common faint and it is the most common type of syncope. It occurs frequently in teenage girls and young women, but it may occur in any otherwise normal person. It is often precipitated by emotional stress or physical pain, or threat of pain, and it tends to be recurrent. The sight of a shocking accident and the sight of, or receipt of, a hypodermic needle or blood withdrawal may also evoke an attack. The primary event appears to be a sudden excess of vagal activity which produces a prompt bradycardia and decrease in cardiac output. At the same time, there is a striking decrease in splanchnic and extremity vascular resistance, and the combined result is a progressive fall in blood pressure until syncope occurs. Sometimes there is no evidence of excessive vagal activity and it appears that peripheral vasodilation alone is causative (vasomotor syncope). This condition is aggravated by fasting, poor physical conditioning, a warm environment, and excessive fatigue. A few clonic jerks often occur and occasionally an intense, generalized tonic spasm in extension results. Fecal or urinary incontinence is uncommon. Simple syncope rarely produces a rhythmic, clonic convulsion.[3]

Orthostatic Hypotension with Syncope

This type of syncope occurs in individuals who have either chronic or transient vasomotor instability. Sudden rising from the recumbent or sitting position to a sitting or standing position, or standing still for several

Table 2. Types of Syncope

I. Neurogenic Syncope
  A. Vasovagal
  B. Orthostatic Hypotension
    1. occasional normals
    2. peripheral neuropathy
    3. medications
    4. primary autonomic insufficiency
  C. Reflex
    1. micturition
    2. cough
    3. acute pain states

II. Cardiogenic Syncope
  A. Bradyarrhythmias
  B. Tachyarrhythmias
  C. Valvular Disease
  D. Myocardial Disease

minutes, results in the pooling of blood in the lower extremities and viscera due to the loss of their compensatory reflex peripheral vasoconstriction. Blood pressure falls and syncope ensues. Orthostatic hypotension occurs in otherwise normal people after physical deconditioning, especially with prolonged bed rest, fasting, and with alcohol use. Varicose veins and normal pregnancy may also be implicated, and some patients following surgical sympathectomy also suffer orthostatic hypotension with faints.

Diabetic and other neuropathies may affect the autonomic nervous system causing impotence, impaired sweating, and paralysis of vasomotor reflexes. Diminished ankle jerk reflexes and sensation in the feet should be sought on examination as evidence of a peripheral neuropathy and should suggest the possibility of a concomitant autonomic neuropathy.

Antihypertensive, antidepressant, and some other medications commonly cause orthostatic hypotension by plasma volume depletion and/or sympatholytic vasomotor effects.

Primary autonomic insufficiency may be caused by several primary degenerations of the autonomic nervous system, and a common feature of these diseases is orthostatic hypotension with syncope.[4] The Shy-Drager syndrome is a disease of the middle-aged which usually begins with sphincter disturbances and progressive orthostatic hypotension. Defective sweating is common, and tremor, rigidity and severe myoclonic jerking while falling asleep appear later in the disease. The postural hypotension is attributed primarily to degeneration of pre-ganglionic autonomic neurons in the intermediolateral cell column of the spinal cord. Both Adie's syndrome and the Riley-Day syndrome (familial dysautonomia) may be associated with orthostatic hypotension and syncope.

Reflex Syncope

Micturition syncope is usually seen in elderly males who arise from sleep to urinate and faint while voiding. Orthostatic hypotension may be a significant part of the cause, but sudden decompression of the distended bladder may induce a reflex vasodilation of the peripheral vasculature.

Cough syncope usually occurs after a vigorous paroxysm of coughing in men with chronic obstructive lung disease. It may be caused by the sudden increase in intrathoracic and intra-abdominal pressure which increases intracranial pressure and decreases cerebral blood flow. The Valsalva effect may also diminish cardiac output. A prolonged Valsalva maneuver in association with hyperventilation certainly appears to produce syncope in breath-holding spells of infancy, weight lifter's blackout, and various blackout pranks indulged in by teenagers.

Acute pain states occasionally produce syncope. These stimuli probably act by barraging the dorsal motor nucleus of the vagus nerve which then induces a prominent bradycardia and splanchnic vasodilatation. Vagal and glossopharyngeal neuralgia, gall bladder colic, perforation of a viscous and needling of body cavities are examples of this disorder. Carotid sinus syncope is discussed below. Intense vertigo and migraine headache on occasion induce syncope presumably through this reflex vagal mechanism.

Cardiogenic Syncope

Various disorders of the heart may decrease cardiac output and cause syncope.[5] Transient rhythm disturbances or valvular obstructive lesions are the most common cardiogenic causes. Presyncopal symptoms may occur without fainting, or the patient may experience immediate loss of consciousness, depending on the rapidity and magnitude of drop in the cardiac output and the posture of the patient. Frequently the patient has no cardiac symptoms associated with the syncope while at other times there may be palpitations, "skipped beats," chest pain, or other symptoms of cardiac awareness. In a normal individual the mean blood pressure must drop to a critical level or there must be a period of asystole for four or five seconds before syncope results.

Bradyarrhythmias

Marked slowing of heart rate or asystole may result from increased vagal tone and/or disturbances of the cardiac pacemaker and conduction system.[6] In normal individuals, intense fright or pain may induce sufficient vagal stimulation to produce syncope. Increased sensitivity of the carotid sinus may induce severe bradycardia or cardiac standstill in patients with the carotid sinus syndrome. Disease of the sinus node, as in the sick sinus syndrome, may result in periodic sinus arrest and bradycardia or asystole with or without associated tachyarrhythmias. This condition should be suspected in patients with unexplained sinus bradycardia, a wandering atrial pacemaker, or intermittent atrial fibrillation. These patients do not have the normal increase in heart rate expected with exercise or with the administration of atropine or isoproterenol. Periods of complete heart block may occur in patients with disease of the conduction system and result in Stokes-Adams attacks. One should be especially suspicious of this condition in those patients whose electrocardiogram shows bifascicular block, as this conduction defect progresses to complete heart block in a significant number of patients.

Tachyarrhythmias

Paroxysms of atrial tachycardia, flutter or fibrillation may result in sufficient reduction in cardiac output to cause syncope. These tachyarrhythmias may be followed by periods of marked bradycardia and may be identified only after periods of prolonged monitoring of cardiac rhythm.

Valvular Disease

Conditions causing obstruction to the flow of blood through the heart may result in syncope by various mechanisms. Aortic stenosis or hypertrophic subaortic stenosis may be associated with syncope, especially with exercise. This is due to inadequate cardiac output either from direct obstruction to outflow, myocardial ischemia, associated tachyarrhythmias or intraventricular conduction blocks. Pulmonic stenosis may also be associated with exertional syncope. Syncope associated with mitral stenosis is generally the result of associated atrial arrhythmias.

Left atrial myxomas may resemble mitral valvular disease clinically and may be associated with syncope as a result of intermittent obstruction of atrial outflow or as a result of atrial tachyarrhythmias.

Myocardial Disease

Patients with disease of the myocardium, eg, ischemia, infarction, or cardiomyopathy, may experience syncope as a result of drops in cardiac output associated with decreased myocardial contractility. Increased systemic blood flow demands or reduction in peripheral resistance may not be compensated for by increased cardiac output, and syncope results. Arrhythmias may also occur in this group and cause syncope.

Differential Diagnosis

Several disorders are commonly confused with syncope even though an accurate history usually avoids this confusion. Particularly in the absence of an accurate history, the following disorders should be considered: (1) hypoglycemia, (2) epilepsy, (3) cerebral vascular disease, (4) "drop attacks," (5) hysterical faints, and (6) hyperventilation syndrome.

Hypoglycemia typically produces confusion or behavioral abnormalities and then hunger and salivation. This is followed by sympathetic hyperactivity manifested by sweating, tachycardia, and nervousness. Obtundation progressing to coma and seizures may ensue. It is unusual for hypoglycemia to produce a relatively abrupt and transient loss of consciousness, and thus this condition is not generally confused with syncope.

Epileptic seizures are usually different from syncope in that they have an immediate onset, they occur in the day or night and while active or supine, they often result in injury and they are commonly associated with incontinence. Clonic convulsive activity often lasts for several minutes, and after the ictus there is often confusion, drowsiness, and headache. While no single aspect of a seizure differentiates it from syncope, the total sequence of events usually does. Temporal lobe (psychomotor, limbic) seizures may be confused with presyncope or syncope because of autonomic symptoms and signs and because the patient loses awareness during the seizure (even though he may not convulse). Usually there is an aura at the onset of the ictus. While it may resemble the symptoms of presyncope, it commonly produces a "dreamy" or confused state and patients often experience hallucinations, illusions involving themselves or their environment and/or unusual feelings of "déjà vu." The patient frequently carries out automatic activity during the ictus which is generally stereotyped for that patient. Akinetic seizures are brief attacks of unconsciousness with loss of muscle tone, and although these seizures are rare and occur primarily in children, they may be confused with syncope.

Cerebral vascular disease is rarely a cause of transient loss of consciousness that might be confused with syncope. Subarchnoid hemorrhage may cause temporary unconsciousness, but it usually has an explosive onset with severe headache, and the patient is typically left with obtundation and neck stiffness and often prominent neurological deficits. A major cerebral embolism or thrombosis may produce transient unconsciousness, but residual neurological deficits are present. Transient cerebral ischemic attacks involving the carotid artery distribution almost never produce transient unconsciousness. While dizziness is one of the cardinal signs of brainstem ischemia, true syncope is uncommon. When dizziness results from brainstem ischemia, it is associated with tinnitus, deafness, diplopia, dysarthria, extremity paralysis or numbness, or some other symptoms of brainstem ischemia in the majority of cases. Therefore, cerebral vascular disease usually produces many associated symptoms of focal cerebral dysfunction that readily distinguish it from syncope.

"Drop attacks" are a puzzling phenomenon. They occur usually after the sixth decade and are characterized by a sudden drop to the ground with no apparent loss of consciousness. The patients may bruise their knees but they are usually otherwise unhurt and are able to stand immediately. While the cause is not known, many believe there is basilar artery insufficiency producing transient ischemia to that part of the reticular formation that regulates postural tone. These patients often have attacks for many years without other evidence of brainstem ischemia, and most authors believe that a diagnosis of "vertebral-basilar insufficiency" cannot be made with confidence.

Hysterical faints or swoons must be differentiated from syncope, particularly emotion-induced vasovagal syncope. Hysterical faints occur dramatically in the presence of others and are not associated with pallor, diaphoresis, or weakening or slowing of the pulse. They draw attention to the hysterical personality seeking secondary gain through this and other physical complaints. There may be prolonged periods of "unresponsiveness" with resistance to passive opening of the eyelids, bizarre posturing, or resistance to movement of the limbs.

Hyperventilation infrequently causes syncope. However, it does cause presyncope. This faintness, along with anxiety, dyspnea, palpitations and paresthesias of the distal extremities and perioral area in a setting of overventilation, constitutes the hyperventilation syndrome. It is usually caused by acute anxiety. Sometimes vasodepressor mechanisms are superimposed and syncope results.

Evaluation of Syncope (See Table 3.)

All patients middle-aged or older who have a syncopal episode without an easily identifiable benign cause should probably be admitted to the hospital.

Emphasis is placed on a detailed history because it is the most important aspect of the evaluation. In reflex syncope such as cough, micturition, or acute pain states, the history is virtually diagnostic. The same can be said of vasovagal syncope, although a search should be made for factors contributing to orthostatic hypotension. Gentle massage of one and then the other carotid sinus with monitoring of the pulse rate and rhythm plus the blood pressures is valuable in evaluating possible carotid sinus syncope.

In orthostatic hypotension with syncope, the mechanism is proven by obtaining the blood pressure in the supine, sitting, and standing position. In some patients vasomotor tone will not fail immediately upon standing, so it is important to record the blood pressure immediately after standing and then at one, three, and five minutes or longer. A careful examination of reflexes and sensory and motor function should be made, looking for evidence of associated peripheral neuropathy. If it is found, a specific etiology should be sought. A drug screen and alcohol level should be considered. Central venous pressure can be measured when volume depletion is a concern. Neurologic consultation would seem prudent in a patient with any of the widespread neurological abnormalities suggesting one of the primary nervous system diseases associated with degeneration of the autonomic nervous system.

Two or three minutes of hyperventilation should be observed in any patient suspected of the hyperventilation syndrome. Normal people may become intensely dizzy with this maneuver, so it is important that the patient's spontaneous episodes be mimicked by deep breathing before one attributes etiologic significance to the hyperventilation.

A glucose tolerance test, prolonged fasting or other provocative tests for hypoglycemia and electroencephalography for epilepsy should be reserved for those patients in whom the diagnosis of syncope is uncertain. Rarely will aortic arch and cerebral angiography need to be considered. Even if vascular abnormalities are found in elderly patients, they will not necessarily be diagnostically meaningful.

In the work-up of suspected cardiac syncope, inquiry should be made regarding the association of the syncope with exertion, dyspnea, palpitations, chest pain, or other symptoms of cardiac awareness. A history of rheumatic heart disease, coronary artery disease, or hypertension may be important. While one rarely has the opportunity to examine the patient during an episode of syncope for the presence of an arrhythmia, one should examine for abnormalities of cardiac rhythm or rate, hypertension, signs of congestive heart failure, murmurs, or carotid sinus sensitivity. The chest roentgenogram should be reviewed for evidence of congestive heart failure, chamber enlargement, or calcifications within the heart. The electrocardiogram may be normal, but susceptibility to tachy- or bradyarrhythmias is suggested by unexplained sinus bradycardia, wandering atrial pacemakers, atrial fibrillation, or various conduction blocks. Findings suggesting ischemic heart disease or chamber enlargement may give clues to the etiology of the patient's heart disease. Serial electrocardiograms or continuous rhythm monitoring over several hours may document transient rhythm disturbances.

Table 3. Evaluation of Syncope

History – often diagnostic
BP supine, sitting, standing
Examine for peripheral neuropathy
Examine for heart disease
Consider:
- Chest film for heart failure, chamber size and calcification
- ECG and rhythm monitoring
- hyperventilation
- treadmill test
- echocardiogram for chamber size and valve function
- EEG
- tests for hypoglycemia
- drug and alcohol screen
- gentle carotid massage
- central venous pressure measurement

Rarely consider:
- cardiac, aortic arch, and cerebral angiography

Treatment

The treatment of neurogenic syncope should be directed toward preventing or correcting the cause of the decreased cerebral perfusion. Vasovagal syncope is best treated by eliminating precipitating factors such as emotional excitement, fatigue, hunger, inactivity, or sedative drugs. These patients should be instructed to assume a sitting or recumbent position with the first presyncopal symptoms. The use of measures to increase peripheral resistance such as support hose or ephedrine may be useful in the patient with vasovagal syncope or orthostatic hypotension. Antihypertensive and diuretic medications should be avoided. Volume expansion through increased salt intake or fludrocortisone may be useful. Cardiac slowing associated with vasovagal syncope may be benefited by atropine. Reflex syncope associated with micturition or coughing may be benefited by having the patient avoid standing at the time of these activities.

Treatment of cardiogenic syncope is dependent on making the correct pathophysiologic diagnosis. Tachyarrhythmias may be treated in various ways depending on the seriousness of the arrhythmia. Maneuvers such as breath-holding, Valsalva or carotid sinus massage may abort supraventricular tachyarrhythmias. More serious arrhythmias may be controlled by the use of drugs such as quinidine, procainamide, digitalis, or propranolol. Electrocardioversion or insertion of a pacemaker may be necessary in other patients. Syncope due to a valvular lesion may not be relieved without surgical correction.

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Contraindications: Pregnancy or the possibility of pregnancy within three months following vaccination; infants less than one year old, except that measles-containing vaccines may be administered during the first year of life in certain populations (infants vaccinated under such conditions should be revaccinated after 12 months of age); sensitivity to eggs, chicken, chicken feathers, or neomycin, and, for rubella-containing vaccines, duck, or duck eggs or feathers; any febrile respiratory illness or other active infection; for measles-containing vaccines, active untreated tuberculosis; therapy with ACTH, corticosteroids (except as replacement therapy, e.g., for Addison's disease), irradiation, alkylating agents, or antimetabolites; blood dyscrasias, leukemia, lymphomas of any type, or other malignant neoplasms affecting the bone marrow or lymphatic systems; gamma globulin deficiency, i.e., agammaglobulinemia, hypogammaglobulinemia, and dysgammaglobulinemia.

Precautions: Administer subcutaneously; *do not give intravenously.* Epinephrine should be available for immediate use should an anaphylactoid reaction occur. Should not be given less than one month before or after immunization with other *live* virus vaccines, with the exception of monovalent or trivalent poliovirus vaccine, live, oral, which may be administered simultaneously. Vaccinations should be deferred for at least three months following blood or plasma transfusions or administration of more than 0.02 ml human immune serum globulin per pound of body weight.

Attenuated measles, mumps, and rubella virus vaccines, live, given separately, may result in a temporary depression of tuberculin skin sensitivity; therefore, if a tuberculin test is to be done, it should be administered before or simultaneously with any virus vaccine.

Measles-Containing Vaccines—Due caution should be employed in children with a history of febrile convulsions, cerebral injury, or any other condition in which stress due to fever should be avoided. The physician should be alert to the temperature elevation which may occur 5 to 12 days after vaccination. The occurrence of thrombocytopenia and purpura has been extremely rare.

Rubella-Containing Vaccines—Excretion of live attenuated rubella virus from the throat has occurred in the majority of susceptible individuals administered rubella vaccine. There is no definitive evidence to indicate that such virus is contagious to susceptible persons who are in contact with vaccinated individuals. Consequently, transmission, while accepted as a theoretical possibility, has not been regarded as a significant risk.

Adverse Reactions: To date, clinical evaluation of the combination vaccines has revealed those adverse reactions expected to follow administration of the monovalent vaccines given separately.

Measles-Containing Vaccines—Occasionally, moderate fever (101-102.9 F); less commonly, high fever (above 103 F); rarely, febrile convulsions. Infrequently, rash, usually minimal without generalized distribution. Reactions at injection site. Local reactions characterized by marked swelling, redness, and vesiculation at the injection site of attenuated live measles virus vaccines have occurred in children who received killed measles vaccine previously; the combination vaccines were not given under this condition in clinical trials.

Experience from more than 44 million doses of all live measles vaccines given in the U.S. by mid-1971 indicates that significant central nervous system reactions such as encephalitis, occurring within 30 days after vaccination, have been temporally associated with measles vaccine approximately once for every million doses. In no case has it been shown that reactions were actually caused by vaccine. The Center for Disease Control has pointed out that "a certain number of cases of encephalitis may be expected to occur in a large childhood population in a defined period of time even when no vaccines are administered. A survey conducted in New Jersey in 1965 showed that 2.8 cases of encephalitis (of unknown cause) occurred per million children, ages 1-9 years per 30-day period." † However, the Center for Disease Control has analyzed the reported reactions following measles vaccines and pointed out that "the clustering of cases in the period 6 through 13 days after inoculation as well as the recovery of measles virus (probably the vaccine strain) from the CSF of one patient does suggest that some of these cases may have been caused by the vaccine." The risk of such serious neurological disorders following live measles virus vaccine administration remains far less than that for encephalitis with measles (one per thousand reported cases).

Rubella-Containing Vaccines—Adverse reactions may include fever and rash, mild local reactions such as erythema, induration, tenderness, and regional lymphadenopathy; thrombocytopenia and purpura; allergic reactions such as urticaria; and arthritis, arthralgia, and polyneuritis.

Moderate fever (101-102.9 F) occurs occasionally, and high fever (103 F) occurs less commonly. Rash occurs infrequently and is usually minimal without generalized distribution. Encephalitis and other nervous system reactions have occurred very rarely.

Transient arthritis, arthralgia, and polyneuritis vary in frequency and severity with age and sex, being greatest in adult females and least in prepubertal children. Symptoms relating to joints (pain, swelling, stiffness, etc.) and to peripheral nerves (pain, numbness, tingling, etc.) occurring within approximately two months after vaccination should be considered as possibly vaccine related. These symptoms need not be associated with other features of rubella, such as fever, rash, and lymphadenopathy. In prepubertal children, the symptoms have generally been mild and of no more than three days' duration, with an incidence of less than 1 percent for reactions that would interfere with normal activity or necessitate medical attention. In teen-age girls, the rates of reactions are somewhat higher but probably do not exceed 5 to 10 percent. In women, the rates are greater and may exceed 30 percent; the symptoms in older females tend to be more prominent and of longer duration, rarely persisting for a matter of months, but have not generally interfered with normal activity. There is, at present, no evidence that the joint involvement or neuritis accompanying infection with either natural rubella or the attenuated viruses predisposes to any of the known chronic arthritic or neurologic diseases. Transient arthralgia and arthritis in nonimmune males may occur; however, as in the natural disease, the incidence is expected to be lower than in women.

Mumps-Containing Vaccines—Parotitis. Rarely, purpura and allergic reactions such as urticaria. Very rarely, encephalitis and other nervous system reactions. With the monovalent mumps vaccine, mild fever occurs occasionally, and fever above 103 F is uncommon.

Shipment, Storage, and Reconstitution: During shipment, to insure that there is no loss of potency, the vaccine must be maintained at a temperature of 10 C (50 F) or less. Before reconstitution, store vaccines at 2-8 C (35.6-46.4 F) and *protect from light.* Use only diluent supplied to reconstitute vaccines. If not used immediately, store reconstituted vaccines in a dark place at 2-8 C (35.6-46.4 F), and discard if not used within eight hours.

Color change: The usual color of the vaccine when reconstituted is pinkish to red due to the presence of phenol red, a pH indicator. Some vaccine which has been shipped in dry ice may exhibit a variation in color when reconstituted because carbon dioxide has been absorbed from the dry ice. This vaccine, if crystal clear on reconstitution, is acceptable for use whether it is red, pink, or yellow.

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M-R-VAX® (Measles and Rubella Virus Vaccine, Live, MSD)—Single-dose vials of lyophilized vaccine, containing when reconstituted not less than 1,000 $TCID_{50}$ of measles virus vaccine, live, attenuated, and 1,000 $TCID_{50}$ of rubella virus vaccine, live, expressed in terms of the assigned titer of the FDA Reference Measles and Rubella Viruses, and approximately 25 mcg neomycin.

MUMPSVAX® (Mumps Virus Vaccine, Live, MSD)—Single-dose vials of lyophilized vaccine, containing when reconstituted not less than 5,000 $TCID_{50}$ of mumps virus vaccine expressed in terms of the assigned titer of the FDA Reference Mumps Virus, and approximately 25 mcg neomycin.

Each of these vaccines is supplied as a single-dose vial packed with a disposable syringe containing diluent and fitted with a 25-gauge, 5/8" needle, and as a box of 10 single-dose vials with an accompanying box of 10 diluent containing disposable syringes with affixed needles.

For more detailed information, consult your MSD representative or see full prescribing information.

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†National Communicable Disease Center, Encephalitis Surveillance Report, 1965 Annual Supplement, July 1, 1966.