PHYSICAL DIAGNOSIS

SOME OLD

SOME NEW

Internal Medicine Grand Rounds

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This is perhaps a back to basics grand rounds. The "low tech" end of our profession has fallen on hard times for many reasons. The extraordinary precision of some of the newer diagnostic techniques, especially imaging, is compelling. The continued pressure from the public to dispel any level of uncertainty lest they assume the litigant's role and the demand on all of us that we see more patients in fewer time blocks have all combined to denigrate the physical examination.

There are some equally compelling reasons why we might wish to focus on the physical examination as we prepare physicians for the 21st Century. My goal today is to review some of these reasons for renewed attention to the physical examination. We will review some old and some new physical findings. We will focus briefly on the hand examination as a particularly rich source of clinical information. Along the way, I will present some unknowns for your challenge and to keep this fun; and I will hazard a prediction about an additional tool for the examination of the future. The focus throughout will be on the physical examination, not the history. This with the caveat we all accept that the majority of the diagnoses in medicine can be made with a carefully constructed interview with the patient and this percentage goes even higher following the physical examination. In the vast majority of clinical interactions between doctors and patients, the laboratory is merely confirmatory[1].

WHY THE PHYSICAL EXAMINATION?

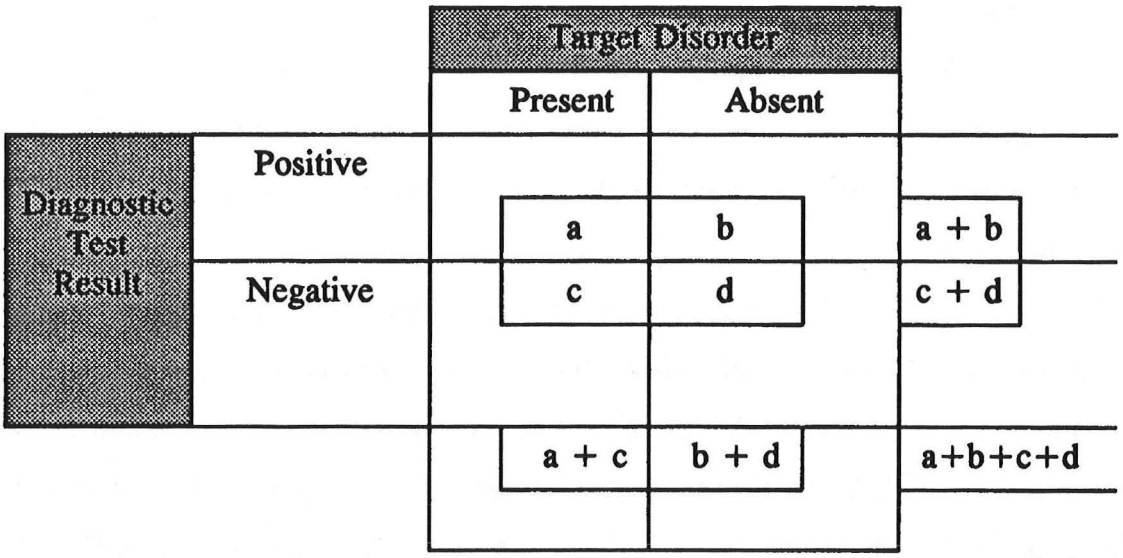
1. To Make a Diagnosis
2. To Exclude a Diagnosis
3. As a Pre-Test
4. To Touch the Patient
5. Because it's Fun

Certain diagnoses can only be made by the physical examination. There are, for instance, no simple laboratory tests for the diagnosis of early hypertension, an extremely common malady. To be certain, some early diagnoses, depend on the laboratory - diabetes for instance.

Equally, the physical examination can exclude a diagnosis. In the comatose patient, the visualization of retinal vein pulsations absolutely excludes increased intracranial pressure as the reason for the coma[2]. This might permit a quick lumbar puncture for the diagnosis of meningitis rather than delay for the imaging procedure. This, of course, depends on the reliability of the examiner - more on this later.

The use of the physical examination as a pre-test is becoming the most compelling reason why more attention is merited. Some of the lexicon on the new health care is instructive. Technology assessment, outcomes research, clinical epidemiology, decision analysis, and cost effectiveness research are largely economically driven. The physical examination is cheap - relatively. It is quite likely that part of the new "Managed Care" will include algorithms which prohibit the use of costly diagnostic tests absent certain findings from the physical examination which increase the likelihood of a positive finding.

There is statistical enforcement of this notion. More than 200 years ago, a Presbyterian minister in Tunbridge Wells England, Reverend Thomas Bayes worried with this problem and his treatise, published post-humously, demonstrated that the accuracy of the test depended to a large extent on the prevalence of the condition being tested for[3].



For Accuracy

a/(a+c) = sensitivity.

SnNout: When Sensitivity is high, a Negative test result rules out the target disorder.

d/(b+d) = specificity.

SpPin: When Specificity is high, a Positive test result rules in the target disorder.

a/(a+b) = positive predictive value or post-test probability of having the target disorder among patients with positive test results.

d/(c+d) = negative predictive value or post-test probability of not having the target disorder among patients with negative test results.

c/(c+d) = post-test probability of having the target disorder for patients with negative test results.

(a+c)/(a+b+c+d) = prevalence or pre-test probability of having the target disorder.

Sensitivity/(1-specificity) = likelihood ratio (of having the target disorder) for a positive test result = [a/(a+c)]/[b/(b+d)].

(1-sensitivity)/specificity = likelihood ratio (of having the target disorder) for a negative test result = [c/(a+c)]/[d/(b+d)].

Post-test probability of the target disorder (expressed as odds) = pretest probability of the target disorder (expressed as odds) x likelihood ratio for the test result.

For precision (and k)

Observed Agreement: (a+d)/(a+b+c+d) =

Expected Agreement:

Cell a: ([a+b]x[a+c])/(a+b+c+d) =

Cell d: ([c+d]x[b+d])/(a+b+c+d) =

(expected a + expected b)/(a+b+c+d) =

Agreement Beyond Choice = k:(observed agreement - expected agreement)/(100% - expected agreement =

Conventional Levels of k: 0.0-0.2, slight; 0.2-0.4, fair; 0.4-0.6, moderate; 0.6-0.8, substantial; 0.8-1.0, almost perfect.

While this table is generally applied to laboratory tests, it is applicable to any test including the physical examination. It has been reproduced large size so that you can reproduce it or cut it out for study and use. Note especially, the probability or diagnostic accuracy of the test and the precision or a measure of independence from chance[4].

Working with these formulae, it will become obvious that a very sensitive (few false negatives) and very specific (few false positives) test is markedly influenced in its diagnostic precision by the prevalence of the disorder in the population being tested. A test of 90% specificity and 90% sensitivity if applied to a population with an incidence of the disease of 10% has a diagnostic accuracy of 80%. If, however, the prevalence is 0.1% the diagnostic accuracy falls to 4.3%[5]. (The legislators who mandated PKU testing in newborns should have been told this.)

The physical examination can be very effective in selecting the patients more likely to have a disorder and vastly enhance the diagnostic accuracy of the subsequent more costly tests. For this purpose, it is important that we favor tests which are more sensitive than specific - that is, we can tolerate false positives but we get nervous with false negatives who would not then get definitive examination.

ACCURACY OF THE PHYSICAL EXAMINATION FOR ASCITES

	Sensitivity			Specificity		
Sign	Cummings et al	Simel et al	Cattau et al	Cummings et al	Simel et al	Cattau et al
Flank dullness	---	0.80	0.94	---	0.69*	0.29
Bulging flanks	0.72	0.93	0.78	0.70	0.54	0.44
Shifting dullness	0.88	0.60	0.83	0.56	0.90*	0.56
Fluid wave	0.53	0.80	0.50	0.90	0.92	0.82
Puddle sign	---	0.43	0.55	---	0.83	0.51

*Test for heterogeneity suggests these values are significantly better across studies ($p < .01$)

This table illustrates several studies that attempt to define the sensitivity and specificity of several physical findings for ascites. The conclusions here are first - the puddle sign is a waste of time, second, the most compelling positives are the presence of a fluid wave, shifting dullness or bulging flanks[6]. The gold standard here was ultrasound of the abdomen. (The reason it is called the gold standard is because it is expensive.) While not demonstrated in this study, it is reasonable to assume that combinations of findings enhance the diagnostic accuracy as each could be considered a pretest for the next.

Ah, you say, I'll bet those were all skilled gastroenterologists doing those exams. How would we fare if the examiners were residents or general internists. The answer is "not so well". The decline in importance given to the physical examination is reflected in poor performance in conducting the examination. A group of residents at Duke University (a small medical school on the other side of Texarkana) were tested on Harvey (the cardiac teaching model). They were presented with classic findings of mitral regurgitation, mitral stenosis and aortic insufficiency. The error rate was 63%[7]. I can hear John Carpenter groaning. Did they fail to learn or were they not taught? The disquieting answer is probably that the faculty failed. St. Clair and colleagues tested the faculty ability to detect errors on the part of residents. The residents were programmed to make errors and were videotaped - the error pick up by the faculty was tragic[8].

The final reasons for "Why the Physical" are; to touch the patient. There is a level of expectation that the stethoscope be laid on and that the abdomen be palpated or the throat inspected. There is also a therapeutic balm in the act. Finally, the examination can be fun. There is enormous satisfaction in establishing the diagnosis by your senses. Puzzles are where you find them and the cocktail party, bus stop, and stadium can all challenge your diagnostic skills - albeit at a

distance and without commentary or touch. What you do when you spot the enlarged thyroid in a stranger could be the subject of another grand rounds.

THE HAND AS A MIRROR OF SYSTEMIC DISEASE[9]

As part of the review of some old and some new, I would like to start with the physical examination of the hand. We will then review some findings of note for some of the specialties of internal medicine, then remind you of the physical findings of a few conditions where the diagnosis can only be easily made with the physical examination - disorders of connective tissue as the paradigm.

The hand is easy to get to and a careful examination can be rewarding. The initial handshake greeting with the patient reveals much. Clammy and cold, warm and dry, firm, limp, steady or tremulous.

Examination of the nails gives diagnostic and chronologic information.

Beau's Lines: These are horizontal depressions across several nails. The common etiology is a major event to the physiological system with a transient halt to nail growth. General anesthesia, myocardial infarction, serious trauma are common causes. Nail growth is variable but on the order of 6-8 months from cuticle to tip. The distance of the lines is a crude calendar of the event[10].

Half and Half Nails: The proximal nail is usually a dense white and the distal half a rusty brown. The lunulae disappear. This is not an extremely sensitive test but it is fairly specific for a reduced creatinine clearance. A few other causes include the administration of androgens or chemotherapy especially 5FU[11].

Terry's Nails: Easily confused with half and half nails, this finding includes a normally colored distal nail and a pale proximal nail. Unlike the half and half nail, when compressed the distal color of the Terry's nail will blanch. It is most often seen with cirrhosis, congestive heart failure and adult diabetes[12].

Muehrcke's Lines: These are pale parallel white bands in the nail which blanch when pressed. When seen, there is a high probability that the albumin is less than 2.2 grams[13].

Mee's Lines: These are non-blanching white transverse lines on several nails. They are lines of microfractures within the nail itself. They are very white and do not blanch since they are in the nail itself. They are not seen frequently but when apparent the causes may include chemotherapy, arsenic poisoning, carbon monoxide poisoning, pellagra and thallium exposure[14].

Clubbing: Patients are either clubbed or they are not. I have never seen "early clubbing" go to late clubbing. This is a disorder of the nail growth bed. There is a hyperplasia of the tissue imparting a spongy feel to the nail bed which is distorted to obliterate Lovibond's angle. The suspected culprit with all of the etiologies is platelet derived growth factor. In all cases, the digital blood flow is increased. Causes include, cyanotic congenital heart disease, cirrhosis, congenital and pulmonary disease. If clubbing is associated with full blown hypertrophic osteoarthropathy, the diagnosis is usually carcinoma of the lung. Curiously, clubbing will not occur in a paretic limb[9,15,16].

Onycholysis: This is an irregular separation of the nail from the bed at the distal end. The causes are legion. It may result from irregular growth of the nail as in hyperthyroidism or from the accumulation of debris under the nail as in psoriasis. I have found it a helpful clue for hyperthyroidism[17].

SYSTEMIC DISEASES ASSOCIATED WITH ONYCHOLYSIS

Amyloid and Multiple Myeloma
Anemia
Bronchiectasis
Carcinoma (Lung)
Erythropoietic Porphyria
Histiocytosis X
Ischemia (Peripheral)
Leprosy
Lupus Erythematosus
Neuritis
Pellagra
Pemphigus Vulgaris
Pleural Effusion
Porphyria cutanea tarda
Psoriatic Arthritis
Reiter's Syndrome
Scleroderma
Syphilis (Secondary and Tertiary)
Thyroid Disease
Yellow Nail Syndrome

Source: Nails in Systemic Disease. In: Dermatologic Clinics 3(3):467, 1985

Splinter hemorrhages: Not a very helpful clinical sign but woe be to the housestaff who neglects this in the list of negative findings. Worldwide, the most common cause of splinters is probably trichinosis. In the United States it is probably a small sliver of wood under the nail. Arguments rage about splinter representing embolic events or vasculitis. Both probably occur. While splinters represent pathology in the bed of the nail, they are dragged distally with time[18].

CAUSES OF SPLINTER HEMORRHAGES

Altitude (high)	Fungal	Pemphigus
Behçet's Syndrome	General Illness	Peptic Ulcer Disease
Blood Diseases	Hemochromatosis	Pityriasis Rubra Pilaris
Buerger's Disease	Hepatitis	Psoriasis
Cirrhosis	Histiocytosis X	Purpura
Cryoglobulinemia	Hypertension	Raynaud's Disease
Cystic Fibrosis	Hypoparathyroidism	Rheumatoid Arthritis
Darier's Disease	Irradiation	Sarcoidosis
Diabetes mellitus (seen in about 10% of patients)	Keratosis Lichenoids Chronica	Scurvy
Dialysis	Letterer-Siwe Disease	Systemic Lupus Erythematosus
Drug Reactions (in general)	Leukemia	Tetracycline
Emboli, arterial	Malignant Neoplasms	Thrombocytopenia
Endocarditis	Mitral Stenosis	Thyrotoxicosis
Eczema	Mycosis Fungoides	Trauma
Exfoliative Dermatitis	Normals	Trichinosis
	Osler-Weber-Rendu Disease	Vasculitis

Source: Nails in Systemic Disease. In: Dermatologic Clinics 3(3):466, 1985

Koilonychia: Spoon nails have not been written about much. These nails are spooned from side to side and from end to end such that a drop of water on the nail would not fall off. General inanition may lead to spoon nails. When

associated with palor of the palmar creases suggesting anemia, they may be indicative of Plummer Vinson syndrome of iron deficiency anemia and esophageal webs[19].

Periungual telangiectasia: A distortion of the capillary bed in the nail fold has been frequently associated with systemic sclerosis. In addition to an obliteration of the demarcation of the nail and bed, the capillaries will be seen to be dilated and tortuous. Nail bed capillaroscopy is a tool used by some sophisticates but is probably unnecessary. In addition to systemic sclerosis, the finding is less often seen in dermatomyositis and systemic lupus erythematosus. It is also occasionally seen in diabetes and in response to chemotherapy especially with those agents known to be vasculotoxic. It has also been reported in AIDS - as has everything else[20,21].

The palms add additional diagnostic clues.

Dupuytren's Contractures: Described more than 100 years ago, we are still learning about this fascinating disorder. It is a fibrotic condition of the palm of the hand with characteristic progressive flexion tethering of the ring and little finger. It is seen in about 6% of the male population and much less frequently in women. It's diagnostic significance falls with age. It may be inherited, but it is independent of HLA type or type I collagen genes. The most important medical condition with which it is associated is heavy alcohol consumption - a suggestion often denied by patients. It is independent of alcoholic liver disease. It is also seen in epileptics who have been on medication for a long time. The association with occupation has not been proven but is suggested. It is also seen more commonly in diabetics and curiously less commonly in rheumatoid arthritis patients. Most studies suggest reduced microcirculation to the affected tissue with a proliferation of fibroblasts and scarring[22,23].

Palmar erythema: This condition really represents deep tissue angiomas in the thenar and hypothenar eminences and shares etiologies with spider angiomata seen elsewhere. The common denominator in most cases is hyperestrinism. Cirrhosis, birth control pills, and pregnancy for instance. Less certain is the etiology of palmar erythema in mitral stenosis and in graft versus host reaction. Spiders and telangiectasia elsewhere have been noted to occur after radiation, in CRST, and hyperviscosity syndromes[24].

Great mystery #1: Why don't spider angiomata occur below the umbilicus?

Palmar arches: Approximately 3% of the population has a congenital absence of the palmar arterial arch. This together with the frequency of arterial line placement in the radial artery may account for the almost yearly loss of a hand or part of a hand following prolonged arterial monitoring. There is a simple test to determine the patency of the arch. Compress both the radial and ulnar artery with the arm elevated. Have the patient repeatedly make a fist until the hand is pale. Then, release the radial artery compression and watch the blush come to the palm. If the arch is absent the blush will not cross the midline and the ulnar side of the hand will remain pale.

Dermatoglyphics: While mostly reserved to those who deal extensively with genetic diseases, there are a few simple observations of fingerprints and hand creases worth noting. Fingerprints begin to develop in the 6th week, then involute and reappear from 11 to 17 weeks of fetal development. They spread from distal to proximal and from radial to ulnar side of the hand. This is, of course, a critical period of fetal development and major dislocations can be reflected in abnormal dermatoglyphics. Helpful clues include the presence of ulnar loops (open loops pointed to the ulnar side) on the fifth finger, a wide angle between the tri-radii of the second, fifth digit and the palmar tri-radius and the presence of single palmar crease. Ulnar loops may be an important clue to the presence of structural renal

disease such as bifid collecting systems, polycystic kidney disease, or single kidney. The single palmar crease is most often the result of fusion of all creases because of a foreshortening of the hand as seen in the various trisomies.

The Hand in Rheumatic Diseases: Many of the classic arthritides can be diagnosed by the pattern of disease in the hand.

Rheumatoid arthritis: The hallmark findings of rheumatoid arthritis can be recognized at a distance. Probably the most specific finding is the ulnar drift of the fingers. This occurs because the interosseous muscles of pronation are slightly stronger than those of supination. (This is also why screws have a right hand thread in a civilization where most people are right handed). The various subluxations are also characteristic.

Osteoarthritis: Generally easily distinguishable, there may be some confusion with the inflammatory variety where the patient's complaints of morning stiffness, swelling, and erythema may suggest rheumatoid disease. The distal location of affectation is helpful.

Psoriatic arthritis: The variety of psoriatic arthritis which affects small joints may be diagnosed in the hands. The small distal joints are most prominently involved and the diagnostic clue is, of course, the pitting of the nails and onycholysis. It may occur, by the way, with very little skin disease. Psoriasis mutilans is a rare devastating bone resorbing variety which is unmistakable.

Reiter's syndrome: With many similarities to psoriatic small joint arthritis, the hand may help with the diagnosis. Onycholysis may still occur with the skin lesion, keratoderma blenorrhagica, but there will not be nail pitting. In both psoriatic and Reiter's, there may be intense inflammatory disease of the distal interphalangeal joints.

Gonococcal arthritis: The hand or wrist is a common locus for the monarticular or pauci-articular migrating tenosynovitis of gonococcal infection. The pustule of GC also often appears on the hand or finger.

DOES THIS PATIENT HAVE CORONARY DISEASE?

One of the most common diagnostic challenges we face as internists is to determine the presence or absence of coronary artery disease. I would like to review some old and some new findings regarding the contribution of the physical examination to the answer of that question.

The recent good news is that the history and physical examination proved very good at discriminating groups of patients to high and low risk for coronary disease. This was true even when compared to treadmill testing as a screen where the careful history and physical was shown to be slightly superior[26].

While the history contributed the majority of the discriminant information, there are physical findings which aid in the diagnosis of likelihood.

Arcus senilis: This finding in men between 30 and 50 is a marker of coronary disease of the same magnitude as family history of coronary disease, hypercholesterolemia, hypertension, or smoking. If found together with hypercholesterolemia, it is strongly predictive[27].

Horizontal ear lobe crease: Horizontal ear lobe creases are not present at birth. When this crease is detected in a patient, man or woman, it is probably quite specific for the presence of coronary disease. Studies have been small in number with predictive values ranging from 70-94%. The crease is independent of smoking or lipid disorders[28,29].

Carotid artery bruits: While there has been a great deal of attention given to the proper investigation and management of carotid artery stenosis in order to prevent stroke, it has been little appreciated that the presence of carotid artery disease is

a strong predictor of coronary disease. Patients with carotid artery disease who also have diabetes, intracranial arterial disease, or detectable peripheral vascular disease even when asymptomatic for coronary disease have a very high probability of coronary lesions[30].

Physical findings of angina: The patient with chest pain prompts many interventions. The r/o MI diagnosis fills many beds. There is room for a careful physical examination to help establish the diagnosis of coronary disease as the reason for chest pain. Diminished cardiac function as a result of ischemic induced chest pain may be reflected in a paradoxical splitting of the second heart sound in the absence of a left bundle branch block, the transient appearance of an S3 (S4 if present during pain is likely to be present when the pain dissipates as well), and a short soft systolic murmur at the left sternal border of mitral regurgitation because of an ischemic papillary muscle. If these finding disappear as the chest pain abates, they represent strong evidence for coronary disease[31,32].

A murmur which does not disturb the second sound is probably benign (no reference - a personal observation).

Eye Tracking: This is both an old and new observation. In 1908, Diefendorf and Dodge described abnormal eye tracking in schizophrenia. These observations went forgotten until 1973 when Holzman rediscovered them. Schizophrenics, it appears, have difficulty with smooth tracking of slowly moving objects. Instead of a smooth sweep of the globe, there are hesitations followed by catch up saccades. These can be detected at the bedside. Although probably of little value in making the diagnosis, these findings have interesting suggestions regarding the etiology of the illness or the locus of pathology in the superior temporal sulcus. Further investigations reveal this finding in 60% of schizophrenics, 42% of severely depressed patients, and 5% of control subjects. It is also frequently seen

in first degree relatives of schizophrenics. The abnormality is putative in schizophrenics but may disappear with remission of depression[33,34,35].

Diseases of Connective Tissue: At the outset, I stated that one of the reasons for physical diagnosis was to establish a diagnosis and that there were some conditions where there is no easy laboratory or radiologic investigation which will otherwise establish the diagnosis. I have selected as paradigmatic of this category, the diseases of connective tissue.

As we continue the long history of nosology in medicine, there is a continual rolling front end of back and forth from the laboratory to the bedside. Eponymic disorders give way to more precisely named disease as we reach consensus on etiology. Connective tissue diseases are very much in this state as we close the 20th century. The literature is ripe with rancor. What, for instance, constitutes Marfan's syndrome? Is there room for a diagnosis of "marfanoid"? Dare we diagnose a patient with Ehlers-Danlos absent the demonstration of one of the very many currently described genetic mistakes? If it is not the disease today, will it be tomorrow when there is another proband with the same amino acid substitution?

At most recent count, there are more than nineteen collagen types containing more than 30 distinct polypeptide chains the genes for which are on more than 12 different chromosomes. Also holding us together are fibrillin, fibronectin, elastin, a family of proteoglycans, glycosamines and basement membranes with their families of laminin, nidogen and others.

Diseases of these parts of our basic scaffolding may indeed represent the largest category of illness. The following table lists some of the diseases proven to be, or thought to be, a result of a distortion of normal connective tissue[36,37].

Osteogenesis imperfecta
Ehlers Danlos (all nine varieties)
Marfan syndrome
Achondrogenesis
Stickler syndrome
Alport syndrome
Epidermolysis bullosa
Aortic aneurysms
Osteoporosis
Osteoarthritis

The importance to the clinician of these diseases is first to ameliorate and prevent what might be prevented and counsel where appropriate and second, to continue to supply material to the laboratories to help make sense of the nosology and hopefully the natural histories.

Marfan syndrome: The clinical presentation of classic Marfan syndrome is well known to this audience. When presented with a patient demonstrating tall stature, arachynodactyly, dislocated lens, pectus excavatum, pes planus and a blowing murmur of aortic insufficiency together with a positive family history, there is little dispute about the diagnosis.

Originally thought to be a disorder of collage, we now know that the culprit is probably fibrillin. Immunofluorescent antibodies to fibrillin show a striking diminution of this connective tissue in skin and blood vessels in more than 95% of "classic" cases[38].

The importance of establishing the diagnosis is now quite firm. Prophylactic surgery on the aorta when it reaches 55 mm saves lives as does the long term use of beta blockers to reduce the shear stress (dp/dt) on the aortic root[39]. Patients

with Marfan syndrome have a 40% reduction in life expectancy and 95% of the deaths are cardiovascular.

Ehlers Danlos syndrome: This is another of the hereditable disorders of connective tissue not readily diagnosed by anything but the physical examination or at autopsy after a catastrophic event. As with Marfan syndrome there is great clinical variability. The hallmarks are hypermotile joints and lax suspensory ligaments, skin which varies from slight thinness to marked hyperextensibility which easily tears. They may develop characteristic "cigarette paper scars", may have a tendency to easy bruising and can spontaneously rupture their aorta or gut wall. Occasional additional findings are periodontal disease, acquired kyphoscoliosis, and mitral valve prolapse.

The classifications and genetics of Ehlers Danlos are confusing for the clinician. There are perhaps more than nine defined varieties involving many different chromosomes and the inheritance may be dominant, recessive or X linked[40].

Pseudoxanthoma elasticum: Less common, but similar in its clinical dilemma, is pseudoxanthoma elasticum. Probably the most common mode of presentation to the internists is the patient with gastrointestinal hemorrhage who demonstrates angioid streaks in the retina. The disease is of the elastic tissue which becomes calcified and fractured. It is probably recessively inherited and is estimated to have a prevalence of 1 in 100-160,000 although some contend that another dominantly inherited variety exists. The cutaneous lesions of "plucked chicken" skin when present should lead to the diagnosis. The angioid streaks are ruptures in Bruch's membrane of the retina and while sensitive they are not specific since they may also be seen in Paget's disease, sickle cell anemia, and lead poisoning. Loss of central vision may occur.

Even more catastrophic is the involvement of the arterial tree. Claudication, myocardial infarctions and spontaneous vascular rupture are all recorded[41,42,43].

Care for these conditions is difficult. The variable genetics makes counseling for reproduction a guess. Beta blockers and prophylactic surgery for Marfan syndrome helps. None of these patients will have an easy time bearing children. Cigarettes are, and heavy exercise may be, contra-indicated. The tragedy is the at least biannual headline of a young athlete dying during competition.

For every one of these which carries an unequivocal diagnosis, the internist probably confronts a dozen patients with some variant. Unusual wound healing, the mild acquired scoliosis, the click murmur syndrome and the patient with hyperextensible joints. One can only hope that the careful physical examination, attention to previously undescribed findings and well documented natural histories will feed our brothers and sisters at the bench.

A Prediction for Physical Diagnosis: Bed side tools for physical diagnosis for the general internist have changed little in the last several decades. The pocket ophthalmoscope now has a green lens, the debate about the best reflex hammer continues, and a few venturesome souls have Doppler's in the office. I believe the technology now would easily permit the development of a small portable ultrasound. The requirements include small size, variable frequency transducer, and a high resolution digital screen. The following could be routine uses in the office or at the bedside.

PORTABLE ULTRASOUND

Neck masses
Thyroid nodules
Breasts
Cardiomegaly
Gallbladder
Abdominal aorta
Bladder
Uterus, adnexa
Testes

There will be a resistance from others about this invasion of turf, the concern about missed diagnoses (where is that with the stethoscope?) and the contention that this takes special training. Well, internists are for the most part educable - I can for instance tell the difference between bass and crappie on my boat fish finder. Beam me up Scottie!

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