MARFAN SYNDROME DRAWER 3A HEALTH

Abraham Lincoln's Health

Marfan Syndrome

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The Marfan Syndrome: A Study Of 5 Generations In a Kentucky Family*

Abraham M. Gordon, M.D.**

Louisville, Ky.

THIS report will attempt to define a rare disorder of connective tissue best known by its eponym, the Marfan Syndrome, as it was found in five generations of a Kentucky family.

Definition

By definition the syndrome under discussion is a heritable disease of connective tissue manifested in the eye, skeleton and cardiovascular system. Known also as arachnodactyly, a term coined by Achard[5] to describe the spider-like appearance of the fingers and toes, this name is now rarely employed. Since connective tissue is widely distributed, consisting of many diverse materials, combined into many different structures, each with distinctive properties and functions, a disordered connective tissue may produce symptoms of unlimited proportions with multi-system involvement. The degree and scope of the connective tissue changes in the Marfan Syndrome, particularly as they affect elastic tissue, determines the severity of the disease and in turn generates the symptoms peculiar to this disorder.

Skeletal abnormalities were recognized and reported by Marfan[1] in 1896. Ocular and cardiovascular derangements went unappreciated and were slowly added by other observers in later decades. Boerger[2] described ectopia lentis and included disorders of the eye as an integral part of the syndrome. Etter and Glover[3] were the first to associate dissecting aneurysm with the disease. Baer, Taussig and Oppenheimer[4] ascribed aortic aneurysm in young adults to the Marfan Syndrome.

Recognized skeletal abnormalities will include tallness, a dolichocephalic skull, a highly arched palate, lax joints, kyphosis, pigeon breast or pectus excavatum, poor muscle tone, scant subcutaneous fat, pes planus, arachnodactyly, a mobile patella, hammer toes, misshapen ears and hernia. Ocular involvement affects the lens and the suspensory ligament. Ectopia lentis, microphakia, iridodonesis and defects within the zonular fibers may be present. Described cardiovascular disorders include valvular heart disease, aortic aneurysm, medial cystic disease of the aorta, dissecting aneurysm and congestive heart failure.

The family of the propositus has been studied and the pedigree appears on next page. Direct descent from a male ancestor five generations back lists 18 individuals in the pedigree of whom six are believed to have been affected.*** All six appear in the last three generations, including the propositus, his father, an uncle, one cousin, a brother, and one niece. Of the six, examination was possible in three of these cases. All three were tall, suffered with subluxation of the optic lens, lax joints, dolichocephalic skulls and deformities of the chest and spine. Two of the three cases examined have succumbed to cardiovascular complications. Autopsy in one case was permitted and the findings are included in this report.

Case Report 1.

(Hospital #32436)

On Sept. 23, 1946, a 24-year-old unmarried white male, a baker by trade, was hospitalized

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* *Presented to the Jewish Hospital Staff on November 24, 1957, as part of the hospital program in postgraduate medical education. Minor changes have been made for publication.*

** *Instructor in Medicine, University of Louisville School of Medicine.*

*** *Available information does not implicate others in the pedigree with certainty. The appearance of two cases in the third generation makes a mutation unlikely. The disease probably existed as a form fruste in the first and second generation or else the defect was introduced by a spouse.*

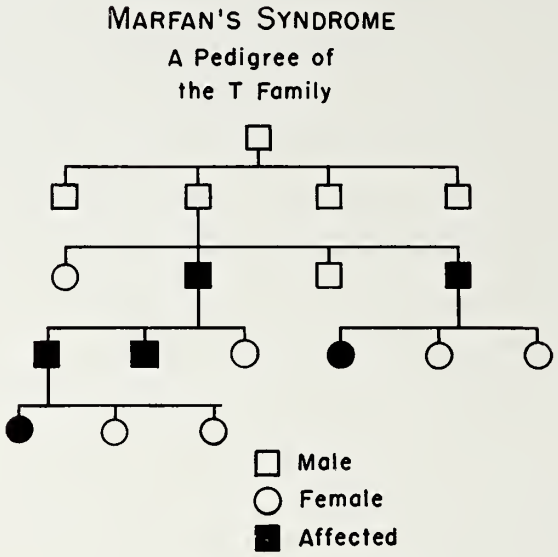
Reprinted from the Journal of the Kentucky State Medical Association, December 1959

complaining of shortness of breath and weakness. Six months before admission he noticed increasing weakness and fatigability. He gave up his job in the bakery as he was no longer able to perform his assigned duties. In July, 1946, he developed pain in the left chest which he attributed to pleurisy. Beginning in August he developed constant nausea and stated that he vomited most of what he consumed. Seven days before admission he was seen by a physician because of severe pain in the left kidney region. The patient suffered the usual disorders of childhood. He denied rheumatic fever. At 16 years of age he was told he had "skipped beats" resulting from heart disease and should take things easy. In 1930 surgery on the right eye for subluxation of the optic lens was successfully accomplished, with removal of the lens. On the same day an older brother had the identical operation in the right eye, also for ectopia lentis. The patient's father died at 44 years of age of heart disease, having suffered a lifetime with his eyes.

Physical Examination

Physical examination revealed a poorly nourished, thin white male who measured over 6 feet in height. Respiratory distress was moderate and he appeared chronically ill. His temperature measured 98.6° F, pulse 116 and the respirations 26 per minute. The blood pressure measured 120/90 mm. of Hg. The head was dolichocephalic. The right cornea (operated eye) was opaque, the eye soft, shrunken and totally blind. The left pupil reacted sluggishly to light. Upward and inward subluxation of the optic lens was seen with the ophthalmoscope. The iris appeared tremulous. The hard palate was narrow and highly arched. The neck veins were visibly pulsatile and distended. The thyroid was normal. The chest appeared moderately pigeon breasted. Rales were heard in both lung bases and persisted after cough.

The heart was enlarged to the left as the PMI was palpable in the 6th ICS at the anterior axillary line. A systolic thrust was felt to the left of the sternum. A thrill was palpable at the apex. A systolic murmur was audible over the whole precordium, loudest at the apex and along the left sternal border. At times a faint diastolic murmur was reported in the aortic area. The liver edge descended two fingers be-



The Pedigree

I-a. Birth 1815. Death at an age and cause unknown. Married twice. All offspring are believed to result from first mating.

II-a. Birth 1841. Death at 70. Liver disease?

II-b. Birth 1844. Death at 77, cause unknown. Wife died at age 75.

II-c. Birth 1847. Death at 50, cause unknown.

II-d. Birth 1850. Death at 60 with "locked bowels."

III-a. Birth 1880. Living. History does not suggest Marfan's Disease. Not available for examination.

III-b. Birth 1884. Death at 44 years of age with "dropsy." Tall and thin. Eye trouble all his life. Sired propositus. Wife born in 1893 and died of meningitis in 1927.

III-c. Birth 1887. Living. Eye trouble all his life. Short in stature. Probable form fruste?

III-d. Birth 1890. Death at 54 years of age with heart disease. Eye trouble all his life. Tall and thin. Sired three daughters one of whom died of heart disease at 32.

IV-a. Birth 1917. Death at 37 with dissecting aneurysm. Ectopia lentis,. six foot, four inches in height, severe pectus excavatum (case #2.). Sired three daughters one of whom is affected.

IV-b. Birth 1923. Death at 24 years from intractable heart failure. The propositus of this report. The image of his father according to surviving sister. Typical Marfan's Syndrome. Necropsied.

IV-c. Birth 1920. Living. Free of eye disease. Normal stature.

IV-d. Birth date unknown. Suffered with eye trouble all her life. Died at 32 years with heart disease.

IV-e. Birth date unknown. Believed to be normal.

IV-f. Birth date unknown. Believed to be normal.

V–a. Birth 1945. Living. Dislocated optic lens in one eye. Tall, thin, scoliosis of spine, dolichocephalic. Typical Marfan's Syndrome.

V–b. Birth 1947. Living. Examined. Not affected.

V–c. Birth 1948. Living. Examined. Not affected.

(The source of the pedigree was III-c, IV-a, IV-b, IV-c, the wife of IV-a, other relatives not shown in the pedigree, as well as the family Bible.)

low the costal margin on deep inspiration. Splenic enlargement by palpation was not found. All reflexes were hypoactive. Pitting edema of moderate degree was present in both ankles and up the leg as high as the knee. Moderate clubbing of all fingers and toes was seen. Rectal examination was negative. The genitalia were clinically normal.

Laboratory Studies: His hemoglobin measured 15 grams. The red blood count was 5.2 million per cu. mm. The white blood count totalled 6,700 with 67% polys., 31% lymphs., 2% eosin. A hematocrit of 52 was reported. The urine was amber, acid, sp. gr. 1.014, with a trace of albumen, one plus sugar and negative acetone. Microscopic examination of the urine revealed 1 to 2 casts per high power field and a rare rbc. The NPN measured 74 mg%. The plasma proteins totalled 5.81 grams. An icterus index of 16 was reported.

Course In The Hospital: With digitalis whole leaf in full doses, a low salt diet, bed rest and diuretics, a fair degree of compensation was obtained. Pulmonary congestion diminished. The heart rate slowed to 96 per minute. The NPN fell to 63.1 mg.%. An electrocardiogram was interpreted as showing a right ventricular strain pattern. A six foot chest film revealed a moderately enlarged heart, pulmonary congestion and elevation of the right hemidiaphragm. On 11-2-46 the patient was dismissed with instructions to continue the hospital regime at home. One week later he was readmitted as his condition at home had deteriorated. A gallop was now audible. Venous pressure measured 180 mm. of water. The blood pressure now measured 150/90. Four weeks later he was discharged without any visible improvement. On 2-24-47 he entered the hospital for the last time. Anasarca, orthopnea and abdominal distention were now severe. Opiates were required for relief. All measures were to no avail and the patient expired on 3-7-47.

Report of Necropsy

Necropsy was performed three hours after death. "The body is that of a young male with severe generalized edema. The right cornea is opaque. The conjunctivae are mildly icteric. The chest is symmetrical, with little subcutaneous fat. The abdomen is distended with fluid."

"Serous Cavities: Each pleural cavity contains about 400 c.c. of dark amber fluid. Adhesions at the apex were freed. The peritoneal cavity contained 2,000 c.c. of light amber fluid. The pericardial cavity is free of fluid or adhesions.

"Aorta and Vessels: The aorta follows a normal anatomical course but appears hypoplastic in the ascending portion of the arch, where it measures 6.2 cm. (normal 7.4 cm.). There is nothing else remarkable about the great vessels.

"Lungs: The right lung weighed 400 grams, the left 430 grams. Both lungs lie collapsed in the pleural cavity. The lower lobes appear purple and subcrepitant. Infarcts are seen in both lower lobes.

"Heart: The heart weighs 480 grams (normal 300 grams) and shows marked dilatation of both ventricles. The epicardium is devoid of fat. The subepicardial vessels are brown and soft. The left ventricle measures 13 mm. (8-10) in thickness and the right ventricle 5 mm. (2-3). The tricuspid valve measures 140 mm. (120) in circumference, the mitral 105 mm. (100), pulmonic 80 (85), aortic 80 (85). All valves except the mitral are membranous and competent. The superior half of the mitral valve is densely adherent to the endocardial surface of the left ventricle anteriorly and at the upper margin of the valve there are small bony spicules protruding into the ventricular cavity. The margin of the valve shows only moderate thickening. There is no thickening of the chordae tendinae. There are no gross mural thrombi. There is no evidence of congenital heart disease, other than the hypoplasia previously described.

"Liver: The liver weighed 1600 grams. It extends several fingers below the costal margin. The capsule is smooth. The liver surface is a deep purple. Cut surface is nutmeg in appearance. The gall bladder and biliary tree are free of calculi.

"Pancreas, Adrenal and Spleen: Not grossly abnormal.

"Gastro-Intestinal Tract: Stomach moderately dilated. Marked thickening and edema of

the wall of the small bowel. The appendix is grossly normal.

"*Genito-Urinary Tract:* The right kidney weighed 210 grams, the left 220 grams. Both kidneys are swollen but show normal relationship of cortex and medulla. The bladder contained 300 c.c. of urine. The prostate appeared normal.

Microscopic

"*Aorta:* Section through the ascending aorta shows moderate increase in thickness of the intima. Some deposition of lipoid within mononuclear phagocytes and some hyalinization is seen. There is no distinct elevation of the intima. Media and adventitia are not remarkable.

"*Heart:* The vessels in the epicardial fat including the smallest capillaries are markedly congested. One coronary vessel shows a dark blue staining material forming a wavy ring about the inner margin of the intima. This appears to be some peculiar change in the inner elastic membrane. The superficial myocardial cells are moderately swollen and degenerated, their nuclei stain poorly. The mitral valve is hyalinized and acellular. At its base there are scattered mononuclear macrophages and an occasional polymorphonuclear leucocyte which has escaped through a small congested capillary. There is no real inflammatory reaction. Nor are there any Aschoff bodies in the myocardium. The endocardium is edematous but intact. The muscle cells of the papillary muscle tend to fragment and occasionally there is a brown pigment about the nuclei of some of these cells. Some thickening of the septa is seen in focal areas. Small microscopic mural thrombi were seen in some of the sections. About the calcified area in the mitral valve there is marked hyaline fibrosis which extends to the free margin of the valve. There is no inflammatory reaction to indicate recent activity.

"*Spleen:* The Malpighian bodies are small and compressed. The central arterioles appear normal. All sinusoids are congested.

"*Liver:* The central half of each lobule consists of a mass of hemorrhage which has completely destroyed the liver. The sinusoids are dilated. The Kupfer cells are so hypertrophic that they compress the hepatic parenchymal cells.

"*Adrenals:* Congested.

"*Lungs:* Heart failure cells are seen. Areas of infarction in both lungs are present. The septa are thickened and edematous.

"*Kidney:* The glomeruli are congested. An occasional fibrinous deposit in the periglomerular space is seen. The tubules show cloudy swelling, with occasional free rbc's in the lumen. The renal artery at the hilus shows some areas of calcification in the media. One small branch of the renal artery shows the same blue wavy line at the junction of the intima and media as was described in a coronary artery.

"*Prostate and Seminal Vesicles:* An area of chronic inflammatory reaction is present in the prostate.

"*Stomach and Mesentery:* Congestion of the vessels.

"*Final Anatomic Diagnosis:* Marfan's Syndrome, chronic mitral endocarditis with calcification, hypoplasia of the ascending aorta, cardiac hypertrophy and dilatation, bilateral hydrothorax, ascites, chronic passive congestion of liver, pulmonary infarction, pleural adhesions, phthisis bulbi, right."

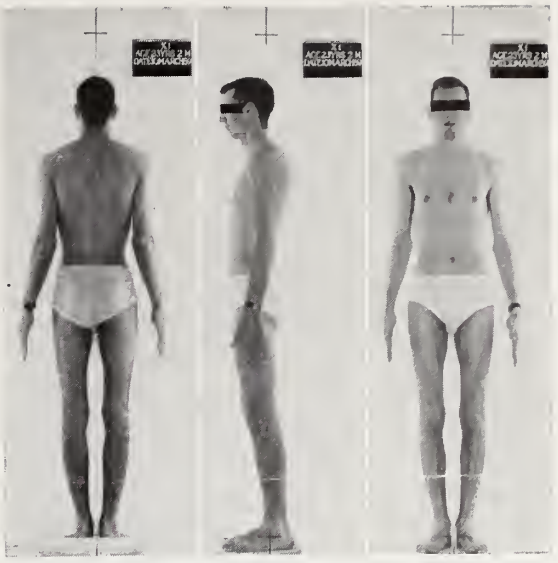
Case 2.

The patient, a 30-year-old married white male, brother to Case 1, was examined at my request in April, 1947. He had no complaints and failed to understand the reason for the interview. He measured 6 ft. 4 in. and weighed 144 lbs. The head was dolichocephalic; the palate highly arched. A prosthesis was worn in place of the right eye. He related that following eye surgery for subluxation of the optic lens in 1930 he developed glaucoma. This was treated until 1944 at which time enucleation of the eye became necessary. Ectopia lentis in the left eye with upward and inward displacement was readily seen with the ophthalmoscope. Correctible vision with glasses, however, was 20/30 in the left eye. A severe degree of pectus

excavatum was present. The lungs were clear. The heart was not enlarged to percussion. Rhythm was regular. An apical rate of 80 per minute and blood pressure of 120/78 was recorded. The PMI was not displaced. A loud systolic murmur was audible over the entire precordium. All joints were lax. A mild degree of kyphoscoliosis was present. No other findings of note were found.

This patient unfortunately failed to return. Inquiry years later revealed that he had died suddenly. His physician gave the following account of his last illness. On April 4, 1954, he sought medical consultation complaining of mild chest pain, aggravated by swallowing. On April 9, 1954 he developed sudden severe chest pain and died moments later. At death, extravasated blood was seen in the subcutaneous tissue of the neck and face. Death was attributed to dissecting aneurysm with rupture. Autopsy was not permitted.

Examination of the three daughters sired by this patient revealed the Martan Syndrome in the eldest. The usual skeletal deformities were well defined and the presence of ectopia lentis in one eye was confirmed by an ophthalmologist. At 12 years of age she measured 5 ft. 6 in. and weighed 86 lbs. She was doing well in her school work and her activity was not restricted. Evidence of cardiovascular involvement could not be found at the time of examination.



This patient was not a member of the T family discussed in the text. He was free of ectopia lentis although slit lamp examination revealed abnormal looseness and branching within the zonular fibres. A sister measured 5 ft. 11 in. with typical skeletal findings. Examination of the father and the family history confirmed the diagnosis.

Discussion

This communication adds a new family pedigree of the Marfan Syndrome to those previously reported. Diagnosis in our cases was established by the presence of ectopia lentis, the obvious skeletal defects and their proven genetic origin. Among members of the T family death at an early age from cardiovascular involvement was frequently recorded. English in descent, the family have lived in northern Kentucky for over 100 years. This origin was considered noteworthy when a paper by Williams[14] published in the 1870's was read. Williams practiced in Cincinnati, Ohio, a city less than 50 miles from the T family homestead. In his report he describes 2 cases of ectopia lentis in patients undoubtedly suffering with the Marfan Syndrome. It is of interest that his report appeared some 20 years before Marfan's publication. My attempt, however, to link his 2 cases with the T family was considered unsuccessful as the available facts failed to support such an agreeable hypothesis.

The practical importance of the disorder has been enhanced by the rapid progress made in cardiac and cardiovascular surgery within the last decade. Poor results[16] have followed attempts to repair vascular lesions in Marfan's Syndrome by surgical means. Surgical correction of eye defects have yielded equally poor results.[12] In our two cases blindness resulted in the eye of one patient and glaucoma followed in our second patient which eventually required enucleation, both as a consequence of surgical intervention. It would appear therefore that all elective surgical procedures in this group of patients had best be avoided. One need only recall the basic connective tissue defect inherent in these cases to realize why poor results with surgery can be predicted.

Published reports of necropsied cases have been reviewed[6,7,10,11,16] by others. Of autopsied cases 60% suffer aneurysm of the aorta while 30% succumb to dissecting aneurysm. Gore[17] attributes dissecting aneurysm to changes within the elastic lamella of the aortic media. Medial cystic disease is believed to parallel these

changes. Deposits of metachromatic coagulum and disorganization of smooth muscle when added to the lamellar changes within the media produce a thickening but weakening of the arterial wall.[7] The spread and confluence of these microscopic lesions may lead to one or more of the following: aortic aneurysm, aortic insufficiency, aneurysm of the sinuses of Valsalva, elevation of the coronary ostia, dissecting aneurysm, coronary insufficiency and congestive heart failure. Similar pathological changes[4,7] have been described in the pulmonary artery.

Uyeyama[8] and his coworkers were the first to report hypoplasia of the ascending aorta in the Marfan Syndrome. Succumbing to dissecting aneurysm found in the distal thoracic aorta, the ascending portion in their case appeared to escape this serious complication. Whitfield, Arnott and Stafford[9] have described hypoplasia of the ascending aorta in a second case of the Marfan Syndrome. In their report they also describe focal connective tissue changes within the myocardium which they interpreted as a myocarditis. These two cases were the only ones found in the literature to associate aortic hypoplasia with the Marfan Syndrome.

Our case is the third reported case to exhibit at autopsy hypoplasia of the ascending aorta and the second to report focal connective tissue changes within the myocardium. Whitfield's[9] case, unlike our patient, did not suffer with mitral valve disease. It is of interest, however, that both patients followed an inexorable downhill course succumbing to congestive heart failure and obtaining no significant relief with digitalis and other measures usually so successfully employed.

The belief has been expressed that heart failure can occur as a consequence of narrowing or hypoplasia of the ascending aorta. Moss[18] states a reduction of 50% or more in the intraluminal diameter of the aorta is required to significantly alter circulatory dynamics. A 50% reduction in the surface area of the aortic valve has been postulated in aortic stenosis before perceptible changes in hemodynamics becomes apparent. The modest reduction in intraluminal diameter suffered by our patient did not, it is believed, affect or alter this patient's clinical course.

Heart failure, it would appear in these cases, is unexplained and of obscure origin. The role of connective tissue residing within the myocardium in producing heart failure is undefined. Yet in acute severe rheumatic fever a disease recognized by its inflammatory effect on connective tissue elements within the heart as well as elsewhere, congestive heart failure is often fatal, especially in the young suffering their first attack. Poor results with digitalis in controlling heart failure is usual in these cases. Histologically, the muscle cells within the myocardium exhibit few changes and fail to correlate with the severity of the clinical picture. Aschoff bodies are frequently prominent in these cases yet as with the Marfan Syndrome modern histological techniques[16] have not been able to demonstrate the nature of the connective tissue derangement.

It would seem that the effects of the Marfan Syndrome on somatic growth may also result in an aorta that is longer, narrower and more vulnerable than normal. The disease produces at a later time, as a result of abiotrophy, the anatomical changes usually described at necropsy. The ascending aorta is known to contain a high content of elastic tissue and for this reason a site of predilection for the T. pallida in syphilitic aortitis. As the Marfan Syndrome also affects that part of the aorta richest in elastic tissue, the ascending aorta becomes the choice site of this disorder. This theory however fails to account for the cases reported by Uyeyama,[8] Whitfield,[9] and our own case.

Recently a patient with the Marfan Syndrome seen on the wards of this hospital was sent to the Growth and Development Center, of the University of Louisville, School of Medicine, for anthropometric study. All measurements, including that of the skull, fell into the normal range. Additional studies of this kind would be of great interest as these patients represent a unique and distinctive group within the human family.

Summary

A pedigree of the Marfan Syndrome in a native Kentucky family has been described. Six of eighteen members were found affected. Three of the six cases were examined and the diagnosis confirmed. Necropsy in one case exhibited hypoplasia of the ascending aorta, focal

connective tissue changes within the myocardium and calcific mitral valve disease. The rarity of these findings is discussed. The genetic origin of this disease is emphasized.

Acknowledgements

I wish to acknowledge the following: Dr. Allen Sakler, Dr. A. E. Leggett, Sr., and Dr. A. E. Leggett, Jr., made available the eye findings from their office records. Dr. Edgar Morgan supplied me with the facts in the final illness of Case No. 2. Dr. H. Blumenthal, formerly pathologist at the Jewish Hospital in Louisville, performed the autopsy and interpreted the microscopic sections. Dr. Frank Faulkner of the Growth and Development Center at the University of Louisville, School of Medicine, did the anthropometric study referred to in this report.

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DR. ABRAHAM M. GORDON
425-426 FINCASTLE BUILDING
305 WEST BROADWAY
LOUISVILLE 2, KY.

April 23,1960

Gerald McMutry
c/o Lincoln National Foundation
Fort Whayne,Indiana.

Dear Sir:

I was refered to you by the curator of the Filson Club here in Louisville when she was unable to give me the information I wanted about Abraham Lincoln.I'm presently engaged in a project in which I'm attempting to show that Lincoln suffered with a rare medical disorder known as the Marfan Syndrome.Any information you may have of medical or genetic interest would be of interset to me.I'm enclosing a reprint of an article on the subject of Marfan's Syndrome which appeared in the December '59 issue of the Kentucky State Medical Journal which might give you a better idea of what the disease is like in its clinical manifestations.

Do you have a picture of Lincolns hands made in Chicago about 1859 by Leonard Volk? At this same time a cast of Lincoln's face was also made. Do you know his foot size?His large hands and feet fit into the Marfan Syndrome as does his kyphoscoliosis and dolichocephalic head.I know he wore glasses?Do you perchance have his prescription? In my reading thus far I have not found the answer to any of these questions.Any assistance from your foundation will be given proper credit in any publication.

Very truly yours,

Abraham M.Gordon M.D.

rr.

April 27, 1960

Dr. Abraham M. Gordon
425-426 Fincastle Bldg.
305 West Broadway
Louisville 2, Kentucky

Dear Dr. Gordon:

I have your letter of April 23rd. I note that you are attempting to show that Lincoln suffered with a rare medical disorder known as Marfan Syndrome. I read your paper, reprinted from the Journal of the Kentucky State Medical Association, December, 1959, with a great deal of interest.

Have you read the book "Lincoln and the Doctors" by Dr. Milton H. Shutes, published in 1933? The index does not mention Marfan Syndrome. We do not have a photograph of Lincoln's hands. We do, however, have casts of his right and left hand made by Leonard W. Volk in 1860.

The size of Lincoln's feet are a matter of record. I am sending you a photostatic copy of two unidentified newspaper clippings published in 1892 and 1893 entitled "A Great Man's Feet" and "Abraham Lincoln's Large Feet". We have quite a number of references to Lincoln's feet in our files.

I am also sending you some Lincoln Lore bulletins which you may find of interest.

There is no information regarding any prescription for Lincoln's glasses. The enclosed clipping states that Lincoln paid 37½ cents for his first pair of glasses. There is a bit of information available that he did not purchase glasses until he was forty-seven years old.

The Chicago Historical Society own a pair of Lincoln's glasses which they have on exhibit. I enclose a photostat of the glasses.

If I can be of any further help, please let me know.

Yours sincerely,

R. Gerald McMurtry

RGM:hw
enclosures

DR. ABRAHAM M. GORDON
425-426 FINCASTLE BUILDING
305 WEST BROADWAY
LOUISVILLE 2, KY.

June 27,1961

R.Gerald McMurtry,
Director
The Lincoln National Life Foundation
Fort Wayne,Indiana.

DearSir:

About one year ago I wrote you requesting information about Abraham Lincoln and as I indicated in my letter than I believed Lincoln suffered with the Marfan Syndrome.Since that time I have done some additional research and I'm more convinced than ever.So much so that at the next meeting of the Kentucky State Medical Association in Louisville I'm going to present a paper titled, "Abraham Lincoln - A Medical Appraisal". My paper will be presented on September 21,1961 at 10:00 a.m.(Thursday).

I would be honored by your presence and would hope that you might make a few comments on this subject.

Hoping you can honor me with your presence,

Sincerely yours,

Abraham M.Gordon M.D.

Its name indicates its character

The Lincoln National Life Foundation

Fort Wayne, Indiana

R. Gerald McMurtry
Director

August 11, 1961

Dr. W. H. Scoins
Medical Department

Dear Dr. Scoins:

From the enclosed letter you will note that Dr. Abraham M. Gordon believes that Lincoln suffered with the Marfan Syndrome.

What is the Marfan Syndrome?

Sincerely yours,

Gerald

RGM:mm

R. Gerald McMurtry

MARFAN'S SYNDROME
(Arachnodactyly)

A Textbook of Medicine. Edited by Russell L. Cecil, M.D., Sc.D., & Robert F. Loeb, M.D. ... Tenth Edition. W. B. Saunders Company, Philadelphia & London. 1405-1406 pp.

Marfan's syndrome is a generalized hereditary disorder of connective tissue affecting especially the bones, the eyes, the ligaments and tendons and the cardiovascular system. The cause is clearly a mutation, appearing de novo or inherited as a mendelian dominant. Many manifestations, not first evident, appear as the defective connective tissue, especially elastic tissue, succumbs to normal stresses. The disorder shows np preference for race or sex, and the incidence is unknown; cases are discovered in increasing numbers as families are scrutinized and "incomplete" cases included.

A typical case shows dolichocephaly, with high, arched palate, long bones abnormally thin and long (dolichostenomelia, arachnodactyly) and span greater than height. Pectus excavatus (funnel breast) or carinatum (pigeon breast) may result from overgrowth of the ribs, and "spurs" from overgrowth of the os calcis. The eyes characteristically show ectopia lentis, and suspensory ligaments showing redundancy and rupture on slit-lamp examination. Abnormally long eyeballs with myopia and separation of the retina are common; deformities of lens and corneae and blue sclerae occur occasionally. The weakness of ligaments leads to "double-jointedness," dislocations, kyphoscoliosis, pes planus or cavus, genu recurvatum and herniae. Hypotonic muscles are common, probably resulting from redundancy of tendons.

Changes in the aorta, the most serious manifestation of the disease and one which may be present without the full-blown picture of arachnodactyly, result from degeneration of elastic fibers in the media (cystic medionecrosis). Microscopic examination discloses redundancy of smooth muscle masses, "cysts" and dilation of vasa vasorum. Stretching of the aortic ring results in valvular separation and insufficiency, with resultant angina and cardiac failure. Aneurysmal dilation of the ascending aorta is common, and dissecting aneurysm or fupture is a frequent cause of death. The pulmonary artery, frequently prominent on x-ray, may be similarly affected or only displaced laterally by the dilated aortic ring. The valve cusps themselves may be involved and bacterial endocarditis superimposed. Associated abnormalities such as coarctation of the aorta, patent foramen ovale and varicosites of the veins may be present.

The pathological physiology is not known; lathyrism in animals produces strikingly similar changes. Treatment, aside from avoidance of physical exertion, is symptomatic.

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The Louisville Times
Louisville, Kentucky
September 21, 1961

AILING ABE? . . .

Louisville doctor diagnoses a rare malady in Lincoln

Big ears, sloping shoulders were symptoms?

By CONNIE COURTEAU

Abraham Lincoln, that mournful, gangling giant among men, was afflicted with a rare disease which accounted for his stature and even his facial expression, a Louisville doctor believes.

Lincoln towered 6 feet 4, on long, spidery legs and huge feet; was stoop-shouldered, slow of movement, high of voice and woebegone in expression.

His appearance caused those who described him to occasionally call his physical make-up uncouth, despite his "unique traits of mind and spirit," says Dr. Abraham Gordon.

Dr. Gordon, who has done research on Lincoln's physical make-up for several years, presented a paper on the subject today to the Kentucky State Medical Association at Columbia Auditorium.

Believes Sons Had It, Too

The Civil War President, he said, was afflicted by a rare disease of the body's connective tissue called Marfan Syndrome, a disease which can affect the bone structure, the heart and blood vessels, or the eyes, any or all.

Lincoln inherited it from his mother, Dr. Gordon believes, and passed it on to three of his sons. Edward, William and Thomas, all of whom died before reaching adulthood.

Dr. Gordon bases his thetory on descriptions of Lincoln.

As stated by Lincoln's long-time law partner, friend and biographer, William Herndon, it was like this:

"Mr. Lincoln was 6 feet 4 inches high, and when he left the city of his home for Washington 51 years old, having good health and no gray hairs . . .

"He was thin, sinewy, rawboned, thin through the breast to the back, and narrow across the shoulders; standing, he leaned forward—was what may be called stoop-shouldered, inclining to the consumptive build.

"His usual weight was 180 pounds. His organization, rather his structure and function worked slowly. His structure was loose and leathery, his body shrunk and shriveled; he had dank skin, dark hair and he looked woe-struck.

"The whole man, body and mind, worked slowly, as if it needed oiling . . . When he walked he moved cautiously but firmly. His long arms and giant hands swung down. . . . His arms and legs were abnormally, unnaturally long. . . . It was only when he stood up that he loomed over other men. . . .

". . . His hair dark and almost black lay floating where his fingers or the wind left it. . . . His cheek bones were high, sharp and prominent; his jaws were long and curved upward; his nose large, long, blunt and a little awry toward the right eye. His chin was sharp and upcurved, his eyebrows cropped out like a huge rock on the brow of a hill. . . . His ears large, and ran down almost at right angles from his head." (Another writer said when Lincoln smiled she thought his ears would flap like a good natured elephant's.)

To Dr. Gordon, this meant that Lincoln had curvature of the spine and a slight back hump, flat feet, long stringy muscles and

Col. 2, back page, this section

Louisville Doctor Says Lincoln Had Rare Illness

CONTINUED FROM PAGE 1

spidery legs which are symptomatic of Marfan Syndrome. So is the eye trouble which afflicted Lincoln and his high-pitched almost feminine voice. He apparently did escape any heart trouble.

Dr. Gordon believes Lincoln inherited the disease from his mother, who was tall and thin, and "carried a sad, melancholy facial expression." He thinks it more likely that Lincoln's characteristics were the result of his mother's having the disease than an ordinary inheritance of build from the Lincoln side. His father, Thomas, was stocky and jovial, nothing like his son, but some other members of the Lincoln family were tall.

Dr. Gordon agrees with Lincoln scholars who believe Lincoln's mother was an illegitimate child. And he thinks some of her family may still be located in Virginia.

"I have seen a photograph taken about 10 years ago of a man who entered the Medical College of Virginia who was extremely Lincolnesque . . . had a sad expression, big ears," he said. He believes that most of the family probably carries the "stigma of the disease" and could be found if he had time to do some more research.

He is described as having "a shambling, loose, irregular, almost unsteady gait, a tall, lank, lean man, considerably over six feet in height, with stooping shoulders, long pendulous arms, terminating in hands of extraordinary dimensions, which, however, were far exceeded in proportion by his feet."[1]

Indeed, he was pictured by his partner and longtime friend as being "thin, sinewy, rawboned, thin through the breast to the back, and narrow across the shoulders. . . . When he walked he moved cautiously but firmly; his long arms and giant hands swung down by his side. . . . He put the whole foot flat down on the ground at once, not landing on the heel. . . . His legs and arms were abnormally, unnaturally long and in undue proportion to the remainder of his body; . . . his ears large, and ran down almost at right angles from his head."[1]

Cartoonists of the day always portrayed him as an apelike creature with unnaturally long and loose limbs and as being rather dolichocephalic, flat-footed, kyphotic, narrow-chested, and prognathic. Was this misshapen man suffering from a disease? He may well have been a case of the Marfan syndrome, wrote Gordon in a recent article.[1]

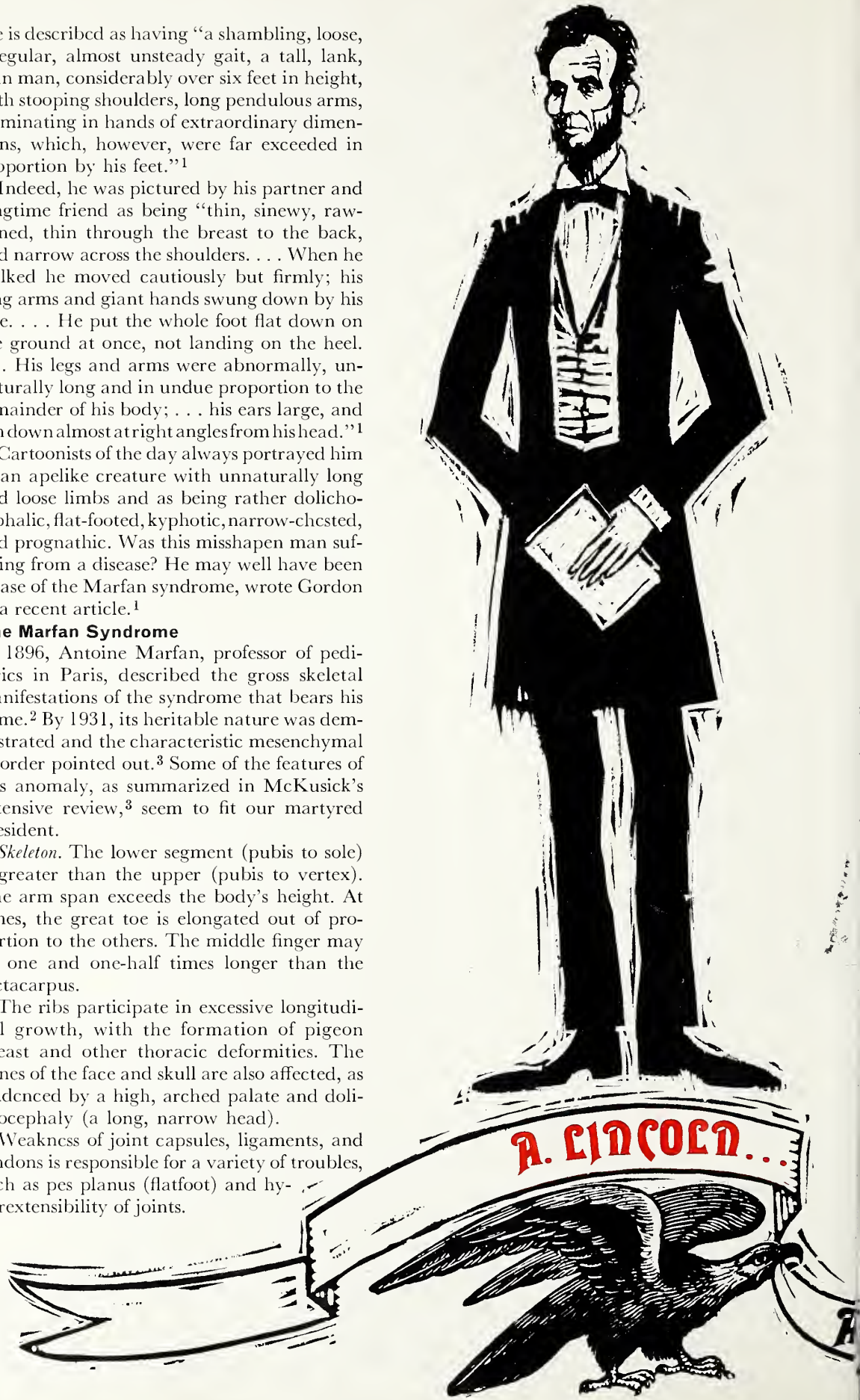
The Marfan Syndrome

In 1896, Antoine Marfan, professor of pediatrics in Paris, described the gross skeletal manifestations of the syndrome that bears his name.[2] By 1931, its heritable nature was demonstrated and the characteristic mesenchymal disorder pointed out.[3] Some of the features of this anomaly, as summarized in McKusick's extensive review,[3] seem to fit our martyred president.

Skeleton. The lower segment (pubis to sole) is greater than the upper (pubis to vertex). The arm span exceeds the body's height. At times, the great toe is elongated out of proportion to the others. The middle finger may be one and one-half times longer than the metacarpus.

The ribs participate in excessive longitudinal growth, with the formation of pigeon breast and other thoracic deformities. The bones of the face and skull are also affected, as evidenced by a high, arched palate and dolichocephaly (a long, narrow head).

Weakness of joint capsules, ligaments, and tendons is responsible for a variety of troubles, such as pes planus (flatfoot) and hyperextensibility of joints.



Muscular underdevelopment and hypotonia are frequent but by no means invariable. Little subcutaneous fat is found.

Eyes. Bilateral dislocation of the lens is a common type of ocular involvement. A defective suspensory ligament is the probable cause. Myopia, which is usually present, appears to indicate involvement of the sclera (fundamentally a ligamentous structure). A shallow anterior chamber may also be found.

Cardiovascular system. There is an inborn weakness (an abiotrophy) of the media of the aorta and main pulmonary artery. This results in diffuse dilatation of the ascending aorta or pulmonary artery or in dissecting aneurysm. Septal or valvular defects have also been described.[4]

Other manifestations. The voice sometimes is rather high pitched and may have a characteristic timbre.[3] Congenital cystic disease of the lung has occasionally been reported.

Inheritance

The disease, uncommon but not rare, is said to have an incidence of 1.5 per 100,000 population. All races and both sexes are equally affected. Inheritance is that of a simple mendelian autosomal dominant.

Descriptions of Lincoln

What is the evidence that Lincoln had the Marfan syndrome? At best, one must use conjecture based on photographs and on information supplied by those who knew him.

Before he was seventeen years old, Lincoln was six feet two inches tall and weighed about 160 pounds. "His body was slim but wiry, his skin shriveled and yellow, his arms were large and muscular, his legs long."[5]

Carl Schurz gave this description of him. "His lank ungainly body was clad in a rusty black dress coat with sleeves that should have been longer, but his arms appeared so long that the sleeves of a store coat could hardly be expected to cover them all the way down to the wrists. His black trousers, too, permitted a very full view of his large feet."[5]

Lincoln suffered from strabismus and at fifty-one years of age had several attacks of double vision.[5] Was this ectopia lentis which is often found in Marfan's syndrome? He also had a high-pitched voice, another characteristic of this anomaly. His ears were large and malformed. Indeed, an artist who painted his portrait complained of Lincoln's asymmetrical features.[1]

Even an anti-Lincoln pamphlet of 1864 described his personal appearance.[5] "His anatomy is composed mostly of bones, and, when walking, he resembles the offspring of a happy marriage between a derrick and a windmill. When speaking, he reminds one of the old signal telegraph that used to stand on Staten Island. His head is shaped something like a ruta-bago [sic] and his complexion is that of a Saratoga trunk. His hands and feet are plenty large enough, and in society he has the air of having too many of them."

The Question of Inheritance

Tad (Thomas) Lincoln, one of Abe's sons, had a cleft palate (also associated with Marfan's syndrome) and died at the age of eighteen of "dropsy of the chest"; perhaps this was a form of congenital heart disease associated with the syndrome. We will never know.

What about Lincoln's parents? Thomas, the father, was only about five feet ten inches tall and was rather stocky. His mother, on the other hand, was tall (five feet eight inches) and thin (130 pounds) and was said to have had a sad facial expression. Many questions remain unanswered about her appearance, yet Lincoln himself said he inherited his mother's qualities.[1] Since her own lineage is somewhat in doubt, there is only circumstantial evidence pointing to Nancy Hanks as the source of his disorder.

Although Lincoln was free of cardiovascular disease, as far as is known, it is true that any one of the three major components (skeletal, ocular, or cardiovascular) may be present with little or no involvement of the other two areas.[3]

Marfan's publication appeared thirty-one years after Lincoln was shot. Perhaps, in a sense, the term "Lincoln's syndrome" is appropriate. At any rate, the image of the Great Emancipator remains as enigmatic as ever.

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Case of the Marfan Syndrome?

The Courier-Journal
Louisville, Kentucky
June 17, 1962

Marfan's Syndrome Studied

Did Rare Disease Affect Abe's Mind?

By TOM REYNOLDS

A rare disease called Marfan's Syndrome may eventually help cut through some of the mystery surrounding Abraham Lincoln.

The Civil War occupant of the White House pretty definitely had the inherited disorder, Dr. Abraham M. Gordon, 85 Wampum Road, reported last fall in a medical paper.

Now Gordon plans a study of the psychological aspects of the disease with psychiatrist Dr. Bernard Hall of Topeka's famed Menninger Clinic.

Colleagues have credited Dr. Gordon with supplying the key to Lincoln's rangy physical makeup. In the new search, the two doctors intend to make a psychological study of people with this disease, try to learn its effects on their minds, and possibly account in part for the immortal President's psychic makeup.

Symptoms Listed

Lincoln typified Marfan's Syndrome, Dr. Gordon says. This collection of symptoms includes long limbs, height, large hands, deep-set eyes, hollow chest, a high voice, stoop shoulders, and a melancholy expression.

"When you hear 'Marfan's Syndrome,' you think of someone Lincolnesque in appearance," he explained. It is an inherited disorder of the connective tissue showing up in the bone structure, the eyes, and at times the heart and blood vessels—any or all of these.

Had No Heart Disease

A Frenchman discovered it in 1896, and only about 500 cases have been noticed and reported so far.

Would Marfan's Syndrome have struck down Lincoln if John Wilkes Booth hadn't?

Lincoln lacked heart disease — another symptom — and if sufferers escape heart trouble, "they usually live a normal span of life."

Behind the sad, hollow eyes of many of these patients does there hide the mind of another Lincoln?

To this speculation, Dr. Gordon says, "A man has to have a rendezvous with history—be there at the right time—to become great, you know." Contributing are "the character of the man and his spirit."

Caused By Mutant

However, apparently related to the disease are intellect, outlook, and original approach in thinking. This has been found in others with Marfan's Syndrome.

"We hope to decide whether there is a common denominator psychologically, a thread woven through all these people, affecting their manner of thinking. I have the feeling it does affect it, but that isn't proof."

The disorder is known to be caused by a mutant, or abnormal, dominant gene (passed to most or all offspring), he said.

The planned study has drawn one interesting result. A Kentucky woman who said she is a distant Lincoln relative on the Hanks side wrote Dr. Gordon that Lincoln had some Indian blood.

It is a family legend, she said, that Lincoln's grandmother, Mrs. Abraham Hanks, was part Choctaw. She felt this would explain the President's dark skin and coarse black hair. Further, she thinks some of the historians wandered off the genealogical path, since there were eight "Nancy Hankses" born about the same time.

Sons Died Young

Dr. Gordon, who has had several patients with the disease, got interested in Lincoln after reading a description of his physical characteristics in a biography.

Lincoln apparently inherited the disease from his mother and passed it on to three of his children. His son, Willie, who resembled him most, died at 12 in the White House, presumably of pneumonia. "Peculiarities in the lungs make some of these individuals prone to pneumonia," Dr. Gordon noted.

Son Tad died at 18 of "dropsy of the chest," likely with heart failure. Early death from heart disease is a frequent visitor with Marfan's Syndrome.

Long, thin little Edward died at 3½ in Springfield of an obscure illness.

Robert Lincoln, relatively short (5 feet 9), showed no skeletal symptoms—and lived to 86. But his son, Abraham, II, said to have been the image of his grandfather, died about 17 with "fluid" in his chest, Dr. Gordon said.

DR. ABRAHAM M. GORDON
425-426 FINCASTLE BUILDING
305 WEST BROADWAY
LOUISVILLE 2, KY.

August 3, 1962

Mr. R. Gerald McMurtry, Director
THE LINCOLN NATIONAL LIFE FOUNDATION
Fort Wayne
Indiana

Dear Mr. McMurtry:

I thought it might be of some interest to you to know that I have recently seen a Nora Sparrow, a direct descendent of Lucy Hanks and Henry Sparrow five generations removed. Miss Sparrow is thin although not tall, she measures 5'3" in height and weights 99 pounds. Her arm span measurements were 65½". Her Upper Segment--Lower Segment ratio is within the Marfan Syndrome limits. She has flat feet with pronation and hammer toes. Loose jointedness was not marked although she is kyphotic. Her eyes were within normal limits. In my opinion, she is hightly suggestive from her morphologic appearance of the Marfan Syndrome.

Another Lincoln relative descended from the Hanks side has also contacted me. From her description of relatives they probably have the Marfan Syndrome.

Dr. Bernard Hall, of the Menninger Foundation in Topeka, Kansas and I are requesting a grant from the NATIONAL INSTITUTE OF HEALTH for psychological and psychiatric studies of these patients with the Marfan Syndrome. As a corollary to this study I am much interested in examining Lincoln's relatives for study. Could you help me in this matter? In addition, do you know of any funds that might be available in supporting this study among the descendents of the Lincoln or Hanks family?

Very truly yours,

A. M. Gordon, M. D.
F. A. C. P.

AMG/ncg

August 10, 1962

Dr. A. M. Gordon
425-426 Fincastle Building
305 West Broadway
Louisville 2, Kentucky

Dear Dr. Gordon:

I am glad to have your letter of August 3 and to learn that you are continuing your study of Abraham Lincoln in relation to the Marfan Syndrome.

I fear I can be of little help to you in the way of giving you leads to living descendants of the Lincoln and Hanks families. We have no such contacts here in northern Indiana.

Of course, I will be glad to help you in any way with regard to published material on the subject of Lincoln's health.

Our Foundation has never made a grant for such a study you propose. Our funds are used to increase our library facilities and as scholarships for undergraduate studies.

Wishing you success in your research venture, I remain

Yours sincerely,

R. Gerald McMurtry

RGM:md

THAT MARFAN'S SYNDROME "AFFLICTION" OF LINCOLN

"Did ILLNESS MAKE LINCOLN GREAT?" So reads a 1963 magazine headline, ironically appearing on February 12th, Lincoln's 154th birthday.

The magazine article is based upon the research of a Kentucky internist, who now, after 20 years study of Lincoln's physical and mental traits, attributes his greatness to an uncommon disease, known as Marfan's Syndrome.

In view of the following article about the PARADOX OF LINCOLN'S RISE and the exalting spiritual qualities of our 16th President that it substantiates, it is in my opinion, sheer vilification to attribute Lincoln's greatness to some frightful disease.

Technically, Marfan's syndrome is defined as a hereditary disease of the body's connective tissues, the bones, muscles and ligaments, the symptoms occurring together, effecting the eyes, the heart and the skeletal structure. As applied to Lincoln, it is said to have produced the sturdy rail-splitter's "long sad face, sunken chest, misshapen ears, loose, spidery arms, immense hands, gangly appearance, uncouth movements, high pitched, almost girlish voice." Some contradictions in Lincoln's personality, such as his periodic shyness and his inclination to tell stories and jokes, and shortly thereafter sink into a deep depression, seem to impress our medical detective, who asserts that these characteristics are due to the rare disorder, known as Marfan's Syndrome. (Marfan was a French pediatrician who identified the ailment in 1896, 31 years after Lincoln's death.)

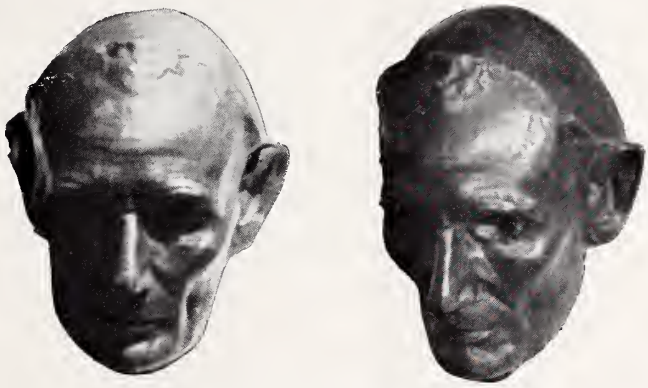
So now, 98 years after Lincoln's death, we are asked to accept a *medical diagnosis* that attributes Lincoln's *superb intelligence and leadership* to a loathsome inherited disease.

Such diagnosis, obviously, was never made. The disease was unknown in 1860, and the patient, though described by a contemporary writer as the "offspring of a happy marriage between a derrick and a windmill," did not submit to a physical check-up

and certainly needed no medical attention. For want of more scientific information, our diagnostician now says, he duly read the description recorded by the "tailors and bootmakers" who fitted Mr. Lincoln, and concluded that abnormalities were indicated.

And how can a dedicated Lincoln student, a layman, dispute a physician's verdict that the great emancipator's humanity, understanding and compassion were part of what Lincoln harvested from his ailment?

By the evidence, of course, evidence which happens to flatly contradict Lincoln's medical detractors.



TWO VIEWS OF THE LIFE MASK
OF ABRAHAM LINCOLN
MADE BY LEONARD W. VOLK IN 1860
Original bronze casting in Lincoln Memorial room of
Blumhaven Library and Gallery, Philadelphia.

In the Lincoln Memorial Gallery of the Blumhaven Library, there is an original bronze casting of Lincoln's face, made by the sculptor Leonard Volk. It is a beardless face, with no structural details concealed by the growth of hair. This impression of Lincoln's face was made in 1860 before he was nominated for president. It reveals a relatively small head for a man 6 ft. 4 in. tall,

and *there is no discernible deformity* or disease visible in that exact replica of Lincoln's face. The ears are not abnormal or misshaped. There is no deviation of the eyes. They are completely balanced and normal.

A top ranking Philadelphia physician examined that bronze mask recently and declared there is not the slightest deviation of the eyes or a skeletal disorder indicated. This doctor also declines to attribute the early death of Lincoln's children to the inherited illness. There is no evidence to support that speculative diagnosis, he declares.

In addition to the life mask, Blumhaven has a bronze casting of Lincoln's hands. The fist of the right hand is powerful, not spidery. The left hand, gripping a broom handle, is a striking reflection of the man's strength and powerful physicial condition. These replicas of Lincoln's face and hands tell us the truth about Lincoln's physical being.

The medical verdict of 1963 will not tarnish the heroic image of Abraham Lincoln, or rob him of the glory in history that is his.

To the medical profession, I respectfully submit Richard Watson Gilder's classic description of Lincoln's bronze life mask. It is worthy of their sober consideration and their contemplation of human fallibility. The Gilder poem;

"This bronze doth keep the very form and mold
Of our great martyr's face. Yes, this is he;
That brow all wisdom, all benignity;
That human, humorous mouth; those cheeks that hold
Like some harsh landscape all the summer's gold;
That spirit fit for sorrow, as the sea
For storms to beat on; the lone agony
Those silent, patient lips too well foretold.
Yes, this is he who ruled a world of men
As might some prophet of the elder day—
Brooding above the tempest and the fray
With deep-eyed thought and more than mortal ken.
A power was his beyond the touch of art
Or armed strength—his pure and mighty heart."

MILTON H. SHUTES, M.D.
ROUTE 2, BOX 78
CARMEL, CALIFORNIA

5-5-63

Dear Gerald McMurtry,

I have no regard for M.D.'s who jump to conclusions by reading a book or two about Lincoln – such as a Los Angeles doctor who in his book has Lincoln color blind. Then there was Edward J. Kempf, M.D., who spread his "knowledge" of Lincoln in 1952 at the American Medical association. A copy of his brochure is full of misinterpretations. Kempf was nicely spanked by T. D. Stewart, Curator of Physical Anthropology at the Smithsonian Institution and now, of course, Dr. Gordon, who, as you know, is full of exaggerations. Perhaps you know that Wayne Temple & his Board members have refused to use Dr. Gordon's staff.

There is Harald Schwartz, M.D., of Huntington Park, Calif. who came to visit me. He has an interesting story of a 12 year old boy who not only is a subject of a Marfan syndrome but also a grandfather who claimed before his death, to be a distant relation of Abraham Lincoln. Dr. Schwartz is intensely interested in both & also cautious. He now is a subscribes to the Lincoln Herald

Of course there is this about Lincoln which you might glance over in the Supplement of any Lincoln's Emotional Life It about covers the physical-temperamental Lincoln.

There is this too, about Lincoln – Tad's lisp & not as keene as he should have been. Robert's early teen-age period was noticably cross-eyed (left) for a while. Nicolay said that Robert Lincoln at 51 years was having trouble with his eyes – to which Robert replied, "I have been pulling along with one eye for many years in a lopsided way..." This gives me thought but I do not take it seriously – by no means.

The skeletal defects are primarily in the legs – abnormally long & slender but also are disproportionately long in relation to the patients own bodies – not just long in the legs.

The muscular + subcutaneous tissue is poorly developed, which gives an ~~emaciated~~ appearance. There are serious cardiovascular defects + also ocular defects. One read that from 60 to 70% of reported cases are myopic. Lincoln's eyes are the opposite – hyperopic.

Finally, what kills any signs of Marfanism in Lincoln is that the Marfan syndrome disease is always progressive, that "Treatment, aside from avoidance of physical exertion, is symptomatic". Lincoln had plenty of powerful exertions in his life-time!

What annoys me with Dr. Abraham is the frequent use of the words "maybe" possibly" Etc.

No, I have not planned to answer Dr. Gordon's this + that – but it is a thought even if I'd be 80 in august.

Incidentally, I have found another Lincoln male-larger than the "famous" one. You can find it easily on the right ear. I have found it in about 250 more photographs – but the best photo is the 11½ x 9 inches by Rand McNally & Co – with these words by Paul Angle" The original photograph was made by Alexander Hesler of Chicago in 1860. It is reproduced here by courtesy of the Chicago Historical Society, which owns the duplicate negative."

anyway, there is no marfan issue in Lincoln's right ear-lobe

Sincerely

Mary Ellis

May 10, 1963

Dr. Milton H.Shutes
Route 2, Box 78
Carmel, California

Dear Dr. Shutes:

Many thanks for your letter dated May 5th. I was glad to get your reaction to the Marfan Syndrome theary advanced by Dr. Abraham Gordon.

I do hope you will reply to this so-called claim that Lincoln suffered from this malady.

Yours sincerely,

R. Gerald McMurtry

RGM:hb

Nate Hanks - Lincoln references.

Marfan's Syndrome

A. B. LOVEMAN, M.D.; A. M. GORDON, M.D.
AND
M. T. FLIEGELMAN, M.D.
LOUISVILLE

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Marfan's Syndrome

Some Cutaneous Aspects

A. B. LOVEMAN, M.D.
A. M. GORDON, M.D.
AND
M. T. FLIEGELMAN, M.D.
LOUISVILLE

Marfan's syndrome and how it affects the skeletal, cardiovascular, and ocular systems is discussed very briefly. A review of the previously reported cutaneous accompaniments of this syndrome is presented, and four cases with striae distensae are reported, along with detailed histologic studies. It is felt that the finding of striae in such cases affords further evidence that the pathogenesis of this syndrome is some abnormality of the elastic tissue.

Marfan's syndrome is a heritable disorder of connective tissue, affecting for the most part three major systems of the body: the skeletal, the cardiovascular, and the ocular. The first clear description of this syndrome was reported by the French pediatrician, Marfan,[1] in 1896. Although the skeletal abnormalities were described by him, it was Achard[2] who suggested the name, arachnodactyly, for the spider-like fingers and toes associated with this disorder. Among the important skeletal abnormalities are tallness, loose jointedness, a dolichocephalic skull, a high arched palate, arachnodactyly, pigeon breast or pectus excavatum, kyphosis, poor muscle tone, scant subcutaneous fat, pes planus, and large deformed ears. Recently, one of us[3] reported that Abraham Lincoln may have had this syndrome which he inherited from his mother. Since then, we have been privileged to study a few of his living relatives who have the Marfan disease, or a forme fruste of the disorder. These patients afford further support of the theory that Lincoln was a Marfan syndrome sufferer.

Boerger[4] was the first to describe ectopia lentis as part of this syndrome, and since

Sections of Dermatology and Syphilology and Department of Medicine, University of Louisville School of Medicine.

then, at least 50% of these cases have manifested ocular involvement.

Cardiovascular disorders have been described [5] and include aortic aneurysm, dissecting aneurysm, valvular heart disease, medial cystic disease of the aorta, and congestive heart failure.

Report of Cases

The following cases are reported because of their interesting dermatological aspects,

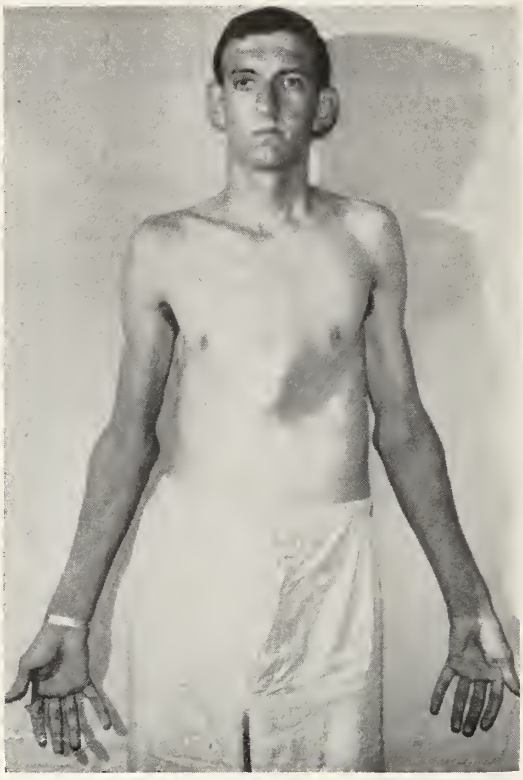

Fig. 1 (Case 1).—Note large hands, pigeon breast, large deformed ears, and dolichocephalic skull.

which, in our opinion, may help to explain the pathogenesis of this disorder.

CASE 1.—A male, age 15, was first seen on July 2, 1962, because of severe pain in the right chest. He was examined by one of us (A. M. G.), who diagnosed a spontaneous pneumothorax and Marfan's syndrome.

Family History.—The father, with classical findings of the Marfan syndrome died at age 30 of a dissecting aneurysm, rupture of the pulmonary artery, and aortic insufficiency (autopsy report). The paternal grandmother died of "heart trouble" at age 29. A sister (Case 2) is included in this series of cases. One brother has a pigeon breast,

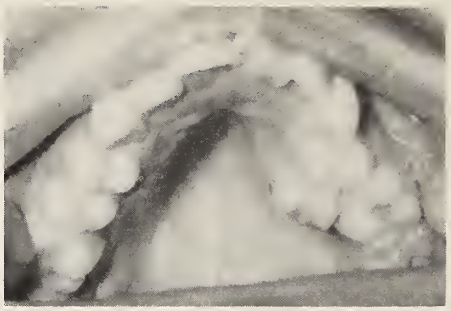

Fig. 2 (Case 1).—Note narrow high arched palate and linguoversion to the arch of the second left maxillary bicuspid.

large deformed ears, a high arched palate, arachnodactyly, and has been operated on for ectopia lentis and bilateral cataracts.

Personal History.—The patient is very bright and has an I.Q. of 147. He too has had his eyes operated on for ectopia lentis and secondary cataracts. He was aware of striae over his body, but could not tell us exactly how long these had been present. His guess was about four or five years.

Examination.—The patient has a dolichocephalic skull with large, slightly deformed ears. The facial expression is sad and owlish (Fig. 1). The eyes revealed bilateral iridectomies, and bilateral aphakia was present. An extremely narrow and high arched palate was noted. De. Harold E. Boyer of the University of Louisville School of Dentistry examined this patient and reported a Class 4 malocclu-

Fig. 3 (Case 1).—Note extensive atrophic striae.

sion and complete linguoversion to the arch of the second left maxillary bicuspid (Fig. 2). There is a moderate kyphosis and mild scoliosis with pigeon breasting. The lungs reveal a right pneumothorax. There was a loud systolic murmur over the precordium, but this disappeared when the patient recovered from his pneumothorax. There was no evidence of cardiovascular disease at this time. The extremities revealed typical findings of the Marfan syndrome which included loose jointedness, genu recurvatum, hammer toes, pigeon breast (pectus carinatum), and large hands and feet with long tapered digits (arachnodactyly). The arms and legs are unduly long. His weight is 147 pounds, height 71¼ inches, and arm span measurements are 76 inches. The skin presents extensive atrophic striae, symmetrically located over the upper thorax, deltoid areas, upper back, lower abdomen, and buttocks (Fig. 3). The striae run in diverse directions and vary in color from ivory white to slate gray with a faint bluish tinge.

Laboratory data were essentially normal except a chest x-ray showed a 50% collapse of the right lung. ECG revealed a normal tracing with sinus arrhythmia.

Histologic Examination.—We are indebted to Dr. Herman Pinkus for his invaluable assistance in the study of the striae from several cases. He prepared the sections, furnished the photomicrographs, and practically all the histologic report is taken verbatim from his letters. "The sections of the glabrous skin show a protrusion which may be partly due to the curling of the specimen in the fixative. However, it corresponds exactly to the area of involvement. In this area, the epidermis has average thickness but does not exhibit any rete ridges and the corresponding cutaneous papillae also are absent. H and E (hematoxylin and eosin) sections show a slight disarray of collagen bundles, but the essential changes are seen only in Orcein and Giemsa sections. The regular brush-like elastic fibers of the papillary layer are thinned out or

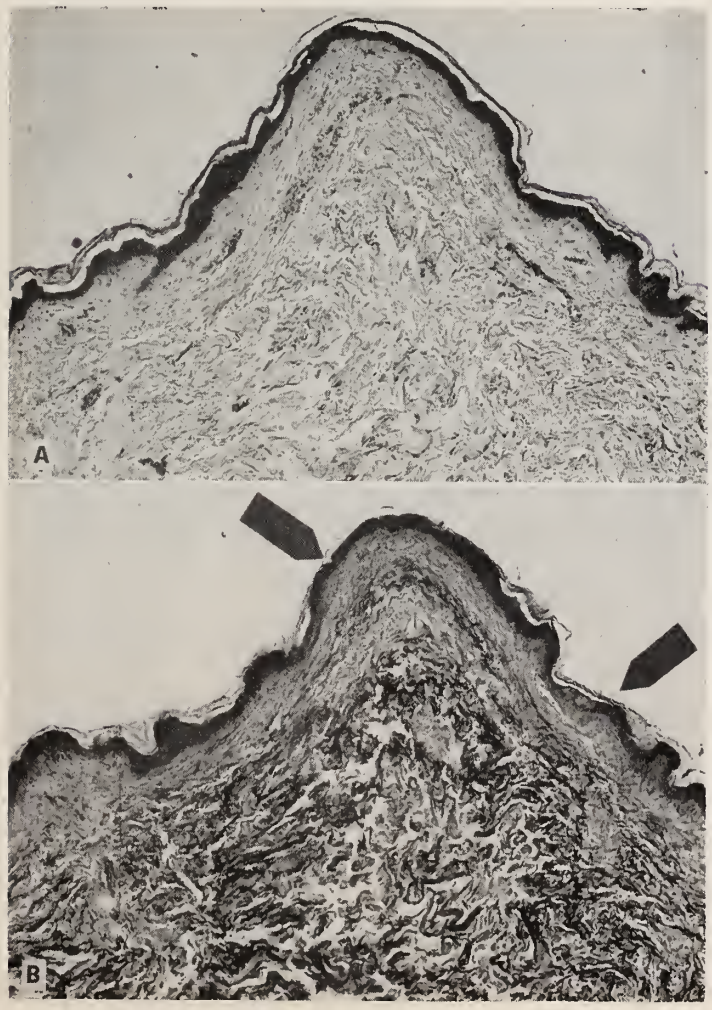

Fig. 4 (Case 1).—Stria in Marfan's syndrome. ×45. *A,* section stained with hematoxylin and eosin shows atrophy of epidermal ridges, some disturbance of arrangement of collagen bundles, and increased vascularity. *B,* section stained with acid, orcein, and Giemsa solution shows disturbed arrangement of elastic fibers.

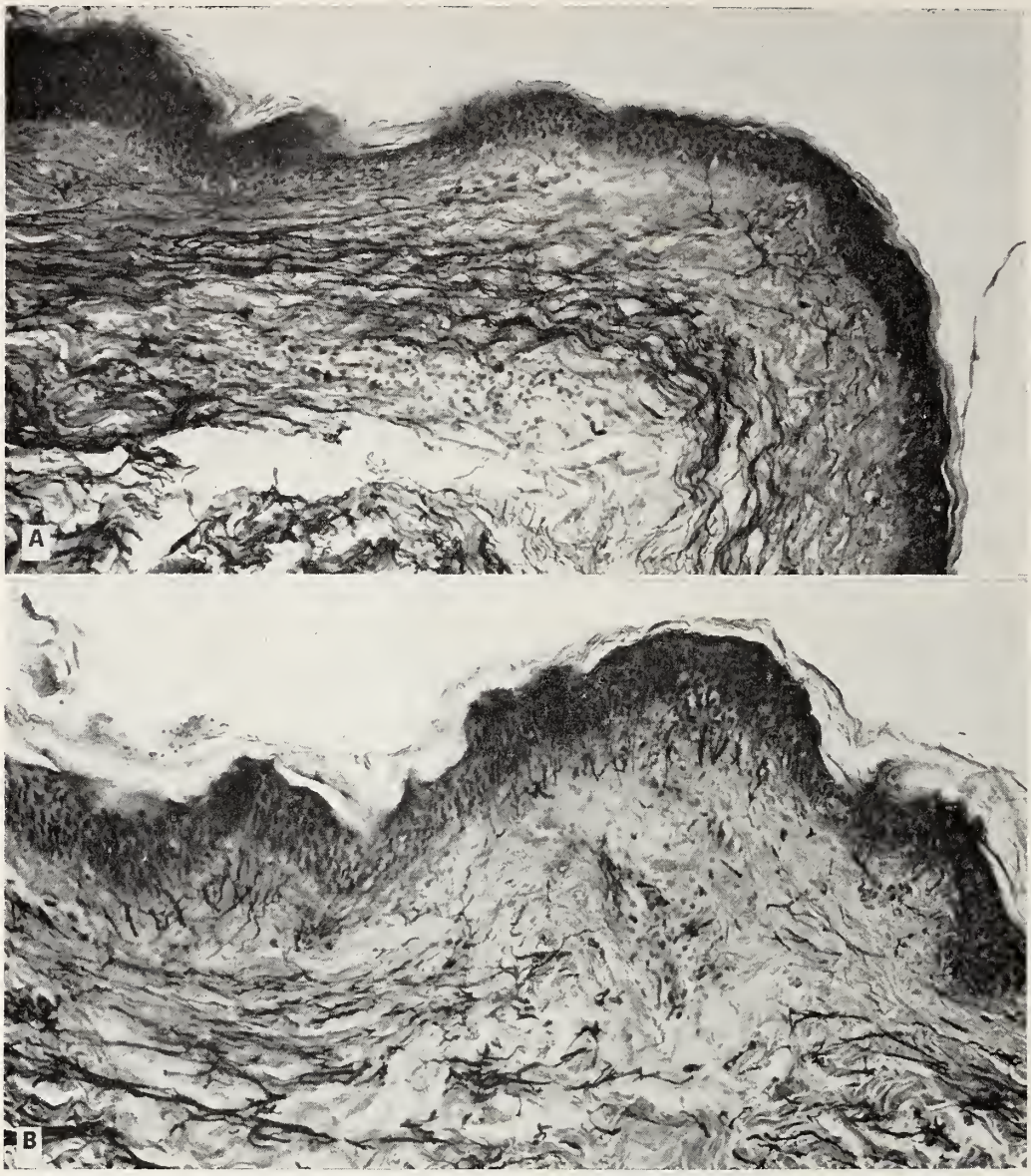

Fig. 5.—Enlargement of two areas corresponding to fields indicated by arrows in Figure 4. Acid, orecein, and Giemsa; × 185. *A,* loss of normal elastic fibers in pars papillaris and pars reticularis of the corium. The latter are replaced by numerous thin elastic fibers arranged parallel to the skin surface. *B,* almost normal amount and arrangement of elastic fibers in skin lateral to the stria.

completely absent. The thick elastic fibers of the pars reticularis of the corium come to an abrupt end at the border of the involved zone at each side of the lesion. The involved area contains fairly numerous fine elastic fibers which run parallel to the skin surface, although they are somewhat wavy and are aggregated in some places more than in others. There are evidently newly formed elastic fibers similar to those seen in old scars, especially if the injury was sustained fairly early in life. In addition, there is mild paravascular oedema and lymphocytic infiltrate in the border of the involved area."

Conclusions.—"All these findings are quite typical of stria distensa in a relatively late stage" (Figs. 4 and 5).

Case 2.—A sister of the boy in Case 1, this 13-year-old female measured 5 feet 8 inches. She is thin and loose jointed. As the result of severe ectopia lentis and cataractous changes, both lenses were surgically removed many years ago, and the patient wears thick corrective glasses. At the time

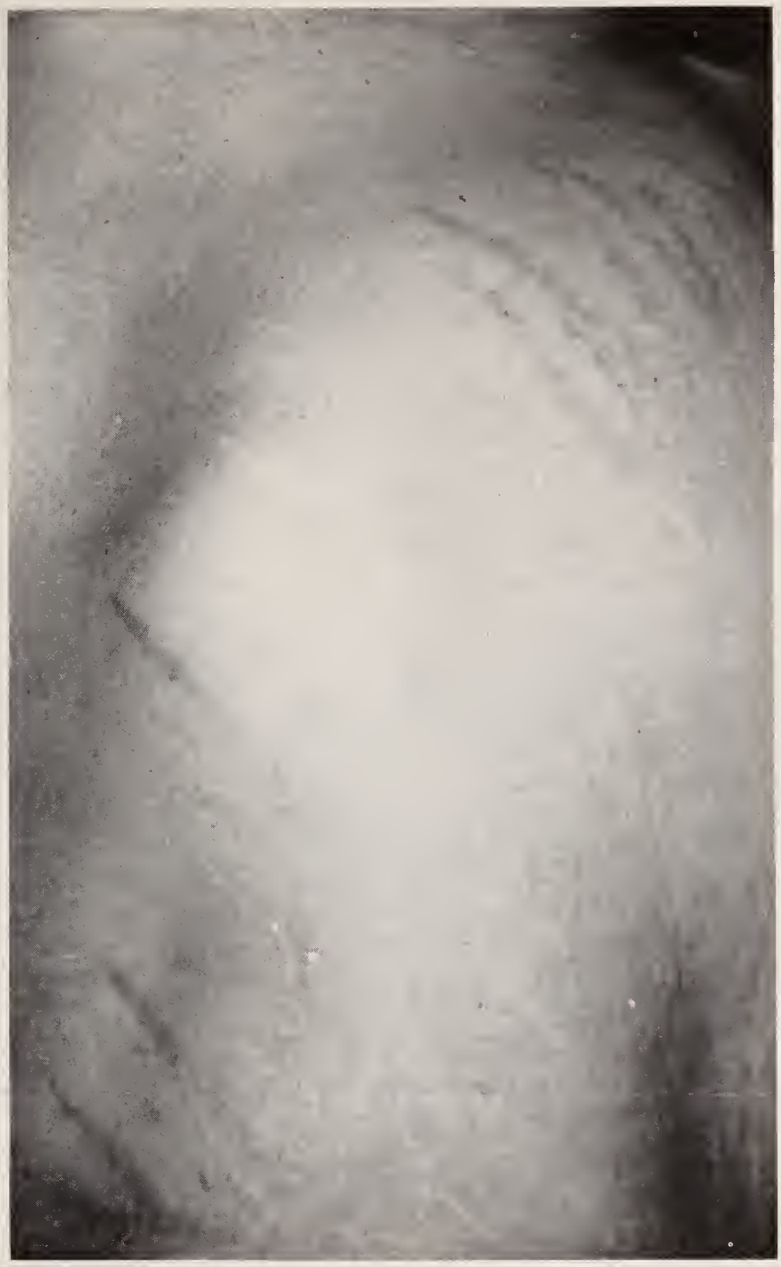

Fig. 6 (Case 2).—Note extensive striae over the hips.

of the examination the cardiovascular system was found to be normal. She presented the usual skeletal abnormalities of the Marfan syndrome, including a very narrow and high arched palate, genu recurvatum, long arms and legs, arachnodactyly, etc. She presented atrophic striae, similar to those seen in her brother (Case 1), distributed over the breasts, upper thorax, hips, buttocks, thighs, and lower abdomen (Fig. 6).

Case 3.—This patient is a 23-year-old white male, a descendant of Lucy Hanks, via a half brother of Nancy Hanks (Lincoln's mother). He is of stocky build and measured 5 feet 8 inches in height. Kyphosis was moderate. No chest deformity was present, and loose jointedness was not evident. He presented a narrow high arched palate, but no discernible dental abnormalities were noted. His younger brother presented an extremely high arched palate and the same peculiar dental abnormality seen in Case 1; namely, a linguoversion to the arch of the second left maxillary bicuspid. His father (a Hanks) died of heart disease at 49 years of age. Cardiovascular disease was not present in this patient, and there was no eye involvement. The Hanks

family appears to escape ocular complications. Atrophic striae were present over the buttocks, lower abdomen, and thighs. In addition he presented fine striae running parallel to each other at the outer portions of both axillae, and these resembled somewhat pseudoxanthoma elasticum in appearance. We tentatively classify this patient as a forme fruste of Marfan's syndrome because of the genetic origin of his skeletal characteristics.

Histologic Examination.—"The changes were similar to Case 1 but with some interesting differences. In Case 1, there were quite a few thin wavy elastic fibers of quite different character, but running in similar direction as the normal fibers in the edge of the striae, but in this case there are numerous thin and thicker elastic fibers forming rather dense bundles, but all running to right angles to the old fibers and therefore also at right angles to the direction of the section. Thus they are chopped into short cross sections by the microtome knife. (This must not be misinterpreted as meaning "fragmentation" of fibers.)"

Dr. Pinkus poses the question as to why there are so many fibers in these cases, especially Case 3, when all the textbooks state that striae show a lack of elastic fibers. It occurred to him that some of the authors made no very definite statements about the kind and age of striae they examined and that they were probably examining striae gravidarum. Dr. Pinkus considered the possibility that the histologic changes observed could be distinct features of the striae in Marfan's syndrome, but feels them more likely to be just a feature of striae arising in young individuals who are still growing and deposit new elastic fibers after the formation of the striae. He sites as further evidence of this view the fact that one sees new elastic fibers in scars in children, but later in life in surgical scars there is usually an absence of elastic fibers for many years and sometimes always.

CASE 4.—The patient, a white male in his middle forties (of Hanks descent), measured 6 feet, 3¾ inches (Lincoln's exact height); he was thin, with marked kyphosis and pigeon breast, long arms and legs, large hands and feet with arachnodactyly of the fingers. He presented a moderately high arched palate, but no dental abnormalities were noted. He was completely unaware of his striae, but upon examination we noted several fine atophic white striae across the midline of the back, sacral area, and hips. These were not nearly as abundant or extensive as those seen in the previous three cases, and it is easy to understand why they were overlooked by the patient. No apparent ocular or cardiovascular disease was noted at the time of the examination. He was loose jointed with genu recurvatum.

Comment

McKusick [5,6] believes that a single mutant gene is responsible for all the many manifestations of this syndrome. He suggests a basic biochemical defect in some element of the connective tissue and that this results in the many symptoms. He feels that at present the defect is yet to be identified, but that the disorder is an abiotrophy of elastic tissue; the involved tissues wear out prematurely, and thus the various signs and symptoms result.

Few references have appeared in the literature relating cutaneous findings to Marfan's syndrome. Fewer of these reports directly associate skin disease with the disorder. The long tapering fingers and toes, scant subcutaneous fat, laxness of ligaments (double jointed), high arched palate, webbing of fingers, relaxation of the ligaments, etc., are considered usual in this syndrome. Bean and Fleming [7] reported purplish-brown thickened, scaly areas of the feet and hyperkeratoses of the toes, but they felt that the findings were probably coincidental with Marfan's syndrome and could best be explained by vascular or trophic factors unrelated to the disease. Sutton [8] in his textbook mentions facial asymmetry, a thin face associated with a sharp chin and nose off center. In 1952, Storck [9] presented a case of Marfan's syndrome with lesions subsequently thought to be identical with Miescher's elastoma.

Bluefarb [10] reported a single case with Raynaud's phenomenon with acrosclerosis, and Zachary Felsher in discussing this patient commented on the scleroderma-like changes which he believed pointed to changes in collagen or in ground substance, associated with changes in elastic tissue. He is quoted as saying, "Some of the changes in the aorta point to the elastic tissue as the primary site of the pathology."

It was not until Kachele [11] reported a case in 1960 that we have cutaneous findings that can be accepted as part of the Marfan syndrome. His report helps explain the pathogenesis of the disorder. Kachele's case was

typical of Marfan's syndrome, including ectopia lentis, and is described as exhibiting symmetrical atrophic striae over the upper thorax, lower abdomen, and buttocks. Clinically and histologically these were typical striae distensae elasticae. Since it is now accepted that the pathogenesis of Marfan's syndrome is truly an abiotrophy of elastic tissue, Kachele suggested that striae in Marfan's syndrome presented additional evidence in support of this concept. He believes that the appearance of ectopia lentis was based on the same defect responsible for the cardiovascular abnormalities. Since Kachele's report, two additional cases have been reported, one by McKusick,[12] the second by Bean.[13] McKusick merely shows a photograph of his patient and comments that it is similar to Kachele's case. Bean's case was a lad with Marfan's syndrome who had two rather remarkably placed examples of striation of the skin, a single line at an odd angle over the prominence of both shoulders.

In reporting four additional cases of striae associated with Marfan's syndrome, along with detailed histologic studies, we feel that additional evidence has been presented to support the view that this syndrome is a disorder of elastic tissue. Two of our four cases were part of a separate study of Lincoln's living relatives. Striae in these two patients helped to confirm the diagnosis of Marfan's syndrome particularly in the one case we consider a forme fruste. Furthermore, we feel that striae in Marfan's syndrome occur much more frequently than has been reported to this time, and if one examines these cases carefully, undoubtedly more patients with striae will be observed. McKusick[14] shares this view, and since reporting his case he has encountered several additional patients with striae.

In our cases we could not be certain how long striae had been present. We feel, however, that they must have appeared early in life and were not associated with severe stretching of the skin. None of our patients were obese, and striae were not confined to those areas where the skin usually undergoes undue stretching. Poidevin[15] has shown very conclusively that stretch is not the major factor in the production of striae, but they may result from some adrenal cortical hyperactivity.

In addition to cutaneous findings, we encountered two patients with unusual abnormalities of the teeth, i.e., complete linguoversion to the arch of the second left maxillary bicuspid. To our knowledge, these abnormalities have not previously been reported in Marfan's syndrome although malocclusion of teeth[16] and hutchinsonian-like incisors have been observed.[17]

Further studies of the family pedigree are being carried out at the present time. Moreover, chromosome studies that have been previously reported in isolated cases[18] are being done on several affected individuals.

Summary and Conclusion

Four cases of Marfan's syndrome with striae distensae are reported.

The histologic aspects are discussed in detail.

Two cases of peculiar dental abnormalities associated with his syndrome are reported.

The dermatological aspects herewith reported affords further evidence that this syndrome demonstrates some abnormality in the structure or functioning of the elastic tissue. It is suggested that the skin be considered, along with the cardiovascular, skeletal, and ocular systems, as a major system involved in this syndrome.

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WORKS OF MERCY (DELLA ROBBIA), HOSPITAL OF THE STUMP, PISTOIA, ITALY see page 80

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November-December 1963

Patient with Marfan's syndrome; his deformities are similar to Lincoln's. Photograph courtesy of A. M. Gordon.
—A. B. Loveman et al., Arch. Derm. 87:428, 1963.

Characteristics of Marfan's syndrome

Bones and joints: dolichocephaly; gothic palate (often associated with high-pitched voice, a Lincoln characteristic); prognathism; kyphosis, sometimes with scoliosis and fusion of lumbar vertebrae; funnel chest or pigeon breast; elongated limbs; arachnodactyly; loose-jointedness, sometimes with genu recurvatum; flat-foot, metatarsus varus and hallux valgus (indicated by Lincoln's own tracing of his feet for the use of a bootmaker).

Cardiovascular defects (present in Lincoln's direct and collateral descendants): aortic aneurysm and other arterial abnormalities; defective valves and septa.

Ocular abnormalities: ectopia lentis; myopia; strabismus.

Miscellaneous soft-tissue defects: scant subcutaneous fat; sad facies; atrophic striae; hernia; lax ligaments and tendons; large, ill-formed ears.

Courtesy of Bettmann Archive

A famous case of Marfan's Syndrome

ABRAHAM LINCOLN was probably the most famous victim of Marfan's syndrome, although the disease was not described until 31 years after his death. He was noted for his physical prowess, but was nevertheless handicapped by deformities characteristic of Marfan's syndrome, as pointed out by A. M. Gordon (University of Louisville).

The typical patient has the same skeletal and soft-tissue peculiarities as Lincoln, whose grotesque appearance was colorfully described by his friend and biographer, William H. Herndon:

> "Mr. Lincoln was six feet four inches high...thin, sinewy, rawboned, thin through the breast to the back, and narrow across the shoulders; standing he leaned forward—was what may be called stoop shouldered, inclining to the consumptive build...His structure was loose and leathery; his body shrunk and shrivelled; he had dark skin, dark hair and looked woe-struck...When he walked he moved cautiously but firmly; his long arms and giant hands swung down by his side...He put his whole foot flat down on the ground at once, not landing on the heel; he likewise lifted his foot all at once, not rising from the toe...His legs and arms were abnormally, unnaturally long and in undue proportion to the remainder of his body. It was only when he stood up that he loomed over other men. Mr. Lincoln's head was long and tall...his long sallow face was wrinkled and dry...his ears large, and ran down almost at right angles from his head; his lower lip was thick, hanging and undercurved, while his chin reached for the lip, upcurved...."

Marfan's syndrome is a protean disorder that has repercussions throughout the body. Skeletal, ocular and cardiovascular abnormalities are its most familiar manifestations, but other systems are also affected. Weak aponeuroses may predispose to herniation, and lax or redundant ligaments to subluxation; short, inelastic ligaments with insufficient musculature and subcutaneous tissue may cause contracture of joints. A lugubrious cast of countenance, like Lincoln's, is attributable to flabby tissues. Malformations of the lungs and of the genitourinary tract have been reported. Striae distensae elasticae, i.e., symmetrical lines comparable to the lineae albicantes of pregnancy, develop over the thorax, abdomen and buttocks; they are caused by atrophy of elastin fibrils.

Arachnodactyly is the official name for Marfan's syndrome, as recommended in *Standard Nomenclature of Diseases* and the *Index Medicus*. Yet some apparently normal people have spidery fingers without other features of the syndrome, and not all Marfan patients have arachnodactyly. More specific criteria are an arm span greater than the height, and disproportionately long legs, with the distance from symphysis pubis to planta considerably greater than to the vertex. Metacarpals measuring more than 8 cm. are also a diagnostic feature. Elongation of the ribs results in the buckling of funnel chest or pigeon breast. Lincoln's flat-looking chest may actually have been concave. By skeletal criteria he qualifies for the diagnosis.

In about one patient out of three, weakness of the suspensory ligaments causes ectopia lentis, often with coloboma and sometimes terminating in blindness brought on

by retinal detachment, cataract or glaucoma. The early stages have been observed in infancy or childhood, but the graver sequelae never develop before adult life. Vision is usually defective even when there are no serious structural defects: Lincoln wore strong glasses to compensate for his myopia; he had episodes of diplopia; and his portraits reveal right hypertropia.

Cardiovascular disease is the chief cause of early death in Marfan's syndrome. Cystic and myxomatous degeneration of the media of the thoracic aorta causes dilatation and sometimes fatal dissecting aneurysm. The pulmonary artery and the abdominal aorta may also be involved. Incompetency of valve cusps and septa results in heart block, congestive failure, and occasionally the tetralogy of Fallot. A diagnosis of rheumatic valvulitis may be made in error because changes observed in the electrocardiogram are similar.

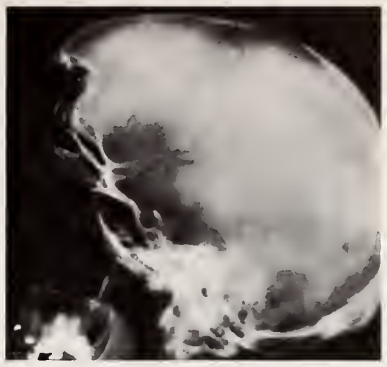
Lincoln's circulatory system seems to have functioned well, but three of his four sons probably had serious cardiovascular disease. Willie, who looked like his father, died at 12 of pneumonia, a frequent complication in Marfan's syndrome. Tad died at 18 "of pleurisy," presumably in heart failure with cardiac asthma. Edward, who inherited Lincoln's build, died at 3½ of unknown causes. Robert, the only surviving son, lived to a ripe old age. His stocky frame was seemingly unaffected by the Marfan trait, but his son, Abraham II, was the image of his grandfather and died at 17 with "fluid" in the chest.

A genetic disease

The multiple expressions of Marfan's syndrome can be described as an abiotrophy of connective tissue—a deficiency of collagen and elastin fibrils. Progress of the disease is indicated by abnormally high urinary excretion of mucopolysaccharides and by imbalance in their composition, notably in the relative amounts of chondroitin sulfate and keratosulfate, which are essential to the formation of collagen and the outer coating of elastic fibers.

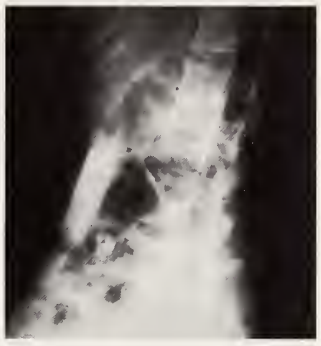
The magnitude of the metabolic error is more precisely estimated by urinary excretion of hydroxyproline, as this amino acid is an essential component of collagen but is not found in other body proteins. In Marfan's syndrome, excretion of hydroxyproline is abnormally high during childhood, and is usually higher than normal during adolescence. This means that at times of rapid growth a significant number of collagen fibrils are broken down or fail to mature. The skeleton, the suspensory ligament of the lens, and the aorta, which are the sites most frequently and most ominously involved, are rich in collagen. Looked at in this way, Marfan's syndrome is a true collagen disease, though not in the same category with the autoimmune, inflammatory collagen diseases, in which high excretion of hydroxyproline is not a factor.

The Marchesani syndrome, typically identified by brachycephaly and brachydactyly, is in some ways the



—X-rays courtesy of Nathaniel Finby, St. Luke's Hospital, New York.

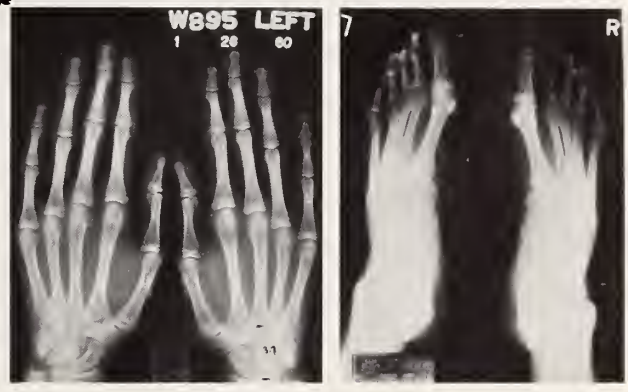
Skull of Marfan patient, showing large sinuses.



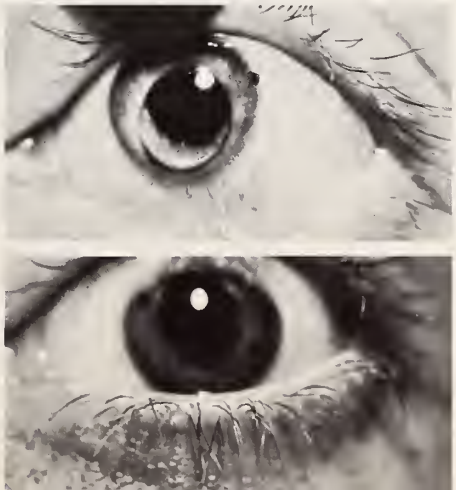
Widened spinal canal, with low sacral junction.

Aneurysm of the aorta, threatening dissection.

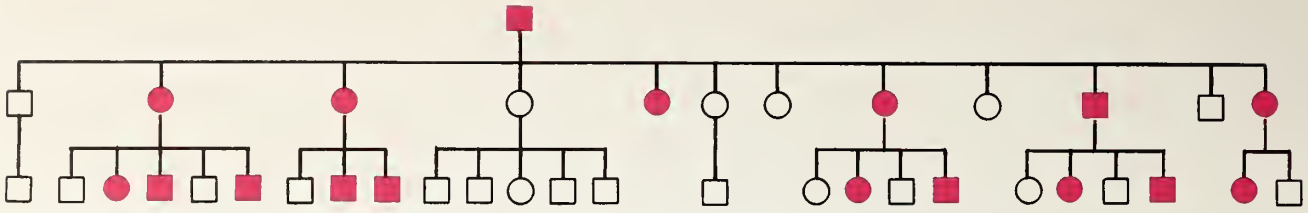
Hallux valgus and abnormally long digits.



—G. E. Kachele, Arch. Ophthal. (Chic.) 64:135, 1960.



(above) Ectopia lentis and glaucoma in Marfan's syndrome. (below) After iridectomy and removal of lens, corrected vision was 20/40.



Pedigree in Marfan's syndrome. Children do not usually inherit the trait if it does not appear in the parents.
—F. C. Lutman and J. V. Neel, Arch. Ophthal. (Chic.) 41:276, 1949.

antithesis of Marfan's syndrome, yet the two conditions are alike in predisposing to myopia, ectopia lentis and glaucoma. In a Marfan pedigree reported by Dorrance Bowers (Knox Clinic, Kelowna, B.C.), one subject had ectopia lentis, brachycephaly and brachydactyly at age 3, but developed long limbs and skull in the next five years. He had passed from one syndrome into the other.

A diagnosis of Marfan's syndrome can be made confidently if the eyes, bones and aorta have specific deformities, but not if only one of these structures is affected. In Lincoln's case, the skeletal abnormality was clear-cut, and the defective vision adds suggestive evidence. Confirmation can be found in the family pedigree.

His mother, Nancy Hanks, was the illegitimate child of Lucy Hanks, who was part Choctaw. (Lincoln told Herndon that his maternal grandfather belonged to one of the first families of Virginia.) Like her son, Nancy Hanks was tall, thin and stoop-shouldered, and her facial expression was habitually sad. Gordon has identified Marfan stigmata in the issue of Lucy's marriage to Henry Sparrow, one of whom had Lincoln's height and build, with arachnodactyly, kyphosis, pigeon breast, gothic palate and atrophic striae. Another member of the family died of heart disease; two of his sons, although of stocky build, were marked by gothic palate and atrophic striae.

It is generally agreed that the trait is transmitted by a single dominant, autosomal gene with extremely variable penetrance. The suggestion has been made that the gene may be localized on chromosome 18, 21 or 22, one of which may be elongated or have enlarged satellites in some cases; but these anomalies may be purely coincidental. The abnormal gene may occasionally arise by sporadic mutation, and it is unquestionably perpetuated in a *forme fruste* by pyknic individuals, who apparently have suppressor genes or are of the Marchesani type. Such inheritance is well illustrated by stocky, healthy Robert Lincoln and his unfortunate son.

The fact that a few patients have been outstandingly intelligent has induced recent observers to consider high I.Q. another expression of the syndrome. Curiously enough, however, mental deficiency has in the past been a more frequent finding than intellectual superiority. Presumably the distribution of intelligence is the same as in the general population and is governed by other factors.

The defects are partially correctable

The manifestations of Marfan's syndrome are often crippling and are sometimes fatal. Gross deformities of the limbs are to some extent remediable by surgery. Lincoln was adept at handball, wrestling and splitting fence rails, but he would surely have moved about more comfortably if his feet had had proper attention. His eye defects, ascribed by legend to reading by the light of a pine knot and by our contemporary theory to Marfan's syndrome, were not totally relieved by glasses, and would be only partially correctable today. Fortunately he did not suffer from ectopia lentis and its sequelae, for which surgery would now be imperative despite poor results in many of the cases. Modern cardiac surgery might have prolonged life for three of his sons and his grandson, although collagen-poor cardiovascular tissue heals inadequately after incision.

Proved cases of Marfan's syndrome are comparatively rare; in the literature to date, fewer than 500 diagnoses have been made. But whenever the abnormal gene is present, it is capable of attacking with machine gun thoroughness. In a Pennsylvania pedigree of 22 known members in four generations, 14 were virtually incapacitated by ectopia lentis, cardiovascular disease, or both. Five died prematurely of cardiovascular disease, and both lenses had to be removed in 4 cases.

There would probably be many more cases if this disability were not an obstacle to matrimony and a cause of early death. Procreation and longevity seem to be less curtailed in women than in men. Marfan patients contemplating marriage should be made aware that the abnormal gene is transmitted to future generations. END

Recognition of Marfan's Syndrome

1896	B. J. A. Marfan described spidery fingers and poor muscular development.
1902	E. C. Achard named the syndrome arachnodactyly; H. Méry and L. Babonneix observed subluxation of the patella in Marfan's original patient.
1912	V. Salle reported patent foramen ovale in conjunction with arachnodactyly.
1914	F. Boerger noted ectopia lentis, arched palate, and abnormalities of the lungs and genitourinary tract.
1926	R. K. Piper and E. Irvine-Jones made a detailed study of the cardiac anomalies.
1931	H. Weve studied genetic aspects.
1955	V. A. McKusick published a comprehensive review of the syndrome.
1962	A. M. Gordon identified Abraham Lincoln with Marfan's syndrome.

Kitchener, Ontario
February 5, 1964

Did Lincoln Have an Often-Fatal Illness?

By JERRY KLEIN

LOUISVILLE, Ky. (NEA)—If Abraham Lincoln had not died of an assassin's bullet, he might very well have gone blind or incurred a disease of the respiratory system that could have claimed his life.

According to a report in the current issue of a popular medical journal, the martyred president was the victim of an "uncommon, but not rare" disorder called Marfan's disease.

A bereditary ailment not identified until long after Lincoln's time, the typical signs of Marfan's include many traits identified so strongly with the Civil War president that other persons with them are still called "Lincolnesque."

* * *

Writing in the Charles Pfizer and Co. journal, Spectrum, Dr. Abraham M. Gordon of Louisville, Ky., reports on years of research proving that Lincoln "was probably the most famous victim of Marfan's. . . ."

As such, if he had not been murdered at 56, Lincoln was prone to serious maladies that might have disabled him or shortened his normal life expectancy.

An internist and faculty member of the University of Louisville, Gordon believes Lincoln inherited the crippling disorder from his mother. He, in turn, passed Marfan's down to his sons and grandson — most of whom died in youth.

* * *

The typical signs of this illness read like an eyewitness description of Abraham Lincoln. They include: long limbs, crooked knees, flat feet, a slender build with narrow chest, thin neck and humped shoulders; a sad face with a pointed chin, high cheek bones and large ears, weak eyes, a loose-jointedness with lax tendons and ligaments that produce an awkward, rolling gait; a long head and a high-pitched voice.

Lincoln's feet caused him continual discomfort. He was forever slipping out of his shoes to walk barefoot, even in the White House. A sketch he drew to send to a bootmaker in New York shows that his flat feet would need a size 16½ shoe today, and that his big toes were turned in.

* * *

By the age of 47, Lincoln required very strong eyeglasses, and he also was color-blind. Dr. Gordon points to Lincoln's nearsightedness, bouts of double vision and the fact that pictures show his right pupil higher than the left as evidence that he eventually might have suffered the worst eye trouble of Marfan's disease. These are glaucoma, cataracts or retina detachment—ending in blindness.

"The offspring of a happy marriage between a derrick and a windmill" was the way one contemporary described Lincoln's jerky movements. "When he was in a good humor," remarked another observer, "I always expected him to flap his ears like a good-natured elephant."

* * *

Dr. Gordon notes that all of Lincoln's sons "seem to have had Marfan's" and that three of the four died before reaching adulthood, probably of the heart congestion typical of the disorder.

Officially, the cause of little Edward's death at 3½ was unknown; William's death was attributed to pneumonia, "a frequent complication in Marfan's"; and Tad (who also suffered a cleft palate) succumbed to "pleurisy" which also can be traced to the cardiovascular disease that is "the chief cause of death in Marfan's."

Robert, the only one to reach manhood, had the characteristic visual weakness and as a child was teased by other children for his crossed eyes. Grandson Abraham II, "the image" of the president, died at 17 of a heart and lung disorder that produced "fluid" in the chest, and again characteristic of Marfan's.

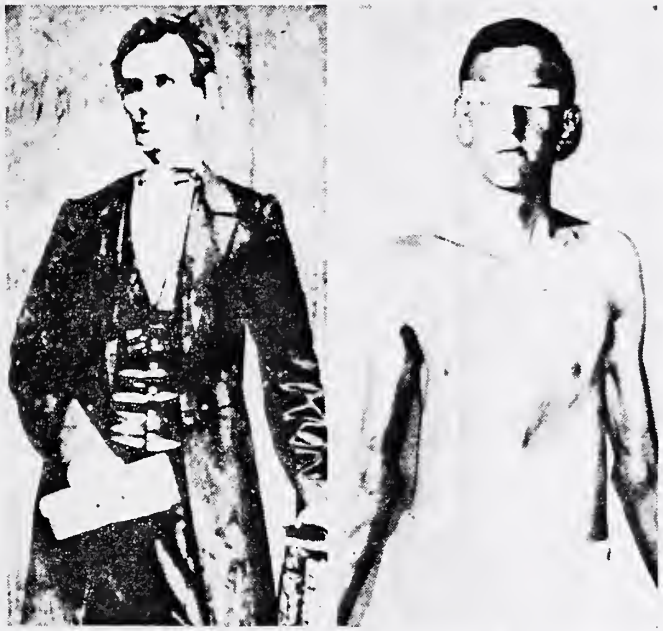
* * *

Dr. Gordon started studying Marfan's in depth when he happened to see his first patient with the ailment at the same time he was reading a biography of Lincoln. "Something clicked in my mind," says the assistant clinical professor at Louisville's medical school, and he has been investigating the disorder since.

Doing so has taken the physician into Illinois, Kentucky, Ohio and Virginia to examine offshoots of the Lincoln family tree. He has found some amazingly similar cases among these people. One of Marfan's victims at 14 was a writer of beautiful poetry; a 15-year-old victim has a genius intelligence quotient and can do college work.

"Unusual qualities of mind and spirit appear in a great many Marfan's people," the magazine reports. They are persons with "high mental ability who have a singular outlook on life." Certainly, the murdered president would be an outstanding example of this.

The Spectrum article says that the strange disease was named in 1896 after B. J. A. Marfan, a French physician who first described the spidery fingers and slack muscles that are typical of it.



Abraham Lincoln (left) is compared with latter-day victim of Marfan's disease.

Evening Bulletin
2-7-64

A 'Diagnosis' of Lincoln — and an Answer

Dr. Herman Blum is well-known for his collection of Lincolniana and for his studies in the life of the martyred President. Tomorrow night, at the Union League, Dr. Blum will receive the Annual Award of the Lincoln Civil War Society, in recognition of his contributions to our knowledge of Lincoln.

By DR. HERMAN BLUM

Is the heroic image of Abraham Lincoln, cherished by the American people so ardently for 100 years, about to be adulterated by a medical portrayal of him by a physician whose diagnosis is ruthlessly eroding the generally accepted ideas about our 16th President?

An outcry has arisen from historians who have disputed a recently published diagnosis of Lincoln, which in essence, attributes his greatness to a strange disease, known as Marfan's Syndrome, an affliction which, in 1865, was not only unidentified but absolutely unknown.

Is there evidence that supports such a medical diagnosis and conclusion? None, other than the use of conjecture based on haphazard photographs of Lincoln and the recollections of those who knew him.

On the other hand, the testimony I am about to present clearly indicates that the diagnosis made 98 years after Lincoln was struck down by an assassin's bullet, is based on a mixture of imagination and superficial research.

'Preposterous'

Despite the fact that it is preposterous for a physician to attempt a diagnosis of a patient he has never physically examined, or to theorize about the symptoms of his ailment, after the man has been dead 100 years, the beguiling diagnosis, which has been widely publicized, is causing growing alarm among historians.

The "Physician's Bulletin," issued last year by the pharmaceutical house of Eli Lilly & Co., of Indianapolis, featured the diagnosis of A. M. Gordon, M. D., of Lexington, Kentucky. Dr. Gordon's analysis of Lincoln's symptoms spoke of Lincoln's "long, and sad face, sunken chest, misshapen ears, loose spidery arms, immense hands, gangly appearance, uncouth movements, and high-pitched, almost girlish voice." In addition to these abnormal characteristics, one specific aspect of the Marfan Syndrome was indicated: Its victim was usually very precocious.

The 'Syndrome'

Marfan was a French pediatrician who identified the ailment in 1896, 31 years after Lincoln's death. Marfan's Syndrome is defined as a hereditary disease of the body's connective tissues, the bones, muscles, and ligaments, the symptoms occurring together, affecting the eyes, heart and the skeletal structure.

The Lilly Bulletin further describes Mr. Lincoln as follows: "He had a shambling, loose, irregular, almost unsteady gait. He was a tall, lank, lean man over six feet in height, with dulous arms, terminating in hands of extraordinary dimensions which, however, were far exceeded in proportion to his feet. Indeed, he was pictured by his partner (Herndon) and long time friend, as being 'thin, sinewy, raw-boned, thin through the breast to the back, and narrow across the shoulders. His legs and arms were abnormally, unnaturally long in undue proportion to the remainder of his body; his ears large and ran down almost at right angles from his head.'"

In fact, cartoonists of his day always portrayed him as an apelike creature which Dr. Gordon defines medically as "dolichocephalic, flat-footed, kyphotic, narrow chested, and prognatic."

'Derrick and Windmill'

"Lincoln suffered from strabismus," states Dr. Gordon, "and at 51 years of age had several attacks of double vision. Was this 'ectopid lentis' which is often found in Marfan's Syndrome? He also had a high-pitched voice, another characteristic of this anomaly. His ears were large and malformed. Indeed, an artist who painted his portrait complained of Lincoln's asymmetrical features. Is there some significance to the fact that an anti-Lincoln pamphlet of 1864 described him as follows: 'His anatomy is composed mostly of bones, and, when walking, he resembles the offspring of a happy marriage between a derrick and a windmill. When speaking, he reminds one of the old signal telegraph that used to stand on Staten Island. His head is shaped something like a rutabago (sic) and his complexion is that of a Saratoga trunk. His hands and feet are plenty large, and in society he has the air of having too many of them.'

"Although Lincoln was free of cardiovascular disease, it is true that any one of the three major components (skeletal, ocular, or cardiovascular) may be present with little or no involvement of the two areas."

A Hint of Malice

As to the hereditary aspect of the disease, Dr. Gordon mentions that Lincoln's mother, Nancy Hanks, had elongated limbs, that she died at an early age, probably of this skeletal disorder; that Mr. Lincoln's three children died early in life of ailments that were otherwise described but could very well have been the syndrome.

How should one who loves Lincoln react to all this? He is

Above, a photograph of Lincoln's life mask in the Blumhoven Library, showing none of the abnormalities so often shown in caricatures. Right, a casting made from Lincoln's right hand, from the same collection. (Photographs by Dr. Herman Blum)

hero had no television personality or beautiful movie profile. He knows that Lincoln had a dry wrinkled skin and a number of moles; but the world is full of people whose bodies are not perfect.

Lincoln was no exception, but to intimate that he had "rocks in his head" or that his genius and intellect were due to his affliction, simply smacks of ill-concealed malice or ignorance. So how does a layman, who reveres Lincoln, go about disputing the physician's studied verdict that Lincoln's humanity, statesmanship, and courage were all or part of what he harvested from Marfan's Syndrome?

One sure way of nullifying the diagnosis of Lincoln's detractors, is to present factual evidence — something one can see and feel and measure. This we offer as follows:

In the Memorial Gallery of Blumhaven Library in Philadelphia there is an original casting of a Lincoln face mask. It was made by the sculptor, Leonard Volk, in 1860, and acquired by Blumhaven Library in 1950 after the famous Lincoln collector, Oliver R. Barrett, had it in his possession for 60 years.

This life mask reveals a beardless face, with no structural details concealed by hair. Every line of it is clear and sharp. One can be sure that it reflects exactly what Lincoln looked like.

This life mask was minutely measured by Dr. T. D. Stewart, U. S. National Museum, Washington. Dr. Stewart did not observe or note any skeletal disorder or deviation of the eyes. His critical examination of the mask and the resulting conclusions were duplicated by other noted experts, among them Dr. Charles M. Sturtevant, of Braintree, Mass., former chief surgeon of the Frankford Hospital in Philadelphia; by Dr. Hilton H. Shules, Carmel, California; Dr. Mark Daniel, of New York, and others.

All agreed that there was no deformity indicated in the actual replica of Lincoln's face, that the ears were not excessively large, abnormal, or misshapen, and that there was no deviation of the eyes.

Besides the life mask of Lincoln, Blumhaven Library has the casting of his two hands. The physicians who examined these two castings agreed that the fists are not spidery or bony. The right hand is closed and is powerful in appearance. The left hand, gripping a broom handle, is a striking reflection of the man's physical strength.

A 'Sunken' Chest?

When it comes down to practical observations, no one really knows exactly if and how Lincoln's chest was "sunken" or how prominent his ears were. Judging from the casts of his hands, they were not spidery or immense for a man 6 ft. 4 inches tall.

voice" as Dr. William Jayne, of Springfield, Illinois, describes it, "began in a rather high pitch, then modulated and remained in the upper, middle range." But his voice had great carrying power. His teeth were excellent and he did not wear glasses to read until he was two years above the average optical age of 45.

Dr. Gordon is wrong when he writes of Lincoln's "several attacks of double vision." The one and only time Lincoln had this trouble was during a period of marked fatigue while resting after the excitement of his election in 1860. Only Lincoln and his wife knew about this, and later, in the White House, Noah Brooks, a close friend, was the only person who repeated the story. It was years later when Brooks told the story.

Will the medical verdict which is now being discussed by physicians and pharmacists, and Lincoln students, tarnish the heroic image of Abraham Lincoln? No, it will take more than Marfan's Syndrome to rob him of the glory that is his.

To the medical researchers who are trying to define the source of his greatness, I respectfully submit Richard Watson Gilder's moving description of the Lincoln bronze life mask, which is being offered as evidence of what Mr. Lincoln was really like. The poem was written by Gilder in 1909 and reads:

"This bronze doth keep the very form and mold
Of our great martyr's face. Yes, this is he;
That brow all wisdom, all benignity;
That human, humorous mouth; all the summer's gold;
That spirit fit for sorrow, as the sea
For storms to beat. Oh, the lone agony
Those silent, patient lips too well foretold.
Yes, this is he who ruled a world of men
As might some prophet of the elder day—
Brooding above the tempest and the fray
With deep-eyed thought and more than mortal ken.
A power was his beyond the touch of art
Or armed strength — his pure and mighty heart."

NEWS RELEASE from
THE AMERICAN MEDICAL ASSOCIATION
535 North Dearborn Street, Chicago, Illinois 60610 • • Area Code 312 527-1500

For A.M. Release Friday, February 14, 1964

CONTENTS

EVIDENCE SUGGESTS LINCOLN HAD MARFAN SYNDROME

CHICAGO--Genetic and genealogic evidence "suggests strongly" that Abraham Lincoln's unusual body build was due to Marfan syndrome, Dr. Harold Schwartz, Huntington Park, Calif., said today.

The syndrome is an inherited disorder of the connective tissue which affects skeletal development, vision, and the cardiovascular system.

Dr. Schwartz' interest in Lincoln was aroused by a seven-year-old boy with the classic features of Marfan syndrome whom he saw in 1959 and later learned was connected with the Lincoln lineage.

The boy's genealogy was traced back nine generations to Mordecai Lincoln II, born in 1686, who was also the great-great-grandfather of President Lincoln.

The mother and 15-year-old sister of the patient also had atypical Marfan syndrome, and a search of the archives revealed other scattered kin of Lincoln with Marfan characteristics as would be expected in an inherited disorder, Dr. Schwartz reported in the current (Feb. 15) Journal of the American Medical Association.

The syndrome is marked by excessive long-bone growth which produces disproportionately elongated arms, legs, fingers, and toes, with a long head and facial features, according to Dr. Schwartz.

Overgrowth of the ribs may lead to various chest deformities or a very thin chest. Weakness and laxness of ligaments, tendons and other tissue produce loose-jointedness, malpositioned ears and other symptoms. There also is a lack of subcutaneous fat.

In the skeletal system, Lincoln manifested many of the characteristics of Marfan syndrome and he also suffered from a squint and severe farsightedness which one physician considered "beyond the range of the acquired form," Dr. Schwartz reported.

Many well-documented and repeated descriptions of the Civil War president show that his extremities were disproportionately long compared with his height of six feet, four inches, and his head was thin and elongated with large ears set at a wide angle, according to the California physician.

Lincoln also was described as having a "sunken breast" and "spiderlike legs" which are typical of the syndrome first recognized by Bernard-Jean-Antonin Marfan in 1896.

Dr. Schwartz cited the observation of William H. Herndon, Lincoln's former law associate and later biographer, that in sitting down on common chairs, Lincoln was no taller than ordinary men from the chair to the crown of his head and it was only when he stood up that he loomed above other men.

Certain pictures illustrate that when seated, the height of Lincoln's knees was considerably above the plane of his thighs, further indicating the excessive elongation of the lower legs, Dr. Schwartz added.

Casts of Lincoln's hands further demonstrate features of the syndrome, according to the Journal report. The casts indicate that Lincoln's left hand was longer than his right, and the thumb on the longer hand was shorter than the thumb on the shorter hand. Such asymmetries are characteristic of the syndrome, Dr. Schwartz commented.

It appears that the criteria for Marfan syndrome are fulfilled in the hands and stature of Lincoln, he said.

Relatively little has been written about the personality of persons with Marfan syndrome, Dr. Schwartz said, but many case reports contain intimations of patterns of superior intelligence and other special talents.

A case reported in 1918 described a six-year-old boy with the syndrome as always undernourished but sensitive and mentally advanced for his age, with a quaint way of expressing himself and a sense of humor of his own.

"A more succinct and specific characterization of the known Lincoln personality and uniqueness of expression would be difficult to formulate," Dr. Schwartz said.

The Marfan syndrome was linked to Lincoln previously by Dr. A.M. Gordon in 1962 but he attributed the genetic defect to the maternal line through Lincoln's mother, Nancy Hanks. Dr. Schwartz said the new genetic evidence makes the maternal theory seem unlikely.

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URGE STOCKPILE OF ANTISERUM
FOR TYPE E BOTULISM

CHICAGO--Recent outbreaks of type E botulism indicate that sufficient quantities of type E antiserum should be readily available for any future emergency.

The recommendation was made in the current (Feb. 15) Journal of the American Medical Association.

The only botulism antiserum now available in this country includes types A and B, but not type E.

"The commercial availability of antitoxin that will include, or independently provide for, protection against type E intoxication is also desirable and under active discussion by the appropriate authorities," a Journal editorial pointed out. "At present only a limited supply is available from Canadian and Danish sources."

Six immunologic types of Clostridium botulinum, the organism that causes botulism, have been identified and are designated as type A through F. Almost all human botulism has resulted from types A, B, and E.

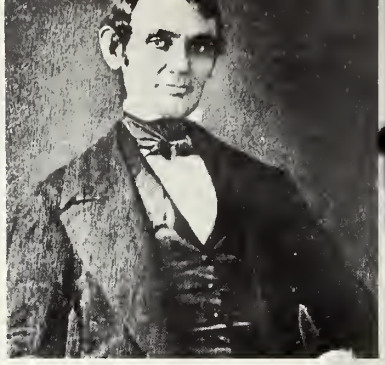
Since 1932 there have been 7 outbreaks of type E botulism in the United States, resulting in 15 deaths, according to a Journal report. These occurred in 1932, 1934, 1941, 1960, 1961, and 1963 when two outbreaks occurred.

"Prior to 1960 there had been only one reported instance of botulism from food commercially processed or canned in this country since 1925," the Journal editorial said.

"There have always been a number of small epidemics yearly, but these have resulted primarily from home-prepared foods, with limited potential distribution.

"The well-publicized occurrence of cases resulting from canned tuna in Detroit, and from prepared whitefish originating in the Great Lakes region, has serious implications. Both outbreaks appeared to have been due to the type E toxin, which had not been common in this country previously.

— Bettmann Archive



Lincoln and Marfan's Syndrome

Descendant of Lucy Hanks studied by Abraham Gordon (case 4 in Arch. Derm. (Chic.) 87:428, 1963). The resemblance to Lincoln (inset) is apparent.

EVIDENCE INCREASES that Abraham Lincoln's infirmities were characteristic of Marfan's syndrome. An article in the last issue of SPECTRUM (November-December 1963) presented the theory that he inherited the trait from his mother. This idea was first developed by A. M. Gordon (University of Louisville). In J.A.M.A. for February 15, 1964, Harold Schwartz (University of Southern California) subscribed to the diagnosis and cited evidence that the disease may have been transmitted by a paternal ancestor.

Both Schwartz and Gordon have patients who are collateral descendants of Lincoln. Gordon's proband is a great-great-grandson of a first cousin of Lincoln's mother; Schwartz's is the great-great-grandson of a paternal fourth cousin. Both patients have arachnodactyly, gothic palate, chest deformity and loose joints. In addition, Gordon's patient has characteristic atrophic striae and Lincoln's exact height—6 feet, 3¾ inches. Schwartz's patient may eventually attain comparable height, as he measures 5 feet 4 at the age of 11.

Biographers have been struck by Lincoln's resemblance to his mother, Nancy Hanks, who was tall, stoop-shouldered, dark-skinned and dark-haired. Her forehead, chin and cheek bones were prominent and her expression was habitually sad. Lincoln often said that he had inherited his mother's qualities. The only physical traits that could be attributed to the father, Thomas Lincoln, were dark hair, muscular strength and poor eyesight. An acquaintance reported that one of Thomas's eyes was blind and the other weak, yet despite this reputed disability his fellow townsmen appointed him road surveyor when he was 38.

Schwartz points out that some of the Lincolns bore "morphologic resemblance to the President." His uncle Mordecai, chiefly known for having died of cold and alcohol (the alcohol being taken for the express purpose of keeping out the cold), fathered three sons who looked like their illustrious cousin. One of these sons, also named Mordecai, was honored by a visit from Abraham during one of his campaign tours. This branch of the family died out in the next generation because, as one member put it, they were "not a marrying set," and they were apparently given to drink. In representatives of a more stable branch, native to Pennsylvania, Ida M. Tarbell saw "the pronounced features made so dear and so familiar to us with the face of Abraham Lincoln."

On the basis of body build and facial resemblance alone it would be unsound to make a generalized diagnosis of Marfan's syndrome in the Lincoln family, either as a *forme fruste* or as a partial expression. But it may be significant that Schwartz found indications of the syndrome in close relatives of his patient. Skeletal abnormalities were suggestive in the mother and sister; like the proband, the maternal grandfather underwent hernia repair, and an aneurysm may possibly have been the cause of his sudden death. The situation appears to be similar in the Hanks line, as a cousin of Gordon's proband had characteristic skeletal abnormalities, and his father died of heart disease.

Editorial comments on Schwartz's article admitted the plausibility of the diagnosis of Marfan's syndrome, and the possibility that it could have been inherited from either the Lincoln or the Hanks side. Although the disease is not a common one, there is the third possibility that it was present in both sides of the family. The expression of the deviant gene would not necessarily be accentuated even if carried by both parents of an affected child, but more children would probably be affected. As concerns the Lincoln-Hanks descendance, the syndrome seems to have been expressed in a sinister form in the President's sons, three of whom died in childhood or adolescence, apparently as a direct or indirect result of cardiovascular disease. The line died out with the only grandson, Abraham II, who succumbed to a congestive disease of the chest at 17. The effect of Marfan's syndrome on the President himself, and on the course of American history, is a matter for speculation. END

SPECTRUM

2323 Louisiana
El Paso, Texas
March 1, 1964

The Editor
The Journal of the American Medical Association
535 North Dearborn Street
Chicago, Illinois

Dear Doctor:

In the February 15th edition of THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION an editorial entitled LINCOLN'S INHERITANCE asks the question:

> "Was it inherited through his mother, Nancy Hanks, as Dr. Gordon suggests, or through his father, Thomas Lincoln, as Dr. Schwartz suggests? The answer to this question would provide Lincoln scholars with much sought-after information...."

The answer to this question is available and is a very brief and simple one: Mr. Lincoln did not inherit Marfan's Syndrome from either parent because he did not have Marfan's Syndrome.

The causes of death of the Lincoln children have long been known to medical historians and especially Lincoln students; none were cardiovascular in origin.

Hanging on the wall of my study before me are autographed photographs of four Army medical officers in uniform and two copies of autopsy reports. Between them the autopsy was performed on Lincoln's body in the White House, witnessed, the report written and certified a true copy. I wrote the second autopsy report using the material they had and incorporating material gained from many years of research in medical history, chiefly that pertaining to Abraham Lincoln.

Respectfully,

J. WILLARD MONTGOMERY, M.D.

2323 Louisiana
El Paso, Texas
March 1, 1964

The Editor
The Journal of the American Medical Association
535 North Dearborn Street
Chicago, Illinois

Dear Doctor:

In the February 15th edition of THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION an editorial entitled LINCOLN'S INHERITANCE states:

> "In this issue of THE JOURNAL one of the Lincoln contributions (p 473) proposes that his unique physical appearance was due to the Marfan syndrome. Such a proposal is not new; a similar hypothesis has also been made by Dr. W. M. Gordon. What is of immediate interest, and a matter of disagreement among investigators, is the manner in which Lincoln inherited this syndrome. Was it inherited through his mother, Nancy Hanks, as Dr. Gordon suggests, or through his father, Thomas Lincoln, as Dr. Schwartz suggests? The answer to this question would provide Lincoln scholars with much sought-after information concerning the genealogy of Lincoln."

The answer to this question is available and is a very simple one: Mr. Lincoln did not inherit Marfan's syndrome from either parent because he did not have Marfan's syndrome.

Abraham Lincoln was, without the slightest doubt, the most healthy and all round able bodied president the United States ever had; in all probability there will never be another president possessing such a magnitude of physical and mental stamina. To prove that statement one needs hardly to search beyond his record in Washington during five years of Civil War. Not a day passed that there was not every reason for him to have a coronary thrombosis, cerebral hemorrhage or even commit suicide in despair; sanitary conditions at the White House were so chaotic at the time it is remarkable that he did not die from an infectious disease.

Judged by modern standards of athletes Abraham Lincoln would easily qualify as top-flight All-American in several field of sports and with proper training as a young man would have returned from the Olympics with several gold medals.

Intellectually he has never been even approached.

William Henry Herndon's Life of Lincoln was most controversial from its first printing. Historians have struggled nearly a century to rationalize some of his "facts" and "philosophy". One reads such things as "Herndon was certainly not a liar" and "Is Herndon reliable?", "Herndon may have been in error but he was not a liar", and this amazing lay interpretation: "He was at his best when describing Lincoln's physical appearance,...".

I am not qualified to evaluate this biography as a whole, however, his physical description of Lincoln from the medical standpoint rates him as the most accomplished liar of the nineteenth century. I do not pretend to comprehend what went on in that lawyer's cerebral cortex but his physical description of Lincoln is largely his own (Herndon's) physical make-up.

This part of the following seemed paragraph, probably the only part of the book Herndon actually wrote, is totally false:

> "Mr. Lincoln was ...thin, ...rawboned, thin through the breast to the back, and narrow across the shoulders; standing he leaned forward - was what may be called stoop shouldered, inclining to the consumptive build...His structure was loose and leathery; his body shrunk and shrivelled; he had dark skin, ...and looked woe-struck...his long shallow face was wrinkled and dry....".

Edward Curtis, M.D., Assistant Surgeon, Medical Department, United States Army, who helped perform the autopsy on Lincoln's body at the White House was fully qualified to make these observations:

> "I was simply astonished at the showing of the nude remains, where well rounded muscles built on strong bones told the powerful athlete. Now did I understand the deeds of prowess recorded of the President's early days.
>
> "A smooth clear skin fitting cleanly over well rounded muscles, sinewy and strong....".

Respectfully,

J. WILLARD MONTGOMERY, M.D.

Copy: Dr. R. Gerald McMurtry, Director
Lincoln National Life Foundation
Fort Wayne, Indiana

(3)

American Medical Association

535 NORTH DEARBORN STREET • CHICAGO, ILLINOIS 60610

AREA CODE 312
527 1500

March 5, 1964

J. Willard Montgomery, MD
2323 Louisiana
El Paso, Texas 79930

Dear Doctor Montgomery:

I am pleased to acknowledge the receipt of your letter on Marfan's Syndrome and Lincoln. We have had several comments on this subject since the Lincoln communications appeared several weeks ago.

I was not clear from the contents of your letter whether you wished this published in the Correspondence section of the Journal. If so, would you please return the enclosed card. When we think we have almost all the letters on this subject, a decision will be made regarding publication. If you have no objection to your letter in whole or part published following editorial revision, please return the postal card.

Sincerely yours,

John H. Talbott, MD

JHT:sjr

Enc: PC

Dr. J. Willard Montgomery, M. D.
2323 Louisiana
El Psao Texas.

8 Mar 64

Dear Doctor Mc Murtry:

Thank you for your letters and the information they contained.

As the enclosures indicate, the battle has been breached. I was amused that Dr Talbot himself wrote; we usually hear from a member of the staff.

Thanks for offering your cooperation; I do appreciate it. I was glad to learn Lincoln was about ~~250~~ 210# at twenty-one.

Please:

I-All the stories thatprove Lincoln was a supube athlete: the fight on the boat with the darkies, the fight with the bullie, Black war experiences, any stories about field labor, etc.

II-Give me proof of Nancy Hanks legitimacy.

III-Give me your opinion of Herndon. It appears to me he was a chronic alcholic and a psychopathic liar. What was David Donalds ipmression? I am going to see if I can locate his book tomorrow.

Kindest regards, and thanks,

J W Montgomery

Encl.: 3

~~Send~~
~~Lincoln Kane~~

Lincoln Kinsman
no 33

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44

Letters

Lincoln-Marfan Debate . . .

. . . Maternal Theory

To the Editor:—I was delighted to learn that my contention identifying Abraham Lincoln with the Marfan syndrome (*J Kentucky Med Assoc* 60:249 [March] 1962) has now been confirmed by Dr. Harold Schwartz (*JAMA* 187:473 [Feb 15] 1964). My address to the Kentucky State Medical Association on Sept 21, 1961, is the first reference to appear anywhere associating Lincoln with the Marfan syndrome. I also know that since 1961 many physicians have asked patients with the syndrome, "Are you by chance related to Abraham Lincoln?" and since there are thousands of living Americans who claim such kinship, I predict the appearance of many more reports similar to the recent JOURNAL communication.

I carefully read the Schwartz communication and aside from his case reports I could not find that he has added anything new to my earlier report.

The image of Nancy Hanks fits the Marfan syndrome. This cannot be said of Thomas Lincoln. Nancy was 5 feet 8 inches tall, weighed 130 lb, was stoop-shouldered, dolichocephalic, and had a sad facial expression. Thomas Lincoln was 5 feet 10 inches tall, weighed 190 lb, and was of stocky build. Dennis Hanks, Thomas' nephew, said that Thomas Lincoln was so closely knit that a finger could not be introduced between his ribs. Lincoln himself on many occasions said he inherited his mother's qualities.

After the birth of Nancy Hanks, her mother Lucy Hanks married a Henry Sparrow. This union produced eight children. Many of their descendants still reside in Kentucky, and I have examined a number of them. A striking resemblance to our martyred President was found. Many of them have the Marfan syndrome. It appears certain that Lucy Hanks transmitted the Marfan trait to her daughter Nancy and to her grandson Abraham Lincoln. The progeny sired by Henry Sparrow have physical characteristics identical to those of Lincoln. I'm aware that Lincoln had on his father's side relatives who were tall and lanky. However, not all tall, lanky individuals have the Marfan syndrome. My considered opinion from the evidence I have uncovered and documented concerning the Hanks-Sparrow-Lincoln line and their peculiar physical characteristics strongly suggests a maternal origin of Lincoln's morphologic appearance.

A. M. GORDON, MD
Louisville

. . . Paternal Theory

To the Editor:—The archives do not permit an unequivocal description of Nancy Hanks or Thomas Lincoln. Lincoln's mother has also been depicted as being of heavy build, squat, and 5 feet 5 inches tall. Dolichocephaly, kyphosis, and a sad visage are no more diagnostic of Marfan's than is mere excessive height and lankiness. Her appearance does not influence my data. Lincoln's father Thomas was just shy of 6 feet tall. Close-knit ribs may in some cases be a manifestation of the entity. Thomas' physique, however, is not relevant since Gordon has elsewhere accepted pyknic, stocky Robert Lincoln (the president's son) as having a *forme fruste* and carrying the gene, and my report compared the two as such. His report is more specific on Lincoln's legacy than his letter, and he quotes the president as saying that he inherited *through* his mother *the qualities of an unknown Virginian* who betrayed Lucy Hanks, his grandmother. This quote is historically unconfirmed. From this statement, however, originated Gordon's initial recommendation that the Marfan trait be sought in some unknown Virginia family whose forbears were neighbors of Lucy circa 1780.

Gordon's position is ambivalent. He accepts my cases as confirming the association between Marfan's syndrome and Lincoln yet denies the paternal linkage on which the association is based. My cases are not in any of the lines anticipated by him; they did not occur in the hypothetical Virginia line nor in the lineage of Hanks, Sparrow, or Nancy Hanks Lincoln. Further, Hank's genealogist, Adin Baber, states: "I . . . deny categorically that the Nancy Hanks who married Thomas Lincoln was a daughter of the Lucy Hanks who married Henry Sparrow. She was the daughter of Abraham and Sarah Harper Hanks."

It was just such difficulties that prompted Barton (1929) to state on Lincoln's heredity: "Theories based on physical resemblances are not very reliable and would better be omitted unless they go far enough to include accurate measurements of a sufficient number of generations to afford a basis for a scientific inference." My paper gave credit to Gordon's descriptive conjecture, but also offered the following as newly found evidence: skeletal indices for the president; his pectus excavatum; a genetic pedigree; three successive eye defects in his direct line; genetic evidence of his paternity; a specific Lincoln proband with x-ray confirmation; six case reports; three generations of the gene in a collateral line traced to a common ancestor, indicating a paternal Marfan origin. I believe that my report, documenting genetic and other evidence for my clinically derived independent postulation of Lincoln as a Marfan (1960), is the first to satisfy the criteria for a scientific inference. That the Sparrow or other cases may yet refute my data or suggest recessivity is conceivable; however, conventional genetic reports and pedigrees are indicated.

President Lincoln said: "We cannot escape history. . . ." It is to be hoped that through these studies his kin and the Marfan syndrome will also be served.

HAROLD SCHWARTZ, MD
Huntington Park, Calif

. . . Neither?

To the Editor.—An editorial entitled "Lincoln's Inheritance" (*JAMA* 187: 530 [Feb 15] 1964) asks the question: "Was [the syndrome] inherited through his mother, Nancy Hanks, as Dr. Gordon suggests, or through his father, Thomas Lincoln, as Dr. Schwartz suggests?"

The answer to this question is available and is a very simple one: Mr. Lincoln did not inherit Marfan's syndrome from either parent because he did not have Marfan's syndrome.

Abraham Lincoln was, without the slightest doubt, the healthiest and the most versatile, able-bodied president the United States has ever had; in all probability there will never be another president possessing such a magnitude of physical and mental stamina.

Judged by modern standards of

athletes. Abraham Lincoln would easily qualify as a topflight all-American in several fields of sports and with proper training as a young man would have returned from the Olympics with several gold medals.

I am not qualified to evaluate Herndon's *Life of Lincoln* as a whole; however, his physical description of Lincoln from the medical standpoint rates him as the most accomplished liar of the 19th century. I do not pretend to comprehend what went on in that lawyer's cerebral cortex, but his physical description of Lincoln is largely his own makeup.

The following passage, probably the only part of the book Herndon actually wrote, is totally false: "Mr. Lincoln was . . . thin, . . . rawboned, thin through the breast to the back, and narrow across the shoulders; standing he leaned forward—was what may be called stoop shouldered, inclining to the consumptive build . , . . His structure was loose and leathery; his body shrunk and shrivelled; he had dark skin, dark hair and looked woe-struck . . . his long shallow face was wrinkled and dry. . . ."

Edward Curtis, MD, Assistant Surgeon, Medical Department, US Army, who helped perform the autopsy on Lincoln's body at the White House, was fully qualified to make the following observations: "I was simply astonished at the showing of the nude remains, where well rounded muscles built on strong bones told the powerful athlete. Now did I understand the deeds of prowess recorded of the President's early days A smooth clear skin fitting cleanly over well rounded muscles, sinewy and strong. . . ."

J. Willard Montgomery, MD
El Paso, Tex

Letters were also received from Joan Titley and Benjamin B. Jackson, MD, Louisville, and from Paul E. Steiner, MD, Philadelphia.

Rumpelstiltskin

To the Editor:—Etymology is a fascinating subject but can go astray, as is shown by interpreting "Rumpelstiltskin" to mean "crinkly foreskin" (*JAMA* 188:309 [April 20] 1964).

Not by any stretch of imagination can such a meaning be attributed to the name. Whoever first gave it such a meaning expressed an opinion completely unclouded by the facts.

First of all, the meaning is completely foreign to Grimm's fairy tales. Secondly, the word is to be divided thusly: "Rumpel" (verb "rumpeln") not meaning to crinkle but to jolt or even rumble; "stilts" (low German for "Stelzen"), meaning stilts; and "kin" (low German for "chen"), simply the well-known diminutive. For example, a "Stiltsfoet" (low German for Stelzfuss) is a wooden foot or leg.

While it is not mentioned, I am sure that the dwarf was endowed with a Stiltsfoet, hence his ability to stamp through the floor.

G. L. Monroe, MD
New York

Ureteral Tumors

To the Editor:—Robards and his associates in the March 7, 1964, issue of The Journal (187:778) in writing on "Primary Tumors of the Ureter" made several statements with which I take issue:

1. I question calling papilloma a benign tumor. Recently, I did radical bladder surgery for a low ureteral metastasis from a previously "benign" mid-ureteral papilloma. In this case a nephrectomy and ureterectomy to the outer bladder wall had been performed. Careful analysis of the initial lesion had failed to reveal any submucosal infiltration, yet, metastasis did occur five years later.

2. The goblet sign is mentioned in the urographic diagnosis of ureteral carcinoma, but no mention is made of the "coiled-catheter" sign. This was originally described by Drs. Friedenberg, Sayegh, and myself in the *American Journal of Roentgenology, Radium Therapy and Nuclear Medicine* (86:707 [Oct] 1961) and later corroborated by others as being of value.

3. The statement that a functionless kidney with an obstruction in the ureter is the more usual roentgenologic finding is definitely debatable. We have reported several very large ureteral tumors which presented with normal ureters and kidneys *above* the lesion, so that intravenous pyelograms were reported as "normal." The claimed preponderance of "hydronephrosis above a nonspecific appearing stricture in the ureter" is also disputed by our findings. The kidney and ureter above the lesion appeared normal in half of our cases, and an important finding was dilatation of the ureter *below* the lesion. We stressed that in hematuria a complete ureterogram must be obtained in all cases, even if the ureter appeared normal on the intravenous pyelogram.

Because of the ureter's thin wall, the prognosis of any malignant ureteral lesion must be guarded. Statistics would certainly condemn the suggested technique of segmental resection of carcinomatous ureters. The conservative approach to truly benign ureteral tumors has been properly emphasized, but, for malignant lesions, radical surgery is the treatment of choice, except in extreme cases such as solitary kidney. The use of open biopsy and frozen section seems fraught with great danger.

I do not see how therapy of ureteral lesions and bladder lesions can be compared. If the authors' logic is to be followed to its conclusion, a papillomatous renal lesion would warrant removal of the involved kidney and ureter, the bladder, and the other ureter and kidney as well, since all have one connected urothelium.

We all practice renal salvage, but in any grade of ureteral carcinoma I feel the risk of jeopardizing the patient's life is too great if conservative surgery is performed.

Harry Bergman, MD
New York

Drs. Robards and Thompson stress individualized management:

To the Editor:—Certainly we can offer no solution to the problem of classifying urothelial papillomas. One cannot predict in which individuals other urothelial tumors will develop. In the case mentioned by Dr. Bergman, could not the vesical lesion which occurred five years after removal of a noninvasive ureteral papilloma have been a subsequent primary lesion?

Concerning the urographic findings in ureteral tumors, we wish to emphasize that the pyelographic findings are often not specific. To make a preoperative diagnosis of ureteral tumors one should have a high index of suspicion and should invariably outline the entire ureter at the time of retrograde urography. The variety of urographic findings pointed out by Dr. Bergman would

American Medical Association

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January 8, 1965

J. Willard Montgomery, MD
2323 Louisiana
El Paso, Texas

Dear Doctor Montgomery:

Since you are so interested in Marfan's Syndrome, I wonder if you would be willing to help us. The enclosed manuscript, "Dissecting Aortic Aneurysm in Marfan's Syndrome," by Dr. Simon has been submitted to The Journal for consideration. What is your opinion of it?

A couple of paragraphs on content, presentation, and general suitability of the manuscript for JAMA will be adequate.

Thank you very much.

Sincerely yours,

M. Therese Southgate, MD

MTS/jk

Enclosure: MS #2649

M E D I C A L C L U E S T O G E N E A L O G Y

b y

Harold Schwartz, M.D.

Southern California Genealogical Society Inc.
835 Locust Ave. Long Beach, California 90813

hands or finger characteristics, other outstanding bony features, color blindness, bleeding tendencies and the like should be recorded. Special talents and unique psychological and physiological attributes should be documented. Any kin with whom these features are held in common should be clearly identified. It is very important that inter-marriages of any degree be indicated. This may be of potential value to the family physician in diagnosing some otherwise obscure conditions and for giving advice where genetic counseling may in some instances be called for.

GENEALOGIC VALIDTY

To substantiate and correlate some of the material and concepts to be offered subsequently, several important points made by a former SOCIETY speaker Attorney Noel C. Stevenson, author of "Search and Research" should be emphasized.

Speaking in the subject of "Pitfalls in Genealogial Research" and considering what material should be accepted and what rejected, the attorney made the following points: Evidence is information and proof is the result of evidence. Direct evidence includes wills, birth certificates and the like. Indirect evidence is largely circumstantial, and by law, children born of a husband and wife living together are declared those of the husband, but marriage surveys have produced admissions by wifes of other fatherhood than the husband. (Several aources have maintained that the only proof of descent is to view the intact umbilical cord. Even this absolute though impractical approach is of no vlaue in the more difficult problem of the determination of specific paternity. H.S.)

Specifically, Stevenson considers that:

1. Practically everything in genealogy in legal terms is hearsay evidence.
2. Very few records can be proven without some suspicionof doubt.
3 3. In law, the rule is that the evidence must prove the truth beyond a reasonable doubt; and in genealogy it should be by a presponderance of evidence.
4. There is no absolute or conclusive proof in genealogy.

Amplifying these points, the attorney-genealogist emphasized that genealogists must be realistic and accept the truism that a pedigree cannot be proven with absolute certainty, but can be accepted by a preponderance of evidence.

ABRAHAM LINCOLN AS A GENEALOGIST

Before going on to the various applications of certain specific genetic traits in the linage of Lincoln, to establish an ancestral association, it is of interest to note that as Abraham Lincoln became a national figure, he revealed a keen interest in establishing his own paternal pedigree. He wrote several requests to various individuals in this regard and carefully answered similar inquiries put to him by kin likeness seeking a relationship. That he f ollow

In discussing ethics for the genealogist, she says, "Don't be like the lady who wrote the president requesting a bit of advice and his signature, so that she might have it for a keepsake. This is his reply.

"Whening asking stranges for a favor it is customary to send postage. There is your advice- and here is my signature." A. Lincoln

(Part III will take up the specific applications of genetics to determination and confirmation and will include evidence providing "proof" of an historic problem in paternity)

MEDICAL CLUES TO GENEALOGY
Harold Schwartz, M.D.
Part III

Having already discussed the essentials of genetics for genealogist as well as the validity of genealogic data, it now become appropriate to discuss the application of medical data to establishing ancestral relationships. In order to appreciate the concept being explored and the examples to be given, an understanding of the Marfan syndrome as it applies to Abraham Lincoln and his kin is indespensible. The applicable Lincoln-Marfan Pedigree Chart was previously presented and should be referred to in the following orientation material on the syndrome, which is an abbreviated and simplified version of the orginal report which appeared in the Journal of the Medical Assn, Feb. 15, 1964.

ABRAHAM LINCOLN AND THE MARFAN SYNDROME

In 1959 I established the diagnosis of the Marfan syndrome in a seven-year-old male whose pedigree was subsequently traced back nine generations to Mordecai Lincoln II. This ancestor, born in 1686, was also the great-great-grandfather of Abraham Lincoln. X-rays substanting the diagnosis in the patient as well as the genealogy establishment of the family relationship were published, along with further evidence of manifestations of the condition in four generations of the president's immediate paternal line. Objective oriteral in use at prsent for the diagnosis of the entity were retrospectively derived for the president, which the cumculative genealogic and genetic evidence, strongly suggest that Abraham Lincoln's unsual physical characteristics were those of a French pediatrician in 1896, some 85 years after the birth of President Lincoln, and more than 200 years after the birth of the common ancestor from whom the gene was transmitted to both the patient and the prsident.

Sometimes known as arachnodactyly (spider fingers) the Marfan syndrome is a dominantly transmitted hereditary disarrangement of connective tissue affeacting one of more of the skeletal, visual and cardiovascular systems. Excessive long-bone growth produces disproportionately elongated arms, legs, fingers, and toes. Long facial bones result in a high arched palate, and buckling of the ribs produces various unusual chest configurations. Laxness of ligaments, tendons and supporting tissues may result in a curvature of the spine, flat feet, loose-jointedness, herniae, and malpositioned ears. A striking lack of subcutaneous fat exaggerates the long lanky body structure. Eye difficulties include dislocated lenses, squint, severe near-sightedness, or marked far-sightedness. The heart valves may be involved and there is a tendency to develope bulging of the main blood vessel from the heart leading to tears or rupture with sudden death.

A search of the archives revealed that Abraham Lincoln was 6 feet 4 inches tall and wieghed 160-180 pounds. His arms and legs were disproportionately long as compared to his height; his fingers were long and bony and he had unusually large long feet. His head was thin and elongated with large ears set at a wide angle. In addition to being a 'sunken breast." Another contemporary depicted him as having 'spiderlike legs', the very same simile used many years later by Marfan in his original description of the condition. Lincoln was markedly far-sighted and was known to have a marked squint. When moving about, he was characteristically loose-jointed. When sitting down, he was no taller than other men; it was when he stood up that the excessive length of his lower extremeties brought him to his towering height.

The boy from whom the study took origin (Case 1) was tall, thin, double-jointed and had a high, arched palate and a characteristic chest configuration. A hernia had previously been repaired and his hands and feet were exceptionally long with gracile elongated fingers and toes. An X-ray of his hands disclosed

an abnormally high metacarpal index as determined by dividing the lenght of the hand bones by their width. Findings in the sister and the mother (Cases 2 and 3) were compatible with mild (forme fruste) manifestations of the entity.

The grandfather (Case 4), in addition to other compatible characteristics appears to have expired from a ruture of the main blood vessel from the heart. Two ancillary cases of correlative value are indicated on the chart (Cases 5 and 6). One is that of a death in a presumptive collateral kin under circumstances similar to that of the grandfather of the patient and in whom the ruptured blood vessel was confirmed at surgery.

The other was that of an 82 year old male whose facial and skeletal resemblance to Abraham Lincoln was a great that he had portrayed the president on stage and screen during his younger years. He was of the same recorded dimensions as the president and in addition has sunken chest that was documented radiographically.

Studying the close paternal linage of the president, it was found that Thomas Lincoln, tha father of the president, was blind in one eye and weak in the other. Robert Lincoln, the president's son, resembled his paternal grandfather, both being relatively stocky in build, even as Robert's only son resembled the president. However, like his father and jis grandfather, the president's son had an eye problem, establishing three generations with involvement of the visual system. The three other children of Lincoln resembled their father. Tad suffered a congenital misshapen palate and died at 18 with symptoms suggestive of heart failure. There is evidence that similar complications may have been present in Tad's two brothers and Abraham "Jack" Lincoln, the son of Robert and grandson of the president.

That Lincoln morphologically fulfilled modern objective skeletal criteria for the Marfan diagnosis was established by determining that his arm span was considerably greater than his height, and that the lower segment of his body (floor to pubis) was greater than his upper segment(pubis to vertex) as found from his sitting and standing heights and reference to certain pictures.

The most recent and most useful criteria is that of the metacarpal index already mentioned, and it was determined independently from the Smithsonian casts of the president's hands (made at the time of his nomination) that his index was also above normal. Analaysis of the president's spectacles revealed lenses of +6.75 diopters, indicating a degree of far-sightedness beyond that found in the acquired form.

Certain aspects of the well-known personality and mental characteristics of Lincoln were shown to occur in other cases of the syndrome, an area of investigation as yet little explored. Taking the total family constellation into consideration, there is evidence for involvement in each of the three major body systems usually concerned.

SPECIFIC EXAMPLES OF THE USES OF MEDICAL CLUES IN GENEALOGY

Example 1. It has been suggested that genetics can provide evidence that contributes to genealogical determination and confirmation, and the first example is that of the young patient with Marfan characteristics.

When he was reconized as being of a collateral line to Lincoln, a presumption was made that the president likewise had the same condition and a detailed morphologic and medical investigation of Abraham Lincoln was compatible with this idea. It was then established that Lincoln and the patient shared a common ancestor.

Since the Marfan condition is transmitted by a single ancestor as a dominant gene, the relationship found fits the genetic pattern expected and confirms the genealogical data that the common ancestor was Mordecai II and that it was he who passed the specific gene for this trait to both lines. The pedigree chart provides additional presumptive evidence that the gene expressed itself in many subsequent lines of Mordecai II and from each of his two wives which attests further to the genealogic and genetic correlations mentioned.

Example 2. Attorney Noel C. Stevenson, legal and genealogical authority whose remarks on the validity of genealogical data have already been referred to, has emphasized clearly the difficulty in proving paternity. Appropriately our second example involves just such a problem and one of great historical interest and fundamental to our main thesis.

Entire books and many articles have been writtem on the question of Abraham Lincoln's paternity. For years the detractors of the president emphasized the lack of a marraige bond attesting to the wedding of Thomas and Nancy Hanks Lincoln. However, around 1880, a knowledgeable student of Lincoln suggested a search for the document in Washington County, Kentucky. It was here that Thomas and Nancy had met prior to their marraige and residency in Hardin (LaRue) County, where the certificate had previously been unsuccessfully sought. As anticipated, the missing document was found, dispelling the idea that the president was born out of wedlock. However, it did not dispense with the rumors that Nancy Lincoln had been unfaithful and that Thomas was not the father of Abraham.

Only as recently as 1920, William E. Barton, Clergyman genealogist and Lincoln scholar published his findings on these matters in a book entitles "The Paternity of Lincoln." By the weight of negative evidence, he felt that it was unlikely that any of the various putative fathers suggested for the president, other than Thomas, could have been responsible for his birth.

It is now proposed to present here genetic data derived through the Marfan study, that appears to constitute a preponderance of positive evidence that Thomas was in fact the father of Abraham Lincoln. This material will be shown to go beyond that of the marraige bond, the Bible records, and any other ancillary testimony yet proposed on this subject.

Since the patient (Case 1) in my original report has been shown to have the Marfan gene; since all the collateral evidence produced is consistent with the postulation that President Lincoln likewise had these same Marfan characteristics; since these two individuals shared a common ancestor; we may conclude that by the genetic principles of Mendelian dominant inheritance that the gene was transmitted to the president through the paternal line that passes through Thomas Lincoln.

This establishes genetically as well as genealogically that Thomas was indeed the father of Abraham and relieves Nancy Lincoln of unfounded suspicions of infidelity. (See chart across)

If Mordecai II, the common ancestor, had only married once (Fig. A) we would not know if he or his wife had transmitted the gene to their children and we would be obliged to assume that the genealogical data established for the original patient (the propositus) was incorrect. (See complete pedigree chart given in Part I and Fig C opposite)

However, he did marry twice. By his first wife evolved the line leading through Thomas to the president, and by his second wife there was established the collateral line of Lincoln leading to the propositus (Fig. C)

MORDECAI LINCOLN II _ THE COMMON ANCESTOR

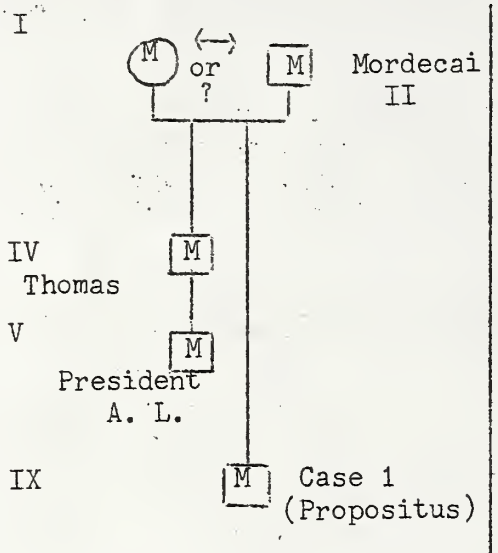

Fig. A.

If either parent of generation I had the M gene, it could appear in both lines of descent.

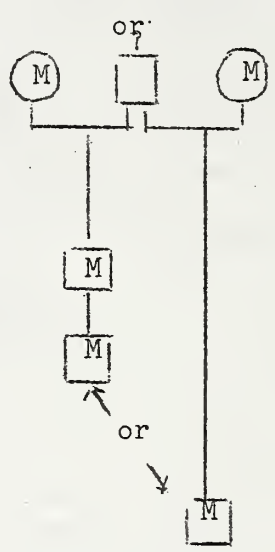

Fig. B.

If either wife had the M gene, it could only appear in her own line of descent but not in both.

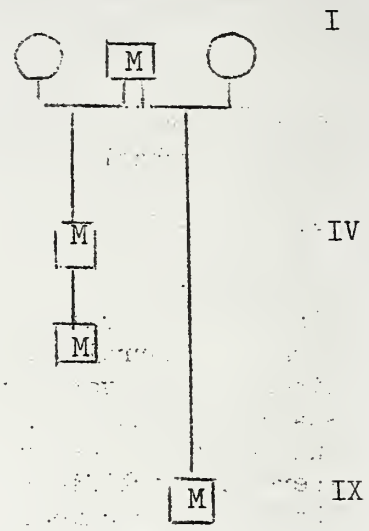

Fig. C.

Since the M gene appears in the lines of descent of both wives, only Mordecai could have had the gene and not either wife.

Key: M Marfan gene in male

Male, no Marfan gene

(M) Marfan gene in wife

Female, no Marfan gene

Example 2 - GENETIC EVIDENCE OF PATERNITY

If either of the two wives had carried the gene concerned, only a descendant of that particular marriage could have inherited the Marfan characteristics but not the other (Fig. B.). Since it has been shown that the syndrome was present in the lines of the children of both wives, the gene could only have been derived from Mordecai II by his successive marriages (Fig. C.).

In summary: Unless Thomas had been in fact the father, Abraham could not have inherited the gene from Mordecai Lincoln II. This single genetic fact supported by all the ancillary genealogical and clinical evidence given in the original paper. I believe constitutes a preponderance of evidence that Thomas was the father of Abraham Lincoln, the 16th President.

The application of medical and genetic material to genealogical investigation is perhaps nowhere better illustrated than by this particular instance among the several examples yet to be given.

MEDICAL CLUES TO GENEALOGY
Harold Schwartz, M.D.
Part IV

(In Part III two examples were given of the use of medical data in genealogy. Several additional examples follow.)

Eample 3 A young lady with two differently colored eyes was seen because of a presumed Lincoln kinship. While this feature has been reported in the Marfan syndrome, it is not of itself diagnostically significant, except as it may fit into the cumulative family pattern of the condition. After learning the characteristics of the syndrome, the individual recalled a cousin in another state whose features were highly suggestive of the type of skeletal elongation being investigated.

When examined some months later, this relative showed many of the bony features of the enity and her siblings demonstrated other correlating manifestations, such as herniae and the highly characteristics chest configuration. The latter feature was present in the boy from whom this study started, as well as in President Lincoln.

Beginning with the clue of the two differently colored eyes, the family pattern in this line is clinically consistent with the Marfan entity. The data appears to constitute a preponderance of evidence that corroborates the documentary genealogic material establishing this family's collateral Lincoln kinship. The Lincoln association likewise confirms the Marfan impression in this line. The family connection goes back to a descendant of the first marriage of Mordecai Lincoln II, the great-great-grandfather of the president, born in 1686.

Example 4. A significant instance of the appilicablility of the morphologic medical approach to establish a genealogic relationship, involves an elderly lady who by tradition was also a collateral relation of Lincoln. In this instance, Doane's previously quoted remarks that pictures may provide genealogical clues is borne out.

The exact lineage of the suspect Lincoln association was unknown to this particular family prior to this investigation. The woman was small, but with several minor skeletal features consistent with the family pattern of the syndrome. She had, in addition, a mole on the right cheek and similar skin appendages had been present on her mother. Her grandfather, as confirmed by family photographs, had a mole in the same spot as that which marked the right naso-labial fold opposite the corner of the mouth of President Lincoln.

The given name of this grandfather was such that with the several morphologic clues a genealogical connection was established to a colateral Lincoln kin, who had lost to the family tree when he was orphaned and had gone to live with other distant relations.

Example 5. The fifth situation illustrates another aspect of our concept, which is that the medical-morphologic data may also disprove a genealogic connection and establish another as the correct one, particularly if there has been any element of doubt in the pedigree.

In this instance, a near relative of the President's father was historically known to have married into a familyof pioneers somewhat closely connected with several succesive sites of the westward Lincoln migration. Individuals said to be of this lineage have been interviewed and the Marfan characteristics are reliably present in this particular family. However, at one critical point, their pedigree as it is known to them does not coincide with the historic record. The presently unresolved problem, then, is to determine which pedigree is to be accepted. The physical clues may yet establish the validity of one over the other, or else suggest a third possibility: that the connection was at some other point of relationship, or remotely that no association actualy does exist.

Example 6. A young female was noted to have dislocated lenses, after which the classis features of the Marfan syndromewere recognized. There was no known or suspect Lincoln kinship. The family name, however, is that of a cognate Lincoln line and the forebears of the young woman are known to have lived in the same county as this particular branch of collateral Lincolns. Attempts to relate these families to each other have been unsuccesful to date.

This particular connection is the most important of the unfinished aspects of this continuing investigation, since displaced lenses as seen in this individual, are the "hallmark" of the Marfan syndrome, and these have not been found in the Lincoln lineage.

If the genealogic connection is ultimately established, the immediate clue would have been the eye findings, which led to the diagnosis, which in turn resulted in the recognittion of the distaff lincoln name and the common county of residence.

Example 7. In another situation, the presence of a number of clinical findings in a young man suggested the Marfan syndrome, yet were not diagnostic thereof. The finding of other applicable features in kin of the suspect could permit the diagnosis to be established.

The surname of the individual was that of a family known to have come to this country before the American Revolution and to have settled in certian areas where early Lincoln kin have resided. The particular name is also one that in an accepted variant spelling is known to have married into President Lincoln's ancestral line.

If the kinship of the young man to the Lincoln lineage could be demonstrated, the diagnosis would be established cumulative family pattern. However, a clear cut relationship of this individual with the pedigree he presumes to be his own is not likely to be substantiated. While the connection could yet prove to be valid, this example illustrates that errors are possible if one would merely accept the morphologic characteristics and the coincidental association of names and places.

Comment: These seven examples from the Lincoln-Marfan association illustrate the application of the concept we are exploring: that medical data may contribute to genealogic investagation. While several of the items are from areas still under study, and the ultimate results remain unknown, the individual pionts being made would in any event remain unaltered. There are overlapping elements among some of the illustrations, yet each represents a different aspect of the example previously given.

Example

I Morphologic data confirmed a Lincoln pedigree and the converse contributed to the medical diagnosis

II Evidence of gene transmission resolved a problem of paternity.

III Eye Color indirectly led to the confirmation of a pedigree as well as a medical diagnosis when the lineage was corroborated.

IV Certain specific features and pictures established a pedigree for a traditional kinship.

V A collateral Lincoln pedigree of historic interest was opened to question through morphologic data in a related kinship.

Example

VI A very specific Marfan-type finding uncovered a surname held in common with a cognate Lincoln family, both families having shared the same area of earlier residence.

VII A pedigree and diagnosis could not be sustained desipte the coincidence of morphology, a distaff Lincoln name and similar areas of earlier residence .

This talk has presented a discussion of the mechanisms of genetic and heredity particularly as applicaible to genealogy. The related factor of genealogic validity has been taken up. It has explored by means of the unusual hereditary characteristics of the Marfan syndrome as it occurs in the Lincoln linage, a concept that suggests that medical data may contributes to genealogic validity and to establishing or corroborating a pedigree where documentary evidence may be inadequate. The converse has also been emphasized; that establishing the pedigree may permit a medical diagnosis by revealing the cumulative family pattern of hereditary manifestations.

The Lincoln migration started in England in 1637 when Samuel Lincoln sailed westward across the Atlantic and settled in the Massachusetts Bay Colony. From there his descendants first moved north into Maine, Vermont and New Hampshire and later south and west into New Jersey and Pennsylvania. The second phase of this steady expansion that concided with the great pioneer movement of this country's history, was into Virginia and Kentucky.

At this point subsequent generations moved into two directions: some further south and ultimately south-west, while the president's more immediate kin moved into Indiana and Illinois.

It has been generally supposed that with the death of the prsident, the Lincoln migration had ceased. The trail, however, can be shown to have extended ever westward into Nebraska, Utah and other western states until it reached the Pacific coast where in California, the collateral Lincoln kin, from whom this whole study took origin, was first seen in 1959.

The kin of Lincoln are located all over America. It is believed that genealogists familiar with the Lincoln and cognate fmaily pedigree will be able to contribute and participate in this continuing investigation and their cooperation is solicited. Criticisms and suggestions relating to the basic premise are also welcome.

Through a thorough medical-genetic and genealogic study of the Lincoln family and the syndrome which explains the president's unusual morphologic appearance and perhaps his unique mental attributes, it may well be that Abraham Lincoln, even in death, will make one more contribution to society as well as to medicine.

Harold Schwartz, M. D.
Huntington Park, California

(Editors Note: Dr. Schwartz has reported a heavy volume of inquiries and contributing data since this presentation before the Southern California Genealogical Society and its subsequent publication in "The Searcher" - into his busy professional activity.)

Mr. Harold L. Holzer
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Little Neck, N.Y. 11362

Dr. R. Gerald McMurtry
Lincoln National Life
Fort Wayne, Indianna

Dear Dr. McMurtry:

I have your interesting letter of March 4, in which you comment on what Dr. Harold Schwartz calls "The Lincoln-Marfan Debate." I hope you will allow me to bring up one or two more points on this issue.

It has been the belief of many Lincoln scholars with whom I've corresponded, including Lloyd Ostendorf, Adin Baber, and Stefan Lorant, that Lincoln's legendary physical prowess precluded the possibility of his having Marfan's Syndrome.

I disaggree with this idea. As I wrote to Mrs. Higgins, doctors in this city have assured me that a non-debilitating type of the syndrome is not uncommon. People who suffer the skeletal and visual symptom's of Marfan's, but who bear none of the cardiovascular ones, are called Form-Frusts.

Lincoln can easily be classified as a Form-Frust sufferer of Marfan's. The lists I presented, in which his obvious skeletal and visual oddities fit into the pattern, make this quite possible. Lincoln could still have maintained his renowned physique and held axes at arm's length, and still had Marfan's!

You wrote to me, "It seems rather remarkable that Lincoln could have been afflicted with a ~~xxx~~ condition that he did not know existed. . . ?" To me, it is not at all remarkable. I would suggest that Lincoln just thought he had large feet and hands, roving eye, hyperopia, slight double vision, etc. He would have ~~xxxxx~~ needed far more knowledge than the most brilliant doctors of his time to put all the pieces together and declare that he had Marfan's. Still, he could have had the disease.

Lloyd Ostendorf wrote that he dislikes "diagnosis from a distance." But in effect, isn't that what all historical analysis is?

I thank you for your interest in this matter, and hope to hear from you again regarding the probability of the Form-Frust idea.

Very Truly Yours,

Harold L. Holzer

HH/

cere, dedicated physician will take heart at such news, but he will usually be too busy to waste time and intelligence on the superficialities of climbing up the pecking order. He who can doesn't have to.

JOHN T. FLYNN, MD
New York

1. Hodge, R. W.; Siegel, P. M.; and Rossi, P. H.: Occupational Prestige in the United States, 1925-1963, *Amer J Sociol* 70:286-302 (Nov) 1964.

The 1963 study is a replication of a 1947 study and is based on 651 interviews. The replication was undertaken to determine whether appreciable shifts occurred between 1947 and 1963, so that effects of improvements in technical procedures could be sorted out from effects of historical changes. Scientific occupations increased in prestige, culturally oriented occupations fell, and artisans enjoyed a mild upward trend. The authors state that the bulk of their analysis is more concerned with characteristics of the distributions of these ratings than with the positions of particular occupations.—ED.

Restriction of Non-Prescription Drugs

To the Editor:—In your commentary on analgesic abuse and the kidney (*JAMA* 190:238 [Oct 19] 1964) you point to the incomplete and misleading nature of the warning now required by Food and Drug Administration regulation to appear on all packages of phenacetin-containing drugs for over-the-counter use. You also indicate the dangers of aspirin abuse and our lack of knowledge about acetaminophen hazard. You question the wisdom of any label warning directed against *abuse*, with special reference to ineffectiveness for people whose psychological makeup leads them to excesses. I agree with you that it is not possible by label warning alone to protect the public against hazards of self-medication with these or many other drugs. Unfortunately, such drugs are widely available. Recently the American Pharmaceutical Association has asked for establishment of a federal classification of non-prescription drugs which can be obtained only from pharmacists. Phenacetin-containing drugs would undoubtedly be so classified. A knowledgeable person, the pharmacist, would thus always be contacted before the drug is purchased. The pharmacist is professionally obligated at the time of sale to explain and extend label warnings and to work toward prevention of abuse of self-medication. I believe such restriction of availability of non-prescription drugs would compensate in large measure for the inadequacies of mere label warnings. I hope that the American Medical Association will support the American Pharmaceutical Association in its request for the creation of this new and needed classification of drugs.

WALTER SINGER, PHD
Assistant Professor of Pharmacy and Pharmaceutical Chemistry
San Francisco

Lincoln—Marfan Debate

To the Editor:—Inasmuch as my opponents in the Debate on the presence of the Marfan syndrome in President Lincoln (LETTERS, *JAMA* 189:164 [July 13] 1964) apparently do not wish to carry the matter further, I consider the fact clearly established now and forevermore that Abraham Lincoln did not have Marfan syndrome, and on Feb 12, 1965, I will regard the Debate concluded.

J. WILLARD MONTGOMERY, MD
El Paso, Tex

Hemothorax Complicating Translumbar Aortography

To the Editor:—The translumbar method of abdominal aortography is advocated by Leadbetter and Markland[1] for renal artery studies, and their compilation of experience shows relatively few serious complications. Perhaps the commonest complication is hemorrhage, which is usually self-limited, manifested as a retroperitoneal hematoma noted subsequently at surgery, and rarely of any magnitude. However, retroperitoneal dissection and exsanguination may develop. Presented here is a most unusual hemorrhagic complication, that of massive bleeding into the chest.

A 38-year-old Negro male was under investigation for arterial hypertension (180/110 Hg mm) of recent origin. The intravenous pyelogram, regitine test, and total and differential renal function studies were normal. Under epidural anesthesia, an 18-gauge needle was passed through the left flank and into the aorta on the first attempt. A 5 cc test dose of 50% sodium diatrizoate was injected and the films examined for needle placement, which was deemed satisfactory. The second injection of 20 cc of contrast medium was made with a hand injector, and the film exposed. The patient promptly complained of a severe, tearing, left chest pain and he exhibited anxiety. The needle was withdrawn and the examination terminated. Blood pressure remained stable at 170/110 Hg mm with a pulse of 80 to 94. No abnormal signs were present on examination, and the patient was observed. The tearing pain and dyspnea diminished during the next hour with no signs of blood loss, but several hours later signs of fluid in the left hemithorax developed. Closed needle thoracentesis of 800 ml of blood was performed that afternoon, and another 425 ml was removed the next day with prompt expansion of the lung and cessation of bleeding. No further fluid accumulated, and no transfusion was necessary. The patient's later course was uneventful. Examination of the aortogram showed extravasation of dye toward the left hemithorax, presumably followed by a leakage of blood. On the basis of the relatively slow accumulation of blood and the tearing pain in the chest, it was felt that dissection of the pleura by an enlarging hematoma, with later rupture into the pleural space, was the course of events.

Hemorrhage into the thorax after translumbar aortography must be a rare event. McAffe[2] in 1957, in his exhaustive paper on 12,832 translumbar aortograms, first reported this complication. Pleural pain, hypotension, and signs of hemodilution followed the procedure, and several thoracenteses over a period of days were sufficient for treatment. I could find mention of only one other intrathoracic hemorrhage, by Ford and Stamey[3] in 1961. The complication in their case, bleeding into the left side of the chest, was progressive and required open thoracotomy, evacuation of clot, and suture of the small aortic perforation.

Complications of the procedure include (1) visceral injuries such as spinal cord damage, renal failure, and gastrointestinal injuries which are presumably related to the effect on the microvasculature with sludging, agglutination, and vasospasm; (2) atopic reactions of varying degrees, extending from urticaria to anaphylactoid reactions; (3) injuries due to misplacement

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347P CST FEB 11 65 DB267 LC103
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DOCTOR J WILLARD MONTGOMERY, MD
[redacted] EL PASO TEX 2323 LOUISIANA
FAILURE TO DEBATE DOES NOT VALIDATE YOUR NEGATION PROCLAMATIONS
I CONSIDER THE ISSUE ENGAGED AS OF FEBRUARY 11 1965
HAROLD SCHWARTZ MD 7507 SEVILLE HUNTINGTON PARK CALIF
(37).

Lincoln-Marfan Debate ∞

More than one year ago, in the Feb 15, 1964, issue of THE JOURNAL, *Dr. Harold Schwartz suggested that Abraham Lincoln inherited the Marfan syndrome through his father. He was opposed by Dr. A. M. Gordon, who believed the inheritanee to be maternal. Letters in the form of a debate were published in the July 13, 1964, issue. Appended to the letters was a third proposal, by Dr. J. Willard Montgomery, that Lineoln did not have Marfan syndrome and that neither theory was correet. There the debate stood, no further counter-proposals being entered, and in the Feb 8, 1965, issue Dr. Montgomery claimed default, bringing the following response:*

To the Editor:—The following telegram was sent on Feb 11, immediately upon receipt of THE JOURNAL, in response to a letter and manifesto which appeared considering and deciding for science and history that by default and as of Feb 12, 1965 (I presume at the stroke of midnight), Abraham Lincoln did not have the Marfan syndrome (LETTERS, *JAMA* 191:505 [Feb 8] 1965):

DR J W MONTGOMERY EL PASO TEX FAILURE TO DEBATE DOES NOT VALIDATE YOUR REFUTATION PROCLAMATION. I CONSIDER THE ISSUE ENGAGED AS OF FEBRUARY 11, 1965.

There may be an analogy in the scheduled duel between Lincoln and Shield in 1842 which was precipitated by a letter to the editor, only to be averted by a bit of humor and an appreciation of differing opinions.[1] With the same intent, I submit that the Lincoln-Marfan matter will not be settled by vacuous debate, but by the weight of cumulative scientific evidence.[2]

HAROLD SCHWARTZ, MD
Huntington Park, Calif

1. Beveridge, A.J.: *Abraham Lincoln 1809-1858*. New York: Houghton Mifflin Co., 1928, pp 335-354.
2. Strange Case of Abraham Lincoln, editorial, *Brit Med J* 1:858 (April 4) 1964.

Data Requested on History of Psychiatry in Canada

To the Editor:—A lively interest has developed across Canada in the history of medicine in general and in the history of Canadian psychiatry in particular. The Canadian Psychiatric Association has established a Committee on the History of Psychiatry which is now attempting to prepare a comprehensive bibliography.

We would like to request the cooperation of your readers in helping us to obtain information about books and articles, including unpublished theses, etc, dealing with this topic. Detailed information or even vague references would be most welcome. They could be sent to *Mr. Cyril Greenland, Associate (Archivist), Dept of Psychiatry, University of Toronto, 2 Surrey Pl, Toronto 5.*

We would also like to receive superfluous or unwanted books of historical interest in this field, reports, pamphlets, letters, or memorabilia which might throw light on the early days of psychiatry in Canada. In cases where documents of unusual interest cannot be released, we would, in any event, like to correspond about them with a view to arranging for reproduction.

J. D. GRIFFIN, MD
Chairman
Committee on History of Psychiatry
Canadian Psychiatric Association
Toronto

An Oddness of Id

To the Editor:—The narration "An Oddness of Id" (*JAMA* 191, Feb 8, 1965, adv p 238) contained an interesting and provocative description of Doctor Odd-Id. It also seemed to demonstrate the pitfalls of the odd-id syndrome since the author proceeded to diagnose the nearly 1,200 medical doctors without examining them carefully himself.

ROBERT G. AUSTIN, MD
Longview, Wash

Dr. Austin's letter was referred to an observer who has kept wateh over medieine and current events. He offers the following observations:

Dr. Richard P. Huemer, author of the narration, expressed dismay that 1,189 psychiatrists, responding to a *Fact* magazine poll, made psychiatric diagnoses, by mail, of a political candidate running for high office. Dr. Huemer recognized a defect in scientific method and applied an appropriate term to it. I compliment Dr. Austin (1) for recognizing that Dr. Huemer himself resorted to mail-order diagnosis, and (2) for pointing out that two extremisms do not make a right. Extremism is certainly not in the mainstream of American behavior, but when the mainstream takes to railroading, a fair majority of Mainstream's citizens are inclined to get off Mainstream's track. Where, then, is the mainstream? Where, then, is extremism?—OBSERVER.

Value of Skin Testing for Penicillin Allergy

To the Editor:—In recent months a number of studies have been published on the value of skin testing for sensitivity to penicillin, especially with the use of various antigenic determinants. Several papers in THE JOURNAL (Rytel et al, 186: 894-898 [Dec 7] 1963, and Budd et al, 190:203-205 [Oct 19] 1964) concur with the report of Parker and Thiel (abstracted, *Ann Intern Med* 58:763, 1963) that penicilloyl-polylysine provides a reliable preparation for the skin testing of patients with a positive history of immediate type sensitivity to penicillin. On the other hand, Siegel and Levine (*J Allerg* 35:488-498 [Nov-Dec] 1964) provided evidence that such a derivative gives negative skin-test reactions in a number of patients clinically sensitive, while potassium penicillin G produces marked immediate wheal. Finke et al (*Amer J Med* 38:71-82 [Jan] 1965) stated, "It may be that skin testing with penicillin G is a more specific index of anaphylactic sensitivity than tests with penicilloyl-polylysine." Van Arsdel (*JAMA* 191:238-239 [Jan 18] 1965) summarizes this complex and controversial problem. With such unreliability of skin testing, as well as with the danger of systemic reaction from the mere test itself, it would be wise to use the patient's history as the guide, no matter how remote the causal relationship may have been. A positive history alone should make the physician wary of choosing this antibiotic.

Several recent experiences in our clinic exemplify the hazard of testing. Four patients with histories of immediate systemic reactions to penicillin gave no reaction on skin testing with penicilloyl-polylysine or with penicillanic acid. Yet all of them gave strong reactions on mere scratch testing with potassium penicillin G. One patient had a moderate systemic reaction to such a test; he was relieved in an hour with the use of epinephrine and antihistaminics. Another patient so scratch tested developed profound anaphylactic shock within a few minutes and was revived only with heroic measures. Had penicillin been administered on the basis of negative skin-test reactions to the other derivatives, death would unquestionably have resulted. The sera of these patients have since been studied on passively sensitized volunteers and on the Macacus irus monkey (L. L. Layton et al, *Int Arch Allerg* 22:87-94, 1963), with safety, and have confirmed the discrepancy in direct skin-test reactions.

TELEPHONE
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BOARD INTERNAL MEDICINE

J. J. ROBBINS, M. D.
INTERNAL MEDICINE
1333 A STREET
HAYWARD, CALIFORNIA 94541

John H. Talbott, M.D., Editor
Journal of the AMA
535 North Dearborn Street
Chicago, Illinois 60610

April 19, 1965

Dear Doctor Talbott:

As with many other problems of medicine as well as other fields of human endeavor, the answer to the controversary as to whether Abraham Lincoln suffered from Marfan's Syndrome can be resolved by application of the basic principles of semantics and logic.

According to Dorland's Dictionary a syndrome is defined as a morbid state or disease characterized by a certain set of symptoms. One would not therefore ascribe the term syndrome to unusual height which is not disabling anymore than to possession of a Roman Hittite nose.

Marfan's syndrome according to standard medical texts is a heritable disorder characterized by weakening and degeneration of connective tissue with particular predilection for the cardiovascular and musculo-skeletal systems.

It is a fact, well established by one of the articles which appeared in the AMA Journal that President Lincoln was a man of prodigious physical prowess and endurance. In fact it was related that at the age of 55, three months before his death, he performed a feat of tree-cutting which astounded the soldiers of the Union Army.

Abraham Lincoln therefore suffered from no disease of the cardiovascular or musculo-skeletal systems.

Ergo, Lincoln did not suffer from Marfan's Disorder. Quod Est Demonstrandum.

Sincerely yours,

J.J. Robbins, M.D.

JJR:mam

THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION

11 June 1965

Dear Doctor Southgate:

I was amused but not in the least surprised to learn in AMA NEWS that medical education began in 1765 in America at the University of Philadelphia and to learn later in the JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION that all this was a mistake; the first medical school was founded in 1520 in Mexico City and that this was all wrong too; that the really first medical school was at the University of San Marcus in Lima, Peru and the date of the founding 1551. Lastly, it was back again in Mexico City and the year this time 1547.

Had it not been for the LINCOLN-MARFAN DEBATE in the JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION the American and British medical professions would have been scooped by lay magazine writers with the first accurate physical evaluation of Abraham Lincoln based on elementary medical evidence a hundred years old! (See AMERICAN HERITAGE April 1965).

The first medical school and hospital in the Western Hemisphere, this side of the Stone Age, was founded at Tenochtitlan (Mexico City) in the year of our Lord 1536. Colegio de Santa Cruz was the name of the school and hospital complex and it was the first colegio in La Real Universidad de Mexico, and therefore, marked its founding. de Tlaltelolco

The Dean of the school, Fr. Bernadino (Ribeira) de Sahagun (? - 1590) describes in detail all about it in his voluminous writings in medieval Spanish.

It had all the problems of similar modern day institutions and many, many more. The school survived nine hectic years: in 1545 the doors were closed and the buildings deserted. Hueyzahuatl caused the closing; pronounced by the Aztec Hoo..oo..za..waitle.

Hueyzahuatl was every bit as fearsome a disease as it sounds.

For publication in the JAMA

Sincerely,

J. WILLARD MONTGOMERY, M.D.

PS: Hueyzahuatl is Aztec for Smallpox; suggest you tear this off and let the rest look it up.

July 7, 1965

Dr. J. Willard Montgomery
2323 Louisiana
El Paso, Texas 79930

Dear Dr. Montgomery:

I was pleased today to receive your envelope containing copies of your correspondence along with copies of your articles which have appeared in the Journal of the American Medical Association relative to Lincoln's so-called disability known as Marfan's syndrome.

I read your letter with a great deal of interest and I am pleased that at last a doctor has stepped in to the argument who believes that Lincoln did not suffer from such a disease. Your statement certainly did stir up a hornet's nest and immediately put the other side on the defensive. Thank you very much for sending me this information.

I also appreciate receiving the miscellaneous literature pertaining to the Lincoln statue in Juarez, Mexico. I am quite eager to see this piece of sculpture.

We will be happy to place Baxter Polk of the library at Texas Western College on our Lincoln Lore mailing list. Perhaps we can send him the back numbers for the year 1965.

Wishing you success and thanking you for your letter, I remain

Yours sincerely,

R. Gerald McMurtry

RGM/hcs

Letters

Controlled Pressure Diskography

To the Editor:—Since Cloward's article[1] on cervical diskography appeared, this procedure seems to have been the object of great controversy. When Holt's evaluation of the procedure[2] was published, many reputable surgeons developed serious doubts as to the validity of this test. Later published opinions of diskography[3] seem to be gradually becoming more favorable.

The author recently listened to a discussion of diskography in which one physician stated that all diskograms could be made positive if enough pressure were applied, just as retrograde pyelography could be similarly distorted. It seemed to me that a simple application of scientific method to this, namely, trying to control the variables, would establish the true value of the test.

Method: Several neurosurgeons were tested while using a syringe attached to a pressure gauge. The variation between the maximum injection pressure used by different neurosurgeons was from 19 to 52 pounds per square inch (psi). Each individual surgeon, however, was fairly consistent in his personal maximum pressure, varying less than 5 psi pressure. By taking five maximal pressure readings from each surgeon and averaging these, the figure of 30 psi was obtained. It was arbitrarily decided to use this as the maximum allowable pressure during diskography.

Using a sterilizable commercial pressure gauge with a range from 0 to 100 psi, we devised a suitable adapter so that pressure could be recorded during diskograpy. The apparatus, illustrated recording the pressure during diskography (Figure), employs a new type of plastic tubing with a four-way stopcock. The apparatus pictured is bulky, but the manufacturer has been requested to develop a more compact unit.

Since the author routinely injects three disk spaces on each patient, many disks have now been tested at up to 30 psi with almost no pain production and no leakage of contrast material from the center of the disk space. It is my feeling that a normal intervertebral disk can easily withstand an internal pressure of 30 psi.

At present, we are beginning a two-year study of diskography with pressure control. It is hoped that the results of this study will clear up some of the confusion which seems to surround diskography.

The main drawback to this method is that the amount of contrast

material injected into a disk cannot be as accurately measured. During injection with this system, leakage from a disk can be felt by the fingertip on the syringe as a sudden "give" in resistance. Care is exercised that no more than 0.5 cc of contrast material is injected beyond this point.

A. Stephen Genest
San Jose, Calif

1. Cloward, R.B.: Cervical Diskography, *Ann Surg* **150**:1052-1064 (Dec) 1959.
2. Holt, E.P., Jr.: Fallacy of Cervical Discography: Report of 50 Cases in Normal Subjects, *JAMA* **188**:799-801 (June 1) 1964.
3. Sugar, O.: "Editorial Comment on Cervical Discography," in *Year Book of Neurology, Psychiatry, and Neurosurgery*, Chicago: Year Book Medical Publishers, Inc., 1965, pp 607-608.

Abraham Lincoln and The Marfan Syndrome

To the Editor:—This week—one short year ago—The Journal brought forth from a physician a letter conceived in admiration but dedicated to the proposition that Abraham Lincoln did not have the Marfan syndrome (**191**:505, 1965). Now we are engaged in a scientific debate, testing whether that notion —or any notion affirmatively dedicated—can long endure (**192**:64, 1965).

In rebuttal, somewhat overdue, I offer new evidence for the diagnosis, noted in a recent volume by Kunhardt on the death of the 16th president.[1] A remarkable anatomical sketch made shortly after the assassination shows the victim's bare feet as they protruded from the end of the bed upon which he lay dying. On another page, the legend to a photograph of the size-14 shoes of the president reads, "Lincoln's feet were long and narrow with extraordinarily exaggerated big toes . . ." These remarks describe precisely the details in the sketch. The diagnostic significance of this can be appreciated by comparison to McKusick's text which illustrates the feet of a Marfan subject, the legend stating, "Extraordinary length of the great toes is well demonstrated."[2] The proband of the original Lincoln-Marfan study was described in 1964 as having "notably long great toes" illustrated by x-rays of his feet.[3] Striking excess length of the more distal bones of the extremities is a characteristic feature of arachnodactyly. Marfan's original description (1896) was confined to the skeletal system. The long great toes, the pectus involvement, and the other bone changes previously reported for the president thus become virtually pathognomonic, even without the ocular, genetic, and cardiovascular associations already established.

It is opportune to refute the negative evidence that the great strength of the rail-splitter excludes the Marfan diagnosis. While muscular hypotonia has been reported, this is an inconstant and infrequent finding and one I have yet to see clinically. When present, it is probably secondary to abnormalities of bones, joints, and ligaments. At times there is an appearance of muscle wasting coincident to the lack of subcutaneous fat and skeletal elongation. Primary muscle involvement in the condition has not been documented. In a recent clinical survey at the Marfan Clinic of the Rancho Los Amigos Hospital independent objective testing of six consecutive cases failed to reveal evidence of muscle weakness. The president's physical prowess is neither myth nor mystery; historian Benjamin Thomas observed that Lincoln's long body and long arms could exert tremendous leverage. The mechanical advantage of Lincoln's unusual skeletal structure constitutes positive evidence for the diagnosis rather than otherwise.

The arm span of the Chief Executive was as much as 6 inches greater than his height; his two episodes of diplopia while in the White House are consistent with

the Marfan entity; heterochromia iridis, pectus carinatum, and other consistent manifestations are present in one of several newly investigated collateral lines of Lincoln. Reported, but unconfirmed as yet in this same family, are several generations of male color-blindness which may be of value in linkage studies. The gene concerned may yet be followed several generations further back than previously, into Norwich, England, before the year 1600.

The total impact of Lincoln's unusual physical composition upon his personality and career has not yet been explored, and despite a large literature a definitive interpretation and biography of Lincoln the man remains to be written. Whosoever attempts this should be mindful that in one man's opinion, "Biographies as generally written are not only misleading, but false. The author makes a wonderful hero of his subject. He magnifies his perfections, if he has any, and suppresses his imperfections." These remarks attributed to Abraham Lincoln conclude with the observation, "History is not history unless it is the truth."

HAROLD SCHWARTZ, MD
Huntington Park, Calif

1. Kunhardt, D., and Kunhardt, P.: Twenty Days, New York: Harper & Row, Publishers, Inc., 1965, pp 46 and 291.
2. McKusick, V.: Heritable Disorders of Connective Tissue, ed 2, St. Louis: C.V. Mosby Co., 1960, p 55, Fig 9C.
3. Schwartz, H.: Abraham Lincoln and the Marfan Syndrome, *JAMA* **187**:473 (Feb 15) 1964.

Crawford W. Long, and Henry Hickman

To the Editor:—I was very much interested to read two recent editorials in THE JOURNAL (**194**:1006, 1008, 1965), namely, "Credit for Discovery" and "Crawford W. Long, Discoverer of Ether for Anesthesia." I believe that, as indicated in the editorial, it is most important to stress that a discovery frequently stems from several sources. Despite the very human tendency to claim priority, it is more important that society in general recognize all who have contributed to a given thought or discovery. If the discovery of general anesthesia is a milestone in medical progress, as stated by Dr. Harry Beckman, then it follows that this contribution is sufficiently large that the credit might well be shared by Long, Wells, Morton, Jackson, and others. Additionally, an English physician, Henry Hickman, demonstrated general anesthesia in animals in 1825 using carbon dioxide. Although this is a somewhat unusual method of anesthesia, there is no doubt that his work was valid; however, the reception of this information by the scientific and medical profession was very limited. In respect to Crawford W. Long, I would point out a further recognition of his discovery with the establishment of the Crawford W. Long Museum of Anesthesia in Jefferson, Ga, where he practiced. Although this museum is not well-known, it should not be forgotten in view of the fact that the discovery of anesthesia was one of the most notable American contributions to medicine during this period.

JOHN E. STEINHAUS, MD
Atlanta

Candida albicans and the Normal Skin

To the Editor:—Two recent articles by F.A.J. Kingery, MD, scored the general diagnostic ineptitude in recognizing possible cutaneous fungal infections. The author presented worthwhile clinical pictures and corrected important pitfalls in diagnosis. However, as he expanded his thesis, he made remarks that are as seriously in error as those he tried to correct.

In "Don't Forget about Candidiasis" (**191**:851, 1965), the statement is made: "Since *C albicans* has commonly been recovered from skin and mucous surfaces in the absence of disease, the clinician must be familiar with the pathological patterns which may result from the presence of this organism. Identification of pseudohyphae in potassium hydroxide mount and culture on Sabouraud's agar provide positive identification."

Though frequently present on normal mucous surfaces, *C albicans* is not commonly found on normal skin.[1,2] This faulty concept is being thoughtlessly propagated in the literature, despite ample recorded evidence to the contrary. It may lead to dismissal of the organism as a "harmless contaminant" when it is isolated from skin lesions or from various body fluids, with potentially disastrous consequences for the patient. This is particularly significant in view of the marked increase of systemic candidiasis and the diagnostic difficulties involved in its recognition. The presence of true and pseudohyphae in potassium hydroxide mounts is pathognomonic. Cultural identification of the species requires procedures beyond isolation on Sabouraud's agar on which most *Candida* species as well as baker's yeast present the same white waxy appearance.

"The Myth of Otomycosis" (**191**:129, 1965) was given a satisfactory rebuttal on clinical grounds by E. H. Jones, MD (**193**:404, 1965). The frequency of primary fungal infection of the external auditory canal may have been exaggerated, but the infection is not a myth.

The basic error implicit in both of Dr. Kingery's communications arose from faulty interpretation of the significance of fungi on the skin. *Aspergillus niger* is a ubiquitous airborne saprophyte which is readily cultured as a contaminant from normal and diseased skin surfaces. Aspergilli are "opportunistic" pathogens whose etiologic role in human disease has to be most carefully verified by repeated demonstration in direct smears, biopsies, and cultures.[3]

Although one may reasonably suspect fungal infections on clinical grounds alone, good practice demands the minimum of a direct smear mounted in 10% potassium hydroxide to confirm the clinical impression.

PHILIP J. KOZINN, MD
CLAIRE L. TASCHDJIAN, MS
Brooklyn, NY

1. Winner, H.I., and Hurley, R.: *Candida albicans*, Boston: Little, Brown & Co, 1964
2. Schirren, C.: *Hefepilze auf gesunder Haut, in Hefepilze als Krankheitserreger bei Mensch und Tier*, Göttingen: Springer-Verlag, 1963
3. Hildick-Smith, G.; Blank, H.; and Sarkany, I.: Fungus Diseases and Their Treatment, Boston: Little, Brown & Co, 1964

Dial M for Murder?

To the Editor:—I would like to take issue with Dr. Liddle's cavalier handling of the dial-a-service facilities provided by various benevolent community-minded agencies. Where does Dr. Liddle get the idea that the proliferation of these services reflects a deep-seated emotional insecurity of our citizenry? The conclusion is totally unwarranted.

Our prosperous progressive town boasts of all the dial-a-facilities of Bloomington with a few extras thrown in. We enjoy a dial-a-date (formerly dial-a-dame, since discontinued because of run-ins with the law) service—fully computerized. We have dial-a-dialogue and dial-a-debate services which provide us with instant conversation amicable or argumentative de-

Lincoln Studies Trace Marfan Syndrome to 1600, Show Pattern of Intelligence

Abraham Lincoln not only inherited a tall, lanky body with unusually long arms, but a seven-year genealogy study indicates that he may have inherited a superior intelligence as well.

Dr. Harold Schwartz, an instructor at the University of Southern California School of Medicine, says his lengthy investigation has produced "extensive evidence" that Lincoln had the Marfan syndrome and so did his paternal ancestors—all the way back to 1600.

Emerging from Dr. Schwartz's studies now, however, is a consistent pattern of superior intelligence in the Lincoln line, a characteristic he says has been documented in case studies "time after time." He said among Lincoln's "eminent ancestors" were several New England governors and six generations of Harvard graduates.

Individuals with the Marfan syndrome, first reported by a French physician in 1896, are often troubled by eye difficulties, as Lincoln was, or in some cases, heart disease and orthopedic problems. In recent weeks, Dr. Schwartz obtained an artist's sketch which showed that Lincoln's big toes compared "precisely" with Marfan toes.

Dr. Schwartz said other Marfan traits in Lincoln were facial and hand characteristics and an arm span which measured from 3- to 6-in. longer than his body from head to toe. Three of his four sons resembled Lincoln and all three died at early ages of what may have been heart disease.

The doctor expects the Lincoln investigation to be more than historically significant. In addition to providing more information on the Marfan syndrome, which he believes is more common than suspected, he is hopeful that the study will help to answer many questions about the behavior of genes.

Dr. Schwartz said the clinical value lies in early diagnosis of the Marfan syndrome and treatment, or possibly prevention, of the eye, heart, and orthopedic difficulties associated with the syndrome.

New Member for WHO

The Maldives, a set of scattered islets in the Indian Ocean, is the newest member of the World Health Organization. The 550-mile string of 2,200 atolls, which recently gave up its role as a British protectorate, became the 122nd full member of WHO.

Newsfront continued on page 43

DR. ABRAHAM M. GORDON
LOUISVILLE, KENTUCKY

May 3, 1966

Dr. R. Gerald McMurtry, Editor
Lincoln Lore
The Lincoln National Life Foundation
Fort Wayne, Indiana

Dear Doctor McMurtry:

I am taking the liberty of sending you a zerox copy of page 135, the third edition, of Dr. Victor A McKusick's book Heritable Disorders of Connective Tissue. I believe you will find this of some interest.

Very truly yours,

Abraham M. Gordon, M.D.
305 West Broadway

AMG:cdd

Enclosure

comparable basis, is said to occur in horses and occurs in epidemic fashion in turkeys,[240] where dietary factors may be responsible.

The treponemal immobilization test for syphilis has created a clinical problem in connection with patients with dilatation of the ascending aorta and aortic regurgitation, who often in the past would have been considered to be syphilitic in spite of negative serologic tests for syphilis of the conventional type. In coming years, cystic medial necrosis of the aorta, previously the exclusive property of the pathologists, will be discussed much more frequently in the clinical literature.

Gordon[135] and Schwartz[325] have suggested that Abraham Lincoln had the Marfan syndrome. Both based the impression in part on Lincoln's long extremities and statements of his contemporaries that he was unusually loose-jointed. Nathaniel Hawthorne described him as a "tall, loose-jointed figure." The Washington correspondent of the London *Times* described Lincoln as a "tall, lank, lean man, considerably over six feet in height, with stooping shoulders, long pendulous arms terminating in hands of extraordinary dimensions, which, however, were far exceeded in proportion by his feet." Lincoln's mother, Nancy Hanks, who in the opinion of Gordon had the Marfan syndrome, was of unknown paternity. (Her death is more usually attributed to "milk sickness," hypoglycemia from milk from cows that have fed on white snake root.) On the other hand, Schwartz[325] believes he has evidence that Lincoln inherited the Marfan gene from his father; he has a patient named Lincoln, with seemingly typical Marfan syndrome, who is a descendant of Lincoln's grandfather.

Gordon suggested that Thomas Lincoln, who resembled his father closely and died at 18 years of age of "dropsy of the chest," had the Marfan syndrome with cardiovascular complications.

Because of the obvious general interest, the conclusions of Gordon and Schwartz found their way into the lay press. The public reaction has had interesting features. For example, in a letter-to-the-editor (*Newsweek,* June 11, 1962), one reader insisted that Lincoln could not have suffered from such a "loathsome disease." The statement reflects an unfortunate and uninformed attitude toward hereditary disease, which is no more (or less) loathsome than poliomyelitis, sarcoma, myocardial infarction, alcoholism, and cerebral arteriosclerosis, with which other Presidents have been afflicted.

SUMMARY

The cardinal manifestations of the Marfan syndrome are skeletal, ocular, and aortic. Dolichostenomelia (long, thin extremities) and redundant ligaments and joint capsules characterize the skeletal changes. Ectopia lentis is the hallmark of the disorder in the eye. In the aorta, predominantly the ascending aorta, diffuse dilatation and/or dissection occur.

The only histopathologic changes described to date are those in the aorta, where degeneration of the elastic lamellae appears to be primary in the pathogenetic chain. Whether the basic defect resides in the elastic fiber or, as seems somewhat more likely, in collagen is unknown.

The pedigrees are consistent with inheritance of this trait as a simple Mendelian dominant with a relatively high grade of penetrance.

Although certainly there are some persons having true cases of the Marfan syndrome without ectopia lentis and without other less equivocally affected mem-

Gordon

May 10, 1966

Dr. Abraham M. Gordon
305 West Broadway
Louisville, Kentucky

Dear Dr. Gordon:

Many thanks for your letter and Xerox copy of Page 135 relative to "Heritable Disorders of Connective Tissue". I have read the article by Dr. Victor A. McKusick with interest and it will be placed in our files.

Yours sincerely,

R. Gerald McMurtry

RGM:hcs

Chicago Tribune
Chicago, Ill.
Oct. 4, 1972

How to Keep Well

Lincoln's Days Before Shooting

By T. R. Van Dellen, M. D.

Many investigators have speculated on Abraham Lincoln's health during his second term in office. Dr. Harold Schwartz of Lynwood, Cal., believes that the President was a sick man and probably would have lived no more than six months had he not been assassinated. He recently found support for his hypothesis that Lincoln had "leakage of the aortic valve" and was in a state of heart failure. Lincoln's lack of well-being usually is attributed to emotional stresses associated with the burdens of office and a tragic war.

Dr. Schwartz was the first to suggest that our 16th President had Marfan's syndrome and that this disorder was responsible for his unusual physical appearance. It is an hereditary condition due to mismatching of the chromosomes. Persons with Marfan's syndrome have little fat under the skin, and the chest is caved in. The tendons and ligaments become weak and lax leading to slouching, loose-jointedness, flat feet, malpositioned ears, and cardiovascular defects, such as leakage of the aortic valve.

A person with this heart defect has pulsating arteries that are readily detected in the neck and extremities. Dr. Schwartz offers evidence and tells a story about Lincoln and Noah Brooks, a newspaperman, who were reviewing a photograph for which Mr. Lincoln had posed a short time before. His legs were crossed and Lincoln could not explain why his left foot was not in focus. He was confident he had not moved it. Brooks suggested that the throbbing of the arteries may have caused an imperceptible motion.

The President crossed his legs and watched his foot. "That's it! That's it!" he exclaimed. "Isn't it?" It is reasonable to assume that this was true because his loose-jointedness and relaxed muscles caused the leg to move slightly with each beat of the heart. And movements of the foot have been observed in other victims of Marfan's syndrome with aortic insufficiency of the heart.

Lincoln's heart was failing, doctor believes

Associated Press

LOS ANGELES — President Abraham Lincoln had a bad heart and probably would have died in office even if he had not been assassinated, a doctor says.

Although his diagnosis is based on circumstantial evidence, Dr. Harold Schwartz says he feels certain that Lincoln suffered from a genetic condition known as the Marfan Syndrome. Indications of the syndrome are abnormally long arms and legs, a sunken chest, crossed eyes and a leaking heart valve.

Lincoln had the long arms and legs, the sunken chest, frequent crossing of the eyes and most likely had a leaking heart valve when he went to the Ford Theater April 14, 1865, Schwartz said.

Schwartz, who is on the staff at the University of Southern California School of Medicine, also has turned up genetic evidence that Lincoln had the Marfan Syndrome. In fact, the doctor became interested in Lincoln's case in 1959, when he treated a young boy who had the syndrome. Schwartz learned that the boy and Lincoln had a common ancestor.

Schwartz then made an extensive study of the Lincoln family and drew up genealogical charts based on his inferences.

Schwartz said he believes he has found the family that introduced the Marfan Syndrome gene to the Lincoln family in England in the 15th Century.

In the last two months of his life, Schwartz said, Lincoln became easily fatigued, suffered frequent headaches and was bedridden for a time. Schwartz said this is an indication that Lincoln's heart was failing.

No paper name or date given when sent in.

Was Lincoln Dying L

President **Abraham Lincoln** would have died of a heart ailment within six months even if **John Wilkes Booth** had never fired his fatal bullet in 1865, says a physician who has been researching Lincoln's health for 10 years.

Dr. Harold Schwartz, a heart specialist in Lynwood, Cal., says the 16th president was suffering from an advanced form of aortic insufficiency. He said Lincoln had a defective heart valve which did not close properly, allowing blood to flow back into his heart.

"It was undoubtedly so advanced that Lincoln was going downhill at the time of his fatal visit to the theater," said Schwartz. "I do not believe the President had six months to live when Booth shot him."

Schwartz said his conclusion is based on written reports of Lincoln's physical condition, a photograph which he believes shows the President unable to control trembling of his legs, and his own tracking down of a genetic conition he thinks Lincoln had.

That condition — called the "marfan syndrome" — results in elongated arms, legs and bones and occasionally is linked with heart problems, Schwartz said.

He said he discovered that Lincoln had the syndrome after treating a man related to him, and then tracing the condition back to the President's great-great-grandfather, **Mordecai Lincoln II.**

Abe's Malady

Was it Marfan's syndrome?

To Dr. Harold Schwartz, the signs left little doubt. The seven-year-old boy visiting his Huntington Park, Calif., office in 1959 had Marfan's syndrome, a genetic disorder of the connective tissue that can cause heart and eye problems, affect skeletal growth and occasionally be fatal. A few months later, the boy's grandmother dropped in to inquire about his condition and revealed that her husband had died of Marfan's. The grandmother's married name was Lincoln.

Says Schwartz: "I call that my 'burning bush' moment. I had read Carl Sandburg's biography of Abraham Lincoln, which contains a great deal about Lincoln's physical characteristics." Suddenly everything connected. The Great Emancipator, Schwartz realized, was probably afflicted by Marfan's syndrome.

Since then, Schwartz, now 60, has traced the Lincoln Marfan gene back to 16th century England and now is more certain than ever about his theory. In the *Western Journal of Medicine*, he strongly suggests that had John Wilkes Booth not fired the fatal shot on April 14, 1865, Lincoln would have died within a year from complications of Marfan's syndrome—for which there is still no cure.

Schwartz points to the well-documented fact that Lincoln had disproportionately long arms, legs, hands and feet, even for a man of his height. While watching a regiment of Maine lumbermen during the Civil War, the President himself noted: "I don't believe that there is a man in that regiment with longer arms than mine." In 1907 a sculptor working with Lincoln casts observed that "the first phalanx of the middle finger is nearly half an inch longer than that of an ordinary hand." The President sometimes squinted with his left eye. All of these characteristics, according to Schwartz, are typical of Marfan's syndrome. In fact Lincoln's "spider-like legs," a phrase used by one of the President's contemporaries, was the very simile used in 1896 by French Physician Bernard-Jean Antonin Marfan when he described the syndrome that was named for him.

Schwartz has also presented an ingenious bit of evidence that Lincoln had a specific cardiovascular problem also associated with Marfan's syndrome: imperfect closure of the valves of the aorta, the large artery that carries blood from the heart. The clue appeared in a picture of the President taken in 1863. Lincoln had his legs crossed, and in an otherwise sharp photo, the left foot—suspended in the air—is blurred. When viewing the print, Lincoln asked why the foot was fuzzy. A friend familiar with physiology suggested that the throbbing arteries in the leg might have caused some movement. Lincoln promptly crossed his legs and watched. "That's it!" he exclaimed. "Now that's very curious, isn't it?" Not to Schwartz. The Marfan-caused defect, he points out, results in "aortic regurgitation," which causes pulses of blood strong enough to shake the lower leg.

Schwartz has also found in the President's own words what he believes to be good evidence that before Lincoln was shot he was "in a state of early congestive heart failure"—brought on by his aortic condition. About seven weeks before Lincoln's assassination, for example, he told his friend Joshua Speed: "My feet and hands of late seem to be always cold, and I ought perhaps to be in bed." Though he was only 56 in 1865, Abe was also easily fatigued toward the end. "There is only one word that can express my condition," he said, "and that is 'flabbiness.'" Once, shortly before his death, he tried to get out of bed but fell back, too weak to rise. Only a day before Lincoln was shot, his wife Mary wrote of the President's "severe headache" and indisposition. Concludes Schwartz: the faulty aortic valves resulted in "a decompensating left ventricle which was the undiagnosed or concealed cause of the President's failing health."

Abraham Lincoln, photographed in 1863

In the blurred left foot, an important clue

Schwartz, who teaches medicine at the University of Southern California, concedes that his 20-year study is an "obsession." When his five children visited Disneyland with him, he recalls, he used to have Lincoln-head pennies in his pocket; they would be awarded to the first child who could identify "a Marfan" in the crowd. His office is cluttered with busts of Lincoln. In 1976 he abandoned private practice and joined the geriatric department of a state mental hospital. Reason: so that he could have nights and weekends free to search Lincoln literature for more clues to Marfan's syndrome. ■

Bread and Iron

Too much of a good thing?

As almost everyone knows, iron is routinely added to "enriched" flour and bread because the element, needed to make hemoglobin, is stripped out in the grain-milling process. But disturbing news from Sweden suggests that too much iron may trigger a serious and often fatal hereditary illness. It is an iron storage disorder called hemochromatosis, and it causes its victims, mostly male, to absorb too much iron. Possible results: liver disease, diabetes, impotence, sterility, heart failure, even sudden death.

The disease has always been regarded as extremely rare. But doctors at Ostersund Hospital and in the Swedish district of Hede have just reported a surprising number of cases. After seeing ten cases in two years, Dr. K. Sigvard Olsson and colleagues screened 347 people, 96.4% of the total community between the ages of 30 to 39, for the disorder. No women, but four out of almost 200 men—"a remarkably high figure" of 2%—showed early signs of hemochromatosis.

Swedes get 42% of their dietary iron from fortified foods. The Swedish doctors are careful not to draw a causal link between the incidence of iron overload and Sweden's 30-year-old iron fortification program. But they warn that under such a program, people genetically predisposed to hemochromatosis are at risk.

Still, their findings may deal the final blow to a proposal, heatedly debated since 1970, to triple the present amount of iron added to U.S. flour and breads. Americans now receive about 25% of their dietary iron from such products. The proposal has been endorsed by nutrition experts as a preventive against iron deficiency, especially in women. But hematologists, led by William Crosby of the La Jolla, Calif., Scripps Clinic and Research Foundation, have steadily argued that on the basis of available information, an increase in iron is neither needed, effective nor safe. ■

AMERICAN MEDICAL NEWS * Oct. 3rd., 1980

Letters for publication should be no longer than 300 words, must be signed, and should be addressed to: Editor, American Medical News, 535 N. Dearborn, Chicago, Ill. 60610. AMN reserves the right to edit letters for publication.

LETTERS

Did Lincoln have Marfan's syndrome? MDs say yes

John K. Lattimer, MD, is positive that Abraham Lincoln did not have Marfan's syndrome (Letters, *AMN*, Aug. 15, 1980). I'm sorry that he does not take the possibility more seriously. Dr. Lattimer is an outstanding medical expert on Lincoln and I would hope that his opinion would be based on more than just one symptom — weakness.

All patients with Marfan's taint are not weak, just as all do not have medial necrosis of the aorta, nor do all have arachnodactyly. But it seems awfully difficult to pass over a dislocated lens, long torso, pectus excavatum, huge span from fingertip to fingertip and signs of aortic insufficiency. The established criteria for the diagnosis of this syndrome are arachnodactyly, ectopia lentis, and no urinary homocystine. Other findings such as flat feet, pectus, long narrow face, aortic insufficiency (all of which Lincoln had), are highly suggestive, especially in the presence of an established family history. We know that a relative of Lincoln was diagnosed as having Marfan's syndrome.

I would hope that Dr. Lattimer might re-examine the evidence, especially the plaster casts of Lincoln's hands at the Armed Forces Institute Museum and give us another opinion.

JOHN B. MOSES, MD
Scarsdale, N.Y.

Referring to a prior news item, John K. Lattimer, MD, challenges the assertion that Abraham Lincoln had Marfan's syndrome.

Dr. Lattimer obviously ignores an extensive clinical genetic investigation originating with a boy in my practice who had the Marfan syndrome (TMS) and was a kin of Lincoln. My report (*JAMA*, Feb. 15, 1964) included a family pedigree showing the common ancestor and others similarly affected. Also: Lincoln was tall, lean, stooped and loose-jointed, with disproportionately elongated arms, legs, and feet. Most important he had a pectus excavatum. His father was blind in one eye while his son Robert also had a squint, making three generations with visual involvement. Two of his children died with cardiovascular symptoms as with TMS. Contemporaries depicted Lincoln's legs as "spider-like," the very simile Marfan used in his original case, arachnodactyly being an alternate designation for the disorder.

In 1863, the president described unusual arterial pulsations in his extremities, just as in aortic insufficiency, a common complication of TMS. Later, just before his assassination, he spoke of being very unwell, of unusual fatigue and of cold hands and feet, all consistent with rapid cardiac decompensation and reflex vaso-constriction from aortic regurgitation.

Against all this and more, Dr. Lattimer offers only one point: Lincoln's great strength. He cites the "muscular hands" seen in casts made from life and still "available for comparison," and the president's ability to hold an axe out at arm's length. Actually, a comparative study was made of the casts by an anthropologist long before my interest, my report demonstrating that the measurements derived were highly consistent with arachnodactyly. Also, B. Thomas, a biographer, indicates that Lincoln's strength was in his long body and long arms, which could exert tremendous leverage, especially as "on an axe." The mechanical advantage of the president's elongated skeletal structure is in fact positive evidence for TMS rather than otherwise.

The diagnosis, it appears, can hardly be considered "groundless."

HAROLD SCHWARTZ, MD
Lakewood, Calif.

Lincoln Did Not Have the Marfan Syndrome

Documented evidence

JOHN K. LATTIMER, M.D., Sc.D.
New York City

College of Physicians and Surgeons of Columbia University and The Presbyterian Hospital

Two medical authors[1,2] have suggested the diagnosis of the Marfan syndrome for Abraham Lincoln; they have been parroted by others, particularly by the popular press. This study examines the arguments they presented and offers firm evidence that Lincoln himself definitely did not fit the classical Marfan syndrome.

In 1962, A. M. Gordon, M.D.,[1] wrote an excellent article proposing the interesting speculation that Abraham Lincoln might have fitted into the classical syndrome described by A. B. Marfan, M.D.,[3] which he had been studying; he presented this possibility as an intriguing conjecture, letting the reader decide for himself how appealing or valid the speculation appeared. He proposed that the defective gene transmitting the Marfan syndrome might have come through Lincoln's mother, Nancy Hanks. In 1964, a second author, Harold Schwartz, M.D.,[2] of California, seized on Gordon's proposal, but disagreed with him, attributing the link to Lincoln's father's family. He offered an inferential family diagram, relating an 11-year-old male patient of his to an alleged common Lincoln ancestor, born some 300 years (nine generations) before, but identified no other cases during this time (since 1687). Since the Marfan syndrome is inherited as an autosomal dominant, these sporadic cases might seem more likely to be new mutations. Both authors[1,2] proposed that Lincoln had the classic, full-blown Marfan syndrome.

Schwartz next declared that Lincoln had aortic insufficiency, and then went on to state that Lincoln was about to die from it.[4] Evidence to support either of these claims is lacking, as we shall see.

If the trait had indeed been present on both sides of Lincoln's family, most of his sons should have had the classic syndrome, yet none of his sons were identified as having any of the inherent characteristics. In fact, on close examination, it seems clear to me that the data by which the "diagnosis" was pinned on President Lincoln are inadequate, and that on the opposite side of the argument, there are many more hard facts and materials available that indicate, and virtually establish, that Lincoln himself did not fall into the Marfan category.

Lincoln was a lanky, tough, muscular frontiersman. In his youth, he outfought the most powerful rough-and-tumble wrestling champions in his part of the country. He was a commander (captain) of troops that marched against the Indians in the Black Hawk War. He made his living as a rail splitter at one stage, and, just before he was assassinated, at age 56, he put on a display of woodchopping with a heavy axe, making the chips fly in all directions, a feat that amazed the audience of convalescent servicemen at the soldier's home near Washington. He finished by standing up and holding the heavy axe out at arm's length by the tip of the handle. A man with the classic Marfan syndrome would not be able to do this, since the connective-tissue defect would have prevented him from locking his fingers and wrist. This cluster of facts alone puts Lincoln outside the syndrome of disabilities later described by Marfan, in 1896,[3] 31 years after Lincoln's death. But, let us examine the criteria and the facts in more detail.

What is Marfan syndrome?

The Marfan syndrome is classified, according to Pyeritz and McKusick,[5,6] as a heritable disorder of connective tissue. Because its clinical and pathologic alterations involve connective-tissue supporting elements, it is assumed to be an inborn error of protein metabolism, particularly in collagen or elastin, in its classic form. It is associated with disorders of the connective tissues of the eye, the aorta, the long bones, and the joints.[7] Marfan,[3] writing in the *Bulletin of the Society of the Medical Hospitals of Paris* in 1896, described in detail a young patient with symmetrical elongation of hands and feet. In the drawings he made of her hands, the skull was elongated and the fingers so slender that they resembled the legs of a spider (Fig. 1); her joints tended to have contractures. One brother had a congenital cataract and one sister a strabismus. Six years later, two other French physicians, H. Mery, M.D., and L. Babonneix, M.D. (1902), described the same patient and noted that severe dorsal kyphoscoliosis had also developed.[8] In other patients, extreme laxity of the joints was noted.

With further study, it has turned out that these

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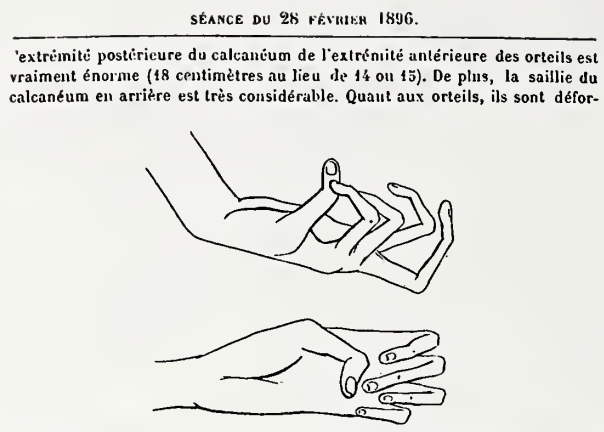

SÉANCE DU 28 FÉVRIER 1896.

l'extrémité postérieure du calcanéum de l'extrémité antérieure des orteils est vraiment énorme (18 centimètres au lieu de 14 ou 15). De plus, la saillie du calcanéum en arrière est très considérable. Quant aux orteils, ils sont défor-

FIGURE 1. Arachnodactyly (spider fingers) as illustrated in drawing taken from Dr. Marfan's[3] original article describing syndrome. Slender bones resemble legs of large spider. (Credit, B. J. Marfan)

patients with the Marfan syndrome are suffering from a connective-tissue deficiency which classically leaves their joints limber and easily bent backward. Their bones are so slender and their interosseous muscle masses so inadequate that their fingers actually do look like the legs of a spider (Fig. 1). Their thumbs are so long that when they wrap their other fingers around their thumbs, to "make a fist," the tips of the thumbs may project well beyond the lateral border of the fist itself.[9]

The supporting structures of the lenses in their elongated eyeballs are often so weak that the lens dislocates in as many as 60 percent of the cases, resulting in a severe ocular disability, following on the myopia from the elongated eyeball. Detached retina is common and the corneas of these patients with Marfan syndrome are nearly always abnormally flat. The flat cornea, the elongated globe, and the easy detachment of the lens or retina make for severe visual problems, beginning with myopia (not hyperopia, as Lincoln had). The connective tissue of the first portion of the aorta is often so weak that dilation of the aorta is common, with either rupture, dissecting aneurysm, or incompetence of the aortic valve.

Classically, the long bones are not only thin, but are also very long, so that the span between the fingertips of the outstretched arms exceeds the patient's height. Unfortunately, as Pyeritz and McKusick[6] and Eldridge[10] have pointed out, this is also so often true of normal tall men as to be of no value by itself as a diagnostic sign. The measurements of the lower segment of the body (from pubis to heels) run longer than the upper segment; this is also so common in normal tall men that it is of no value, by itself, in diagnosing Marfan's syndrome.

Scoliosis, kyphosis, spondylolisthesis, pectus deformity, slipped epiphysis, and extreme laxity and instability of the joints are all very common among patients with the Marfan syndrome, due to their weak supportive ligaments. It is the connective-tissue deficiency that causes the disorders of various systems.

Pyeritz and McKusick[6] point out that the Marfan syndrome is inherited as an autosomal dominant; in only 15 percent of cases, do other family members lack signs of the syndrome. The average paternal age is increased in these sporadic cases, thereby supporting the notion that they represent new mutations. In genetic counseling, families are advised that "skipped-generation" exceptions do not occur.[6]

Pyeritz and McKusick[6] go on to state that one unfortunate result of clinicians' increased awareness of Marfan phenotypes is the growing tendency to label the syndrome, in a person who has few manifestations, a "forme fruste," an atypical form. Patients with various other syndromes, or even healthy subjects, may have one or a few features of the Marfan syndrome; if the term "forme fruste" were to be indiscriminately applied, only confusion would result. In short, a patient with the classic Marfan syndrome, with which it has been alleged that Lincoln was afflicted, would have the following symptoms, not one of which do we know Lincoln had:

1. Lax and unstable joints.
2. Weakness of the suspensory ligaments of the lenses of his eyes.
3. Nearsightedness (he was farsighted).
4. Scoliosis or other skeletal defects.
5. Arachnodactyly, which the casts of his hands prove he did not have.
6. A large hat size, from dolichocephaly.

As to skeletal disproportions, we have no actual measurements to which we can refer, but we know that by themselves these measurements are so often found in normal men that they are worthless in the absence of other conclusive findings.

What evidence is there that Lincoln had Marfan's?

Actually, there is no firm evidence that Lincoln had the Marfan syndrome.

On the contrary, Lincoln's fingers were big, thick, and powerful. Fortunately, we have, for analysis and examination, the plaster casts of Lincoln's hands made by sculptor Leonard Volk in the year 1860 (Fig. 2A, B, and C). They show the thick bones and powerful musculature of a big strong man with normally proportioned hands, rather than the excessively slender bones and excessively long thumbs of someone with the classical Marfan syndrome.

In Figure 2A and B, the casts of Lincoln's hands can be seen alongside the hands of a man of the same height but with Marfan's syndrome. It is quite obvious that Lincoln's hands have thicker bones and more muscularity by a large margin. One of the characteristics mentioned by Marfan[3] is the lack of muscle mass, and it is obvious that the interosseous and thenar muscles are deficient in the Marfan syndrome hands with their excessively slender bones, whereas these features are totally absent in Lincoln's

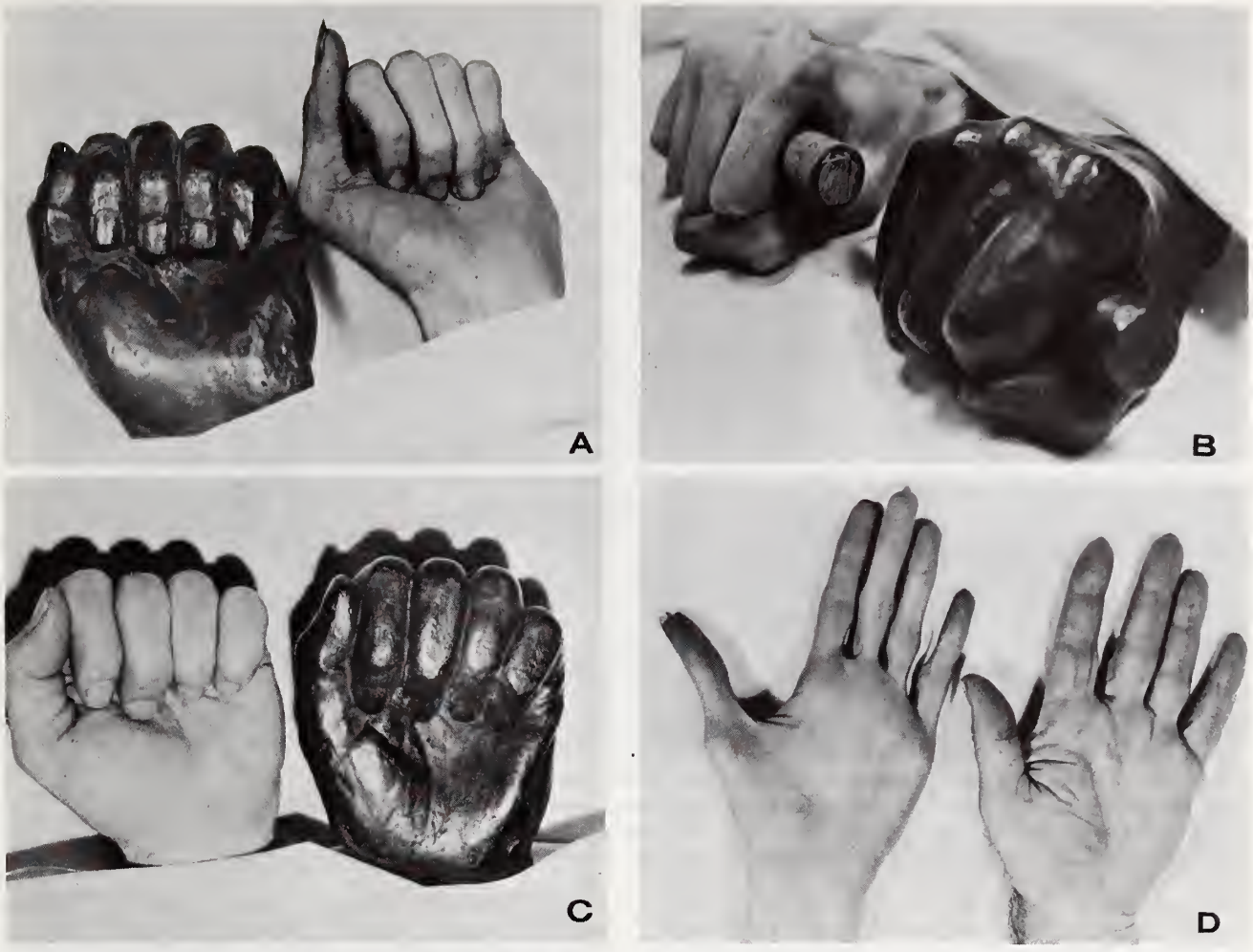

FIGURE 2. (A) Cast of Lincoln's hand (left) alongside hand of man of same height, with Marfan syndrome (right). Lincoln's fingers are thick and hands muscular. Marfan syndrome hand has excessively slender bones and deficient muscularity. Marfan syndrome thumb is excessively long; Lincoln's is of normal length. (B) Lincoln's hand compared with Marfan syndrome hand. Cast of Lincoln's right hand, grasping segment of broomstick, compared with right hand of man of same height with Marfan syndrome, grasping similar piece of broomstick. Fingers of Lincoln's hand are so much thicker and hand so much more muscular than that of Marfan syndrome man that difference is obvious. (C) Cast of Lincoln's left hand and left hand of normal man of same height. Note similar thickness and proportions of fingers, hand muscles, and similar length of thumbs. (D) Marfan hand compared with normal hand. Extended left hands of two men, each 6 feet, four inches tall; left: Marfan syndrome hand, right: normal hand. Normal hand same proportions as Lincoln's hand. (Courtesy, John K. Lattimer, M.D., Sc.D.)

hands. The excessively long thumb of patients with the Marfan syndrome is clearly visible in the patient's hands shown in Figure 2A, B, and D. If a patient with the Marfan syndrome were to wrap his fist around his thumb, the thumb would protrude well beyond the fifth finger[9]; it is obvious from the casts of Lincoln's hands that he does not have this excessively long thumb (Fig. 2A, B, and C). The hand of a normal man of six feet, four inches, is also shown with the casts of Lincoln's hands in Figure 2C and with Marfan syndrome hands in Figure 2D to demonstrate how normal Lincoln's hands were.

The configuration of the fingers of one of Marfan's original patients, which was what attracted his attention to this syndrome, are shown in the drawing taken from Marfan's[3] original article (Fig. 1). These are characteristics of the appearance of patients with the Marfan syndrome when you examine them; they do indeed look like the slender legs of a large spider, and it is quite obvious that Lincoln's fingers did not have this configuration. It has been said that the plaster cast of Lincoln's left hand makes it seem larger than his right hand, but his hands are shown in such different poses that I cannot verify this allegation. The fact that the phalanges of his middle fingers are longer than those of his other fingers is not sufficiently different from those of normal large men to afford it diagnostic significance.

The critics quote sculptors and anthropologists who examined and measured these casts of Lincoln's hands and who commented that he had long hands and fingers in keeping with his elongated frame; they did not suggest that his hands were disproportionately long nor his bones disproportionately

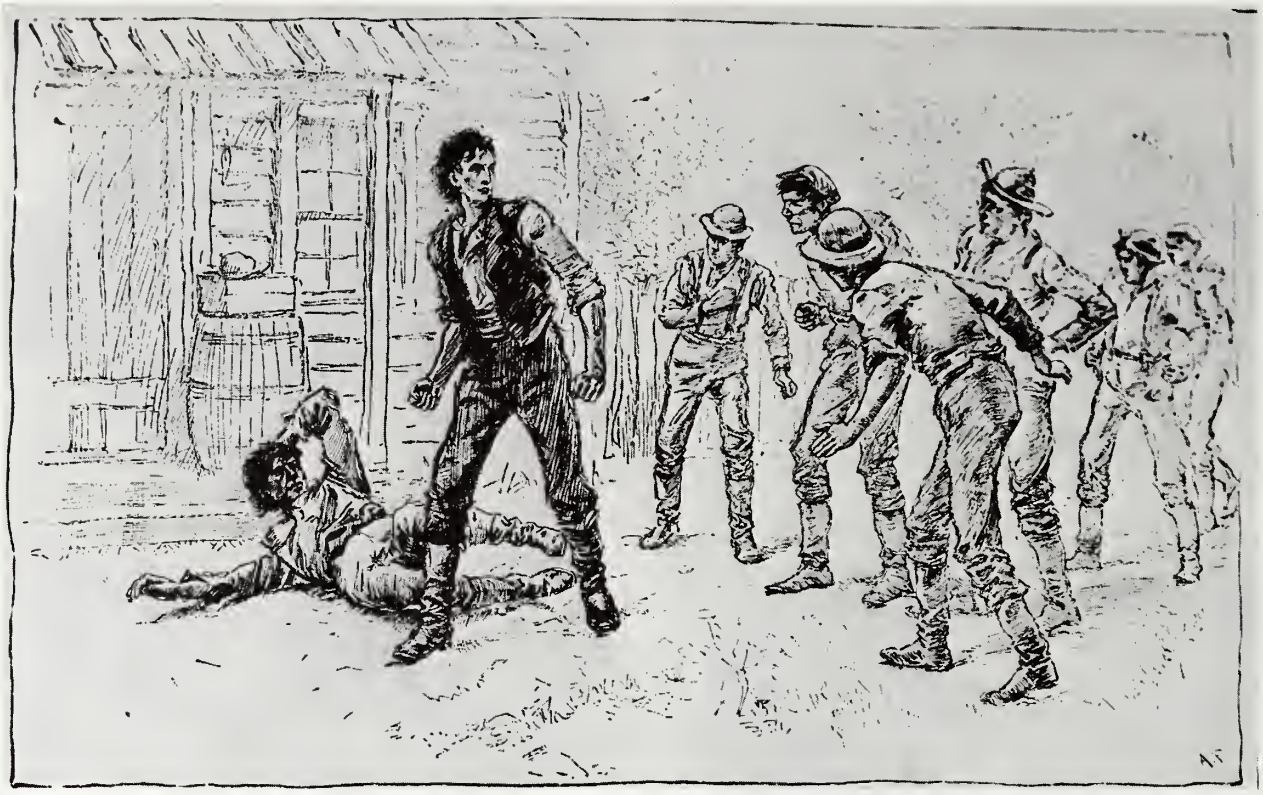

FIGURE 3. Lincoln, champion wrestler. In his youth, Lincoln beat toughest frontier wrestlers. If he had had connective-tissue deficiency of Marfan syndrome, his opponents could have bent his fingers, wrists, and even elbows back and escaped from his grasp, with greatest of ease, but they could not. His head would inevitably have hit ground many times, yet his lenses and retinas did not detach or dislocate even with this trauma. (Credit, Noah Brooks)

slender.[11]

Dolichocephaly

Another common deformity in patients with the full-blown Marfan syndrome is elongation of the skull. The accompanying elongation of the eye sockets is thought to add to the tension on the eyeballs; this helps brings about the lens dislocations and the detachments of the retinas that are so frequent in patients with the Marfan syndrome.

Lincoln's head, judged from the life masks made of him, was not abnormally elongated. On the contrary, he wore a hat size of only seven and one quarter, which is smaller than usual for a man of six feet, four inches, in height.

Here, again, we have a measurement that is a strong argument against the diagnosis of the Marfan syndrome.

The connective-tissue deficiency of the hands of people with Marfan's syndrome would make it impossible for them to grasp a heavy wrestling opponent and hold him down; the opponent could take each finger of the man with Marfan's syndrome and bend it backward. In fact, he could bend back the entire wrist and arm and thus escape from any "hold" at all, even if the man with the Marfan syndrome should succeed in wrestling him down to the ground. Lincoln's history as a champion rough-and-tumble wrestler (Fig. 3) is a powerful argument against his having the weak connective tissues of the hands and arms of a patient with the Marfan syndrome. Anyone who has wrestled knows that a height of six feet, four inches, is enough of a disadvantage to overcome, and that lax joints, in addition, would have made it impossible to be the champion wrestler that Lincoln was.

The lack of muscle mass is another characteristic of patients with the Marfan syndrome (mentioned in the original description[3]), but Lincoln's nude body on the autopsy table was so muscular that it elicited a spontaneous comment by one of the physicians who assisted with the autopsy.[12,13] Edward Curtis, M.D.,[14] writing to his mother within the week after the autopsy, stated as follows:

> I was simply astonished at the showing of the nude remains, where well rounded muscles built upon strong bones told the powerful athlete. Now did I understand the deeds of prowess recorded of the President's early days.

Did Lincoln have pectus excavatum?

Evidence for this "diagnosis" does not exist, as far as I can determine, despite allegations by one of the authors[2] that it might have been present. At the autopsy, his nude remains were viewed by those present, and no mention of pectus excavatum or pectus carinatum was made. On the contrary, Curtis[14] stated his surprise at seeing such "powerful

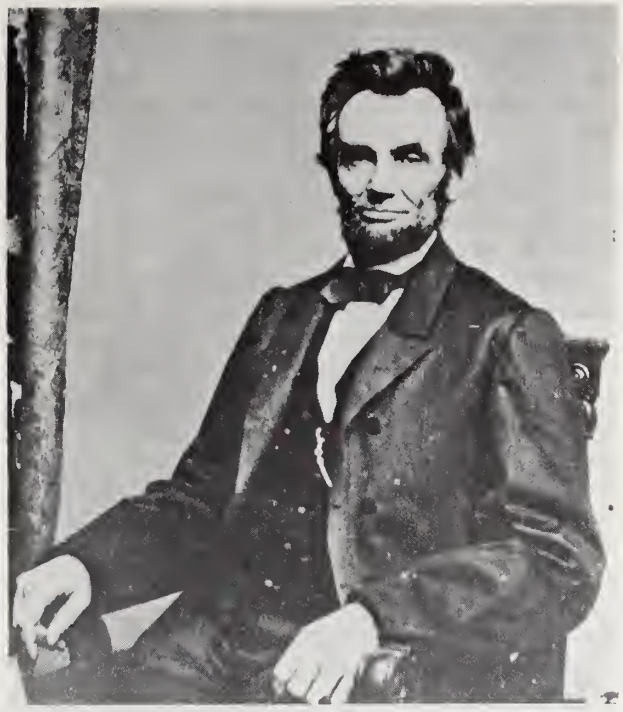

FIGURE 4. Lincoln's chest not depressed. Photograph shows Lincoln's chest well rounded, no sign of pectus excavatum or of scoliosis, both so common in Marfan syndrome patients. (Credit, Matthew Brady)

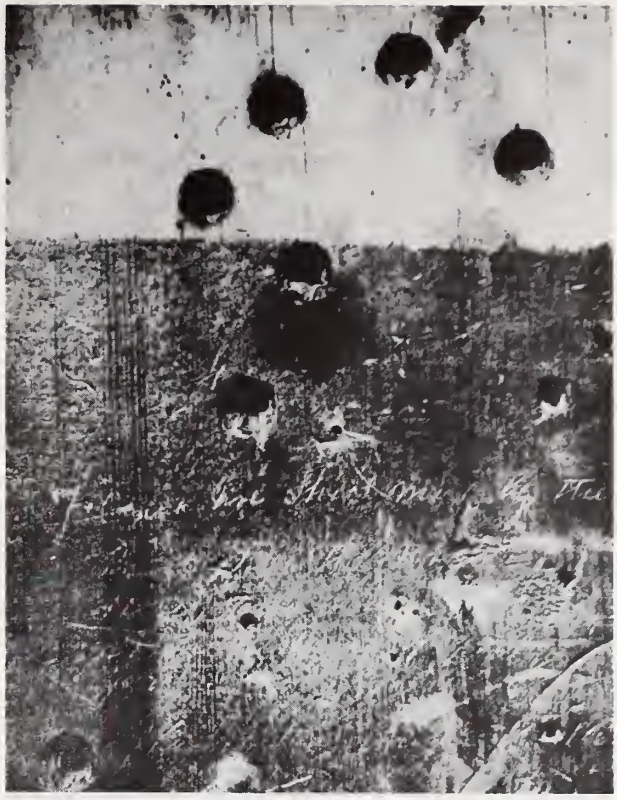

FIGURE 5. Seven bullet holes in board used as target by Lincoln in firing seven consecutive shots. Lincoln was testing very important 50-caliber Spencer carbine that helped to shorten Civil War. His visual acuity was obviously superior, rather than impaired; he stated that he had fired heavy-caliber rifles many times. Head-jolting recoil of these large-caliber rifles is kind of trauma that dislocates lenses and detaches retinas in patients with elongating eyeballs of Marfan syndrome, yet Lincoln had had no such trouble. (Credit, *Saturday Evening Post*[17])

musculature built upon strong bones."

It is true that William Herndon[15] (Lincoln's law partner) once said of him in his later years that "Mr. Lincoln was six feet four inches high, thin, sinewy and raw boned, thin through the breast to the back and narrow across the shoulders." These words could describe many men who are six feet four inches tall, however, and his chest was certainly not flat as can actually be seen in Figure 4, which shows a perfectly well-rounded and normal-looking chest and sternum area. Sandburg[15] once referred to his legs as reminding him of a great spider, possibly a "Daddy Long Legs" type; however, he did not, as did Marfan,[3] characterize his fingers as being abnormally thin (arachnodactyly refers to fingers, not legs).

Eye defects in Marfan syndrome

People with Marfan syndrome also tend to have a weakness of the connective-tissue ligaments that support the lenses of their eyes. Their skulls elongate as they grow, putting stresses on their eyeballs. Thus they are especially prone to have dislocations of the lenses; a very substantial proportion of them, especially the active men, have this condition and thereafter have a severe impairment of vision, necessitating the wearing of thick eyeglasses as compensation. Lincoln, on the other hand, obviously had no such dislocation, despite the fact that he was kicked in the forehead by a horse and knocked unconscious, as a youth.[16] It was inevitable that he was slammed to the hard ground many times as a wrestler (Fig. 3). He had fired, with great skill, a large-calibre military carbine, which he said he had done many times, with a severe head-jolting recoil (Fig. 5); all these things were done with no dislocation of the lens or detachment of the retina. If his lens supports had been weak, they would surely have given way long before he was 56 years of age, with this kind of rough treatment.

Lincoln was farsighted (persons with Marfan's syndrome are nearsighted)

Lincoln's reading glasses were somewhat "strong," but were within the range (plus 6.75 diopters) used for reading by other 56-year-old men who had no electric lights to read by. He inspected reports and wrote incessantly. During his administration, he personally signed all the military commissions of the many thousands of Army and Navy officers, as well as the many thousand papers of officials whose documents of appointment and discharge came to his desk for signature. His handwriting remained precise and admirable, all through his life.

The occasional tendency of his left eye to roll up

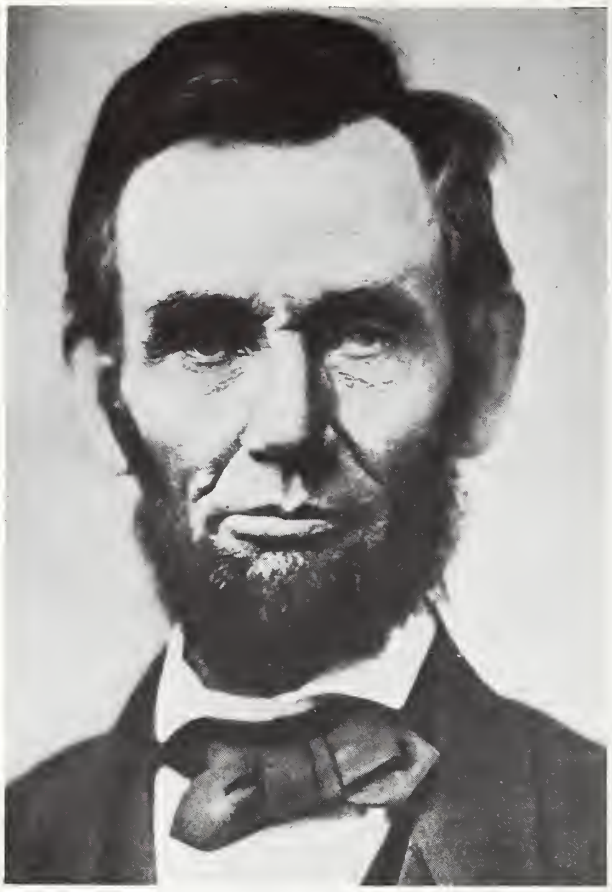

FIGURE 6. Lincoln's slight eye deviation. Very slight (even questionable) upward deviation of Lincoln's left pupil can be seen in this famous portrait by Alexander Gardner taken of Lincoln at age 54. It is certainly not dislocated-lens type of defect so characteristic of Marfan syndrome. (Credit, Alexander Gardner, National Archives, Washington, D.C.)

a bit was so slight, and even questionable, that it is barely discernible in this famous photograph by Alexander Gardner, taken in 1863 (Fig. 6); it obviously was due to a mild muscular weakness rather than to a dislocated lens and was obviously of minor degree. He had had only one episode of double vision and had tried repeatedly to duplicate it without success.

Men with the classic Marfan syndrome are nearsighted; this is due to their eyeballs becoming elongated, as their skulls elongate. Lincoln was just the opposite. This is another powerful argument against Lincoln having the Marfan syndrome.

Visual acuity

Lincoln was perfectly able to fire a heavy military rifle with open sights, with great accuracy, as recently as 20 months prior to his death. We know this because Christopher Spencer, the inventor, brought forth a repeating carbine of such advanced design that it proved to be of great value to the Northern Army.[17] Sherman's troops used it in the campaign to cut the South in half and it was so effective that the Southerners characterized it as practically an immoral weapon. They said the Yankees could "load up on Sunday and shoot all week" with it, and this was just not fair.[17] However, the lethargy of government bureaucracy in Washington was just as bad then as it is now. The Union supply officers steadfastly refused to accept this new invention, preferring to stick with the tried-and-true, single-shot, muzzle-loading muskets; then on order. When Spencer was able to reach Lincoln himself, the President was intrigued and recognized the merit of the new firearm so quickly that he and the inventor walked out to where the Washington Monument was being built to fire at a target made from an old board from the White House lumber pile. Lincoln spoke of having fired many of the high-powered rifles of the day, all of which had an abrupt, severe recoil which would have jarred the eyeballs violently. Lincoln's accuracy was excellent, considering the relative crudeness of this weapon. A photograph of the board with the seven bullet holes in it, which he made with seven shots, is displayed in Figure 5.[17] It would seem extremely doubtful that a 54-year-old man with the Marfan syndrome could perform this feat of visual acuity, in addition to tolerating the hard jolting recoil of each shot; this could have led to dislocation of his lens, or detachment of his retina, if he had indeed had the Marfan syndrome.

Thus we see on actual inspection that the eye lesion of Lincoln was so minimal that it was of absolutely no value in proving the diagnosis of the Marfan syndrome (Fig. 6). Many people, with or without the Marfan syndrome, have this degree of muscle weakness. It has been stated that various relatives of Lincoln had poor eyesight, or a poorly functioning or missing eye, but in the rough-and-tumble frontier community this was so common that it in no way substantiated the existence of a Marfan-syndrome condition in Lincoln, as I see it. The fact that the distance between Lincoln's outstretched arms and fingertips exceeded his height is a finding that is by no means confined to patients with the Marfan syndrome. In the classic article on this point, Pyeritz and McKusick[6] have stated as follows, "Each of the clinical features of the Marfan syndrome occurs with variable frequencies in the general population and it is to be expected that several will occur together by chance alone, in some cases. The overlap between the normal and the Marfan syndrome subjects was so great that it makes this an unreliable point of diagnosis.

Family History

Gordon[1] speculated that Lincoln's Marfan syndrome trait might have descended to him through his mother, whereas Schwartz,[2] the enthusiast, claims that it descended to him through his father. He described a patient with the Marfan syndrome who claimed a distant relationship to Lincoln. Although it is true that this condition is often familial, the overlap in the measurement of very tall, lanky people

FIGURE 7. Blurring of feet due to being out of focus. (A) Famous photograph in which Lincoln, himself, noted that toe of his boot, much closer to camera than to this face, was more blurred, as is evident. Someone suggested that he cross his legs and would see that toe of his boot jiggled from his normal pulse, as is indeed true. In this instance, however, it is far more likely that blurring was due to wide-lens aperture with resulting poor depth of focus, than to pulsations of foot. Note that even foot resting on floor is slightly out of focus. Photograph demonstrates that foot was often out of focus even if resting on floor. It was this possibility that foot was jiggling because of excessive pulse pressure, seized on by critics to indicate aortic valve disease in Lincoln. This seems to me to be total inadequate basis on which to make diagnosis of aortic insufficiency where blurring of foot was actually due to optical effect, rather than to pulsations of arteries in leg. (B) Toe closest to camera often blurred by being out of focus in old, very lengthy time exposures where lens aperture was widely open; this decreased depth of focus. This was sometimes also true of both feet, even when one foot was firmly on floor, as in this photograph. Note that plane of Lincoln's face is only plane that is sharp and in focus. (Credit, Alexander Gardner)

and people with Marfan's syndrome voids the issue. The issue here is whether or not Lincoln himself fell into the Marfan syndrome category, and no evidence has been offered that he did, as far as I can determine.

Lack of evidence for aortic insufficiency

The only evidence offered to support the speculation that Lincoln had aortic insufficiency is the blurred image of Lincoln's left toe, as he sat with his legs crossed for one of Matthew Brady's long time-exposures on November 8, 1863, with his one foot protruding far out toward the camera (Fig. 7A). This fact is used as the only support for the claim that Lincoln had aortic insufficiency, which turns out to be no support at all, as we shall see.

One of the other connective-tissue defects to which Marfan syndrome patients are particularly liable is prolapse of the aortic valve due to connective-tissue weakness. This is a serious malady of which Lincoln had no actual signs, as far as I could research, and I am puzzled as to how this "diagnosis" was reached. As far as I can determine, the only observed fact on which this speculation is based is that when Lincoln sat with his legs crossed and the toe of one boot extended directly toward the camera for one of Brady's lengthy time exposures of 12 to 15 seconds, he noticed that the toe that was closest to the camera was slightly blurred (Fig. 7A). First of all, when you reproduce this position, you find that the toe of that foot would have been at least three feet closer to the camera than the plane of Lincoln's face, on which the camera was focused. Lincoln's toe might therefore have been considerably out of focus, as it is in Figure

7. When you look closely, you find that the toes of both boots are out of focus, even where one rests on the floor. It is true that if you cross your legs in this position, you will find that the tip of your toe bounces up and down very slightly but very definitely, with every pulse; this is true even if you do not have high blood pressure or a high pulse pressure.

Actually, if you examine Figure 7 more closely, you can see that even the opposite toe, which is resting on the ground, is also blurred by being out of focus. This clearly demonstrates that the blurring of the toe of his boot is primarily due to its being out of focus, not to any alleged aortic insufficiency.

If there are other evidences of aortic insufficiency, I have not been able to find them, and would welcome being informed of them by those Marfan syndrome proponents from whom I am sure I will hear with all possible alacrity.

The fact that other family members were not tall and thin has been passed off with the contention that they were "formes frustes" of the Marfan syndrome; this is merely a convenient way out. Again, however, authorities Pyeritz and McKusick,[6] writing in the *New England Journal of Medicine*, quoted a recent study of 18 cases called "formes frustes" because of severe aortic disease resembling that of the Marfan syndrome; it failed to detect a single person who conformed to the classic Marfan syndrome.

The fact has been raised that Lincoln complained of chilly feelings or feeling poorly or having depressions at various times. We know that he had recurring malaria, and it seems to me that he was entitled to these symptoms, considering the severe stresses under which he lived, the monumental decisions he undertook, and the miserably uncomfortable living conditions of war-time Washington. The White House was bordered by swamps, and during the summer months the flies were so bad as to compel him to leave the White House at all possible opportunities for the Old Soldier's Home on the outskirts of Washington. The winters without central heating must have been truly miserable. The symptoms he describes are typical of those my own grandparents described in their relatively stressless farmhouse life of the same period. I can see nothing whatsoever in these symptoms to indicate that Lincoln had the Marfan syndrome.

The fact that Lincoln lived and remained sturdy to age 56 was in itself an unlikely "old age" for a man with the classic Marfan syndrome to attain, in that era.[18] The average age of death in Murdoch's[18] series was 32 years; in the series of Hirst it was 30 years.[18] Old age is rare, although not impossible.

Summary

There is insufficient evidence to support the diagnosis of aortic insufficiency, of dislocated lens, or of any other severe ocular troubles in Mr. Lincoln. The dimensions of Lincoln's body were within the normal range of those for tall, lanky persons. His record of great muscular strength as a champion wrestler and the lack of symptoms that would suggest connective-tissue deficiency make it impossible to pin the diagnosis of the Marfan syndrome on Lincoln himself, as I see it. The most impressive exhibits I have been able to discover to date are the plaster casts of his hands, taken from life, which are grossly heavier boned than those of patients with the classical Marfan syndrome, when you actually place the hands of patients with the Marfan syndrome beside the casts for comparison. It is my firm belief that Lincoln himself did not have the Marfan syndrome. He was a big, tough, muscular frontiersman with no evidence of a connective-tissue, muscular, or bony defect.

As a child, Dennis Hanks (Lincoln's cousin and boyhood playmate) described Lincoln as a "powerful boy, round, fat, plump, well-made and well proportioned."[19] "In rasslin, running, an' hoss-back ridin' and log rollin,' and railsplittin,' he could beat everybody. You'd 'a' thought there was two men in the woods when he got inter it with an axe."[20] His friend Nat Grisby called him "stout, withy and wiry."[19] This is not the description of a man with the Marfan syndrome. Marfan syndrome people are frail, often smart, but physically fragile.

In short, Lincoln did not have arachnodactyly, as do patients with the classic Marfan syndrome. Lincoln did not have joint laxity, as do patients with the classic Marfan syndrome. Lincoln did not have fragile connective-tissue supports for the lenses of his eyes, as do patients with the classic Marfan syndrome.

Lincoln was farsighted, not nearsighted (as are persons with the classic Marfan syndrome). This, in itself, is a powerful argument against the Marfan syndrome. Lincoln showed no demonstrable signs of aortic disease; his "blurred foot" was merely the result of an out-of-focus portion of a photograph.

The familial case of the one boy with the Marfan syndrome who is said to share an ancient common ancestor with Lincoln is so solitary and so distant that it is unconvincing, at least to date.

Although we commend Dr. Gordon[1] on an interesting suggestion, we call on him and Dr. Schwartz[2] to reexamine the actual evidence and to stop encouraging the avid media and the uncritical authors who repeat and thereby perpetuate this unsupported diagnosis. The evidence clearly indicates that Lincoln himself did not belong in the Marfan syndrome category. Borit has said about those who study Lincoln, "Idolatry we do not need, but neither do we need to make our great men small."

Certainly we should not make it an "obsession"[4] to pin the diagnosis of Marfan syndrome on Abraham Lincoln.

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Lincoln's familiar lanky image

By ROBERT LOCKE
AP Science Writer

LOS ANGELES (AP) — Abraham Lincoln's familiar image — the commanding height, lanky frame, great hands and gaunt face of so many faded photographs — was caused by a hereditary disease that trails his descendants to this day, says a physician at the University of Southern California.

Dr. Harold Schwartz said the lifelong effects of Marfan syndrome were reaching a climax in the 56-year-old president in 1865 and would probably have killed him within a year had not an assassin struck first.

The genetic disorder, which Schwartz says produces "a disproportionate elongation of the skeletal system," can also change the aorta, the main blood vessel from the heart, cause its valve to malfunction and, ultimately, result in progressive heart failure.

Based on Lincoln's words and those written about him, Schwartz concluded: "He was already in heart failure at the time he was assassinated. He could not have lived more than 6 to 12 months."

Schwartz, of the USC medical school, has been studying Lincoln and Marfan syndrome for three decades, ever since he examined a 7-year-old boy whose mother was concerned that "he was so disproportionately tall."

The boy was a classic example of the genetic abnormality and "the family's name was Lincoln. I knew immediately that Abraham Lincoln had the same thing."

Since then, Schwartz said in an interview, he has traced the Lincoln family tree, confirming his young patient's kinship with the 16th president, and studied newspapers, books, pictures and letters concerning Lincoln. The study of his suburban Lakewood home is a small museum of Lincoln memorabilia.

"I found a whole world of confirmation ... It's not hypothetical at all," he said. "I have merely put together the facts that have been given ... by the president and his contemporaries and the facts in the medical literature."

Schwartz said he followed the Lincoln roots back to England and found current branches around the United States, where "the Lincolns are all over the country."

Marfan syndrome, which varies widely in severity, has followed the Lincolns. It affects, by one estimate, at least five of every 100,000 Americans of all sexes and races, Schwartz said. "That figure should probably be multiplied by 5 to 10," he added, "and even that may be conservative."

Marfan syndrome, caused by a dominant gene that affects men and women equally, was named for French pediatrician Bernard-Jean Marfan, who first described it in 1896. Schwartz said those affected, in extreme cases, "are usually very tall and lean. Their arms are long and the fingers get very long and thin. The lower part of the body gets very long."

The face is gaunt and angular, with large ears and nose. The overall appearance is one of gawkiness because of the long, loose-jointed limbs.

The scientific term for the syndrome is arachnodactyly, derived from the Latin word for spider.

Schwartz noted that at least one Lincoln contemporary said the president

caused by hereditary disease

had "spiderlike legs" when seated. Lincoln himself commented on the size of his feet and the length of his arms. That's confirmed by the few measurements available of the 6-foot-4 president.

Schwartz said Lincoln's loose posture is also characteristic of the syndrome, as is evidence of severe farsightedness and periodic squinting.

That Lincoln's heart was failing as a complication of the inherited problem, Schwartz said, is strongly suggested by this Lincoln statement shortly before his death: "I am very unwell now; my feet and hands of late seem to be always cold, and I ought perhaps to be in bed."

Cold extremities are a common result of a heart no longer able to pump enough blood for all the body's needs.

Lincoln was widely reported to be ailing near the end of his life. Pictures taken before and after his first term show a man aged and weathered drastically in four years. The face bore what one observer called, "a look as oi one on whom sorrow and care had done their worst without victory."

The most common explanation has been the pressures of presiding over perhaps the most difficult years of America's history.

But, Schwartz argues, the president's condition, especially the quick fatigue — which Lincoln called "flabbiness" — continued to worsen as the war ended and he won re-election, when the pressures should have eased.

He said many clues point to a failing heart, but the key evidence is a photograph in which Lincoln's foot, dangling from a crossed leg, is blurred. In discussing that blurriness, the president said his foot moved with each surge of his pulse.

Schwartz said both physicians and victims have described just such pulsations as a characteristic of aortic insufficiency.

He said he hopes his study of the Great Emancipator "will help take the stigma out of genetic disease" and will make doctors and parents more aware of Marfan syndrome because "it needs to be recognized. It needs diagnosis and it needs observation.

Lanky Lincoln Doomed by Fatal Genetic

Schenectady, N.Y. Gazette-Feb. 12, 1982

By ROBERT LOCKE

LOS ANGELES (AP) — Abraham Lincoln's familiar image — the commanding height, lanky frame, great hands and gaunt face of so many faded photographs — was caused by a hereditary disease that trails his descendants to this day, says a physician at the University of Southern California.

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Schwartz said he followed the Lincoln roots back to England and found current branches around the United States, where "the Lincolns are all over the country."

Marfan syndrome, which varies widely in severity, has followed the Lincolns. It affects, by one estimate, at least five of every 100,000 Americans of all sexes and races, Schwartz said. "That figure should probably be multiplied by five to 10," he added, "and even that may be conservative."

"There is treatment," Schwartz said, for the life-threatening complications of the disease, such as heart problems. Marfan syndrome, like almost all genetic disorders, cannot be cured.

Marfan syndrome, caused by a dominant gene that affects men and women equally, was named for French pediatrician Bernard-Jean Marfan, who first described it in 1896. Schwartz said those affected, in extreme cases, "are usually very tall and lean. Their arms are long and the fingers get very long and thin. The lower part of the body gets very long."

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Disease, Researcher Says

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—(AP Laserphoto)

LINCOLN LOOK LETHAL — President Abraham Lincoln's distinctive long and lanky looks were symptoms of Marfan syndrome, according to a study by Dr. Harold Schwartz of the University of Southern California. The researcher said the genetic disease, which he traced to Lincoln's ancestors and descendants, caused the president to suffer a heart disease that Schwartz believes may have killed Lincoln within a year, had not an assassin's bullet struck first in 1865.

He said many clues point to a failing heart, but the key evidence is a photograph in which Lincoln's foot, dangling from a crossed leg, is blurred. In discussing that blurriness, the president said his foot moved with each surge of his pulse.

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Q&A

A theory on why Lincoln looked so lanky, felt so ill

2-12-1982

Dr. Harold Schwartz contends that Abraham Lincoln's unusual appearance was caused by Marfan syndrome — a hereditary disease that results in elongation of the body and disfigurement of the heart's aorta. The USC School of Medicine professor hypothesizes that Lincoln was suffering heart failure in the last months of his life, and would not have lived more than a year, even if he had not been assassinated. Schwartz was interviewed by Herald Examiner staff writer Don Branning.

Question: What is Marfan syndrome?

Schwartz: It is a hereditary condition that affects the connective tissues of the body — principally, the skeletal system, the visual apparatus and the cardiovascular system. People with this condition are usually extremely tall and thin, with narrow shoulders and wide hips. Sometimes there is some spinal curvature or some deviation of the chest, because the ribs become elongated and have to go in one direction or the other.

President Lincoln was described by his law partner as having a sunken chest, which is one of the confirming pieces of evidence.

Q: What other signs of the disease did he seem to display?

A: All the external degrees. He had an extreme height of 6 foot 4 inches, he was lean, his arms were extremely long — he bragged about his long arms. His hands have been casted and are in the Smithsonian, and they have been independently evaluated by anthropologists — long before I became interested — and were found to be unusually long for a man of even his height.

Q: And this elongation in his body affected his aorta?

A: Yes. This disease also affects the connective tissue of the main blood vessel to the heart, which will tend to become elongated and stretch. It also stretches the aortic valve, which after a certain point can no longer function adequately — so that eventually the patient will go into heart failure.

Q: You've derived your theory from the literature on Lincoln?

A: Well, everybody recognized that in his last few months in office he was declining, though they weren't aware of why. And this was in spite of the fact that everything was going in his favor — the war was over and he had been re-elected. He was quite happy, as his wife told it.

Q: How was he declining?

A: Well, he himself said in his last few weeks of life, when talking to a friend, "I can no longer do what I used to do. At the end of a day, I am exhausted." A prime finding in the medical cardiac literature is that when an individual is going into congestive heart failure, fatigue may be his first complaint.

Another significant complaint is that the extremities become pale and cold, which is considered an ominous sign, particularly in aortic insufficiency. Lincoln also said a few weeks before he was assassinated, "Of late, my hands and legs are always cold. I should perhaps be in bed."

Q: All this was the effect of the elongation that characterizes the disease?

A: Well, the disease was making his aortic valve — the main valve that leads out of the heart — incompetent.

Now, the evidence that he was having this type of a heart difficulty with that particular valve comes from some remarks he made two years previously. He was look-

Q&A/A-7, Col. 1

Associated Press

Four score and 93 years ago ...

Today marks the 173rd birthday of Abraham Lincoln, and while Abe couldn't make it for a CBS-TV photo session, Academy Award-winning actor Gregory Peck did. Peck is portraying the nation's 16th president in the upcoming eight-hour CBS miniseries "The Blue and the Gray."

Q&A

Continued from page A-2

ing at a photograph that had just been taken of him and his legs were crossed, and he said, "Look at this picture. My foot that is crossed over the other foot looks blurred, and I'm certain I did not move the leg or the foot."

Q: What does that indicate?

A: A journalist Lincoln was with looked at it and said, "Well, maybe it was the blood vessels in the extremity that caused the foot to move." And so the president sat down and crossed his legs and observed his foot and said, "That's it. Isn't that curious." His foot was moving.

When I saw that, I recognized that this meant, according to the medical cardiac literature, he had abnormally bounding pulses — which is one of the hallmarks of aortic insufficiency of that type of a valve defect. It is called a "vascular dance," coincident with the beating of the heart.

Q: And Marfan syndrome is hereditary?

A: It will develop on a statistical basis in 50 percent of the children of anyone who has this. It occurs in all races and all sexes. The degree of the disease is variable — it may be very minor and never cause any difficulty.

Q: Did Lincoln pass this on to his children?

A: Three of his children undoubtedly died of this at ages 4, 12 and 18. His oldest son, who survived into the 80s, had the same squint as his father — which is a characteristic of the condition. Also, Thomas Lincoln, the father of the president, has been documented as having eye difficulty.

Q: What were the children's symptoms?

A: The symptoms were all I've described, highly consistent with congestive heart failure. They didn't know the cause of it.

Q: How did you first come on to this theory?

A: In my early years of practice, I saw a case of this. A young boy was brought in to me by his mother for consultation after he had a hernia operation — hernias go very frequently with this.

Later, his grandmother came in and asked how he was doing, and that's when I made the association that Lincoln must have had this — because her name was Lincoln. I had a mental image of the president being tall, lean and sort of different.

I just was convinced that all I had to do was find some additional evidence. I asked her if she was related to the president's family, and she said her husband was. And we worked it out and confirmed that her husband was the descendant of a common ancestor of the president who was born in 1686.

Q: Have you found many members of the Lincoln family who suffered from this?

A: Yes, many families. My very first paper shows many Lincolns who have these same skeletal characteristics. It's a very large family.

Q: Are there direct descendants of Lincoln living today?

A: Only one descendant of Lincoln is still alive — a great, great grandson. He's an elderly man, a very ill man.

The little boy I examined was a *collateral* kin of Lincoln, which means that they both received the gene from one common ancestor.

Q: What were the little boy's symptoms?

A: He was too tall for his age, he had a peculiar chest, he had extremely long fingers, he had the hernia.

Q: Where is that boy today?

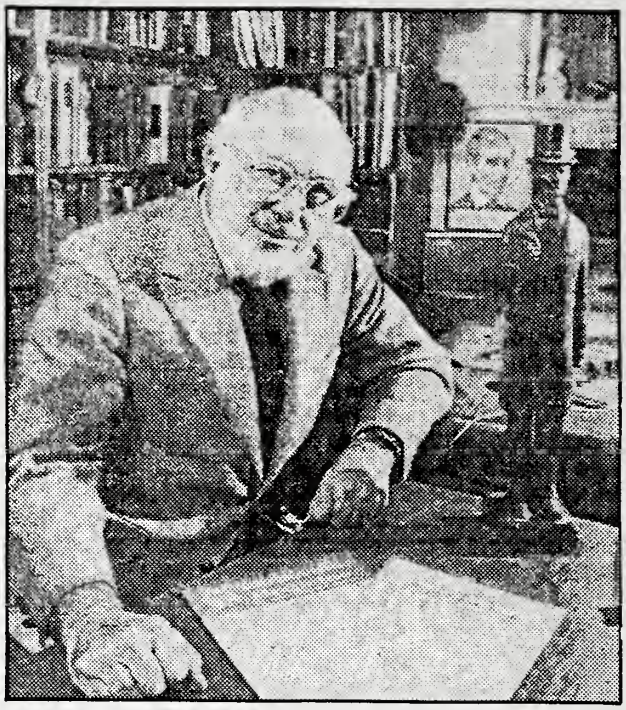
A: He's doing well. He's out in the world working.

Q: How common is this Marfan syndrome?

A: Well, when I got involved it was considered extremely rare. And the question always came up if I wasn't overdiagnosing the condition in the many people I have seen it. In recent years, it's been well-recognized that the disease is a common condition — but I still maintain that it is much more common than the medical public realizes.

Q: What sort of help is there for Marfan syndrome victims?

A: There are ways of reducing the strain upon the heart and the main blood vessel. There is also help for the eye problems that come up occasionally. And if the condition is far advanced or there are complications, there is very effective surgery now.



"Everybody recognized that in his last few months in office (Lincoln) was declining ... in spite of the fact everything was going in his favor — the war was over and he had been re-elected."

Dr. Harold Schwartz
Professor, USC School of Medicine

Peoria newspaper

ELONGATED FRAME

Physician Says Lincoln Had Genetic Disease

LOS ANGELES (AP) — Abraham Lincoln's familiar image — the commanding height, lanky frame, great hands and gaunt face of so many faded photographs — was caused by a hereditary disease that trails his descendants to this day, says a physician at the University of Southern California.

Dr. Harold Schwartz said the lifelong effects of Marfan's syndrome were reaching a climax in the 56-year-old president in 1865 and would probably have killed him within a year had not an assassin struck first.

The genetic disorder, which Schwartz says produces "a disproportionate elongation of the skeletal system," can also change the aorta, the main blood vessel from the heart, cause its valve to malfunction and, ultimately, result in progressive heart failure.

Based on Lincoln's words and those written about him, Schwartz concluded: "He was already in heart failure at the time he was assassinated. He could not have lived more than 6 to 12 months."

Schwartz, of the USC medical school, has been studying Lincoln and Marfan's syndrome for three decades, ever since he examined a 7-year-old boy whose mother was concerned that "he was so disproportionately tall."

The boy was a classic example of the genetic abnormality and "the family's name was Lincoln. I knew immediately that Abraham Lincoln had the same thing."

Since then, Schwartz said in an interview, he has traced the Lincoln family tree, confirming his young patient's kinship with the 16th president, and studied newspapers, books, pictures and letters concerning Lincoln. The study in Schwartz's suburban Lakewood home is a small museum of Lincoln memorabilia.

"I found a whole world of confirmation ... It's not hypothetical at all," he said. "I have merely put together the facts that have been given ... by the president and his contemporaries and the facts in the medical literature."

Schwartz said he followed the Lincoln roots back to England and found current branches around the United States, where "the Lincolns are all over the country."

The Associated Press

Dr. Harold Schwartz

Marfan's syndrome, which varies widely in severity, has followed the Lincolns. It affects, by one estimate, at least five of every 100,000 Americans of all sexes and races, Schwartz said. "That figure should probably be multiplied by 5 to 10," he added, "and even that may be conservative."

The syndrome, caused by a dominant gene that affects men and women equally, was named for French pediatrician Bernard-Jean Marfan, who first described it in 1896. Schwartz said those affected, in extreme cases, "are usually very tall and lean. Their arms are long and the fingers get very long and thin. The lower part of the body gets very long."

The face is gaunt and angular, with large ears and nose. The overall appearance is one gawkiness because of the long, loose-join limbs.

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Lincoln

▲ *from page 1*

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Victims of Marfan syndrome "ar usually very tall and lean. Their arms are long and the fingers get very long and thin. The lower part of the body gets very long."

Doctor: Genetic disease made Lincoln lanky

by Robert Locke
The Associated Press

LOS ANGELES — Abraham Lincoln's familiar image — the commanding height, lanky frame, great hands and gaunt face of so many faded photographs — was caused by a hereditary disease that trails his relatives to this day, says a physician at the University of Southern California.

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Based on Lincoln's words and those written about him, Schwartz concluded: "He was already in heart failure at the time he was assassinated. He could not have lived more than six to 12 months."

Schwartz, of the USC medical school, has been studying Lincoln and Marfan syndrome for three decades, ever since he examined a 7-year-old boy whose mother was concerned that "he was so disproportionately tall."

The boy was a classic example of the genetic abnormality and "the family's name was Lincoln. I knew immediately that Abraham Lincoln had the same thing."

Since then, Schwartz said in an interview, he has traced the Lincoln family tree, confirming his young patient's kinship with the 16th president, and studied newspapers, books, pictures and letters concerning Lincoln. The study of his suburban Lakewood home is a small museum of Lincoln memorabilia.

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The scientific term for the syndrome is arachnodactyly, derived from the Latin word for spider.

Schwartz noted that at least one Lincoln contemporary said the president had "spiderlike legs" when seated. Lincoln himself commented on the size of his feet and the length of his arms. That's confirmed by the few measurements available of the 6-foot-4 president.

Schwartz said Lincoln's loose posture is also characteristic of the syndrome, as is evidence of severe farsightedness and periodic squinting.

That Lincoln's heart was failing as a complication of the inherited problem, Schwartz said, is strongly suggested by this Lincoln statement shortly before his death: "I am very unwell now; my feet and hands of late seem to be always cold, and I ought perhaps to be in bed."

Cold extremities are a common result of a heart no longer able to pump

△ *continued on page 2*

Marfan syndrome' named

Lincoln suffered heart illness

ST PAUL, Minn. (UPI) Abraham Lincoln apparently was dying of a hereditary type of heart disease called Marfan syndrome when he was assassinated in April 1865, a physician said Tuesday.

Dr. Harold Schwartz of Los Angeles, an internal medicine specialist, said in an interview that Lincoln had "quite a decline in the last six weeks of his life."

Before the assassination, Lincoln's illness was blamed on emotional stress, Schwartz said.

"Everybody wrote about it and his wife was much concerned," he said. "This was a time when psychologically he should have been getting better. The Civil War was virtually over and, he was elated about that.

"But he probably had only about a year to live if he had not been assassinated."

He said the syndrome first was discovered in 1896 and was considered "extremely rare." Actually, he said, it is very common but not often recognized because most victims don't have serious complications.

Schwartz said the disease is characterized by elongated tissues. Victims usually are tall and thin, with sunken chests, abnormally long fingers and arms.

Dr. Schwartz said he found signs of the disease in tracing Lincoln's ancestry.

The doctor became interested in the disease 20 years ago when he saw a boy with the ailment. He said he traced the boy's genealogy and found he and Lincoln had a common ancestor, the great-great-grandparent of the president.

Schwartz said Marfan elongates tissues in blood vessels among other things, which can lead to the death of tissue and loss of elastic properties of blood vessels.

The doctor said he believes Marfan led to improper function of a heart valve in Lincoln, and an early stage of congestive heart failure was evident in the president not long before his death.

Lincoln disorder still afflicts heirs, doctor says

By Robert Locke
Associated Press

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That Lincoln's heart was failing as a complication of the inherited problem, Schwartz said, is strongly suggested by this Lincoln statement shortly before his death: "I am very unwell now; my feet and hands of late seem to be always cold, and I ought perhaps to be in bed."

Chicago Sun-Times 2-12-81

Friday, February 12, 1982 Fort Wayne, Indiana 46802

Lincoln dying of heart disease when

See related article Page 1D

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— Abraham Lincoln

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Gazette

25 cents

killed, physician says

THE CHARLOTTE OBSERVER Sunday, Feb. 14, 1982 29A

Lincoln's Looks Due To Illness

Heart Was Failing Before Assassination

By ROBERT LOCKE
Associated Press

LOS ANGELES — Abraham Lincoln's commanding height, lanky frame, great hands and gaunt face were caused by a hereditary disease that trails his descendants to this day, according to a physician at the University of Southern California.

Lincoln

Dr. Harold Schwartz says the lifelong effects of Marfan syndrome probably would have killed the 56-year-old president by 1866 had not an assassin struck first in 1865.

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Schwartz has been studying the 6-foot-4 president and Marfan syndrome for 30 years, ever since he examined a 7-year-old victim whose name was Lincoln.

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Schwartz says there is treatment for the life-threatening complications of the disease, such as heart problems. But Marfan syndrome, like almost all genetic disorders, cannot be cured.

Newsday MARCH 26, '84

your health

Marfan's: A sudden death syndrome

By Michael Unger

His name was Stephen Ciccariello, and he was an architecture student at Washington University in St. Louis. At 6 feet, 5 inches, with a rugged face and a long, gangling build, he looked a little like Abraham Lincoln. In 1969, when he was only 22 years old, he died a few days after running in a college race.

It was only after her son's shockingly premature death that his mother, Priscilla Ciccariello, learned that Stephen, her husband, Gerard, and two of the Port Washington couple's six other sons had Marfan's syndrome, a hereditary degenerative disease of the connective tissue that is one of the causes of seemingly inexplicable deaths among young athletes.

Marfan's victims are noted for their excessively long bones, arm spans that may exceed their height, protruding or sunken chests, rugged features, curvature of the legs and spine, and distortion of the lens of the eye. Patients in the acute stages of the syndrome are subject to sudden death from a ruptured aorta, the main artery feeding oxygen-enriched blood from the heart to the body. A genetic defect of the body's connective tissue causes the artery, and often the aortic and mitral heart valves as well, to stretch and leak, or suddenly burst.

Until recently, Marfan's syndrome could not always be detected before the appearance of severe complications. Then surgery on the aorta and the valve was rarely successful, and patients most often died after living less than two-thirds of their normal lifespans. Priscilla Ciccariello's husband died at age 59 of Marfan's despite five operations.

Today, however, new surgical techniques and early diagnosis and treatment are saving the lives of many Marfan's syndrome patients. New drugs are being used to delay or prevent the need for surgery. And studies show a greater incidence than had been known of the syndrome that some reputable scientists and historians believe Abraham Lincoln might have had.

Lincoln's craggy features and his lanky, 6-foot-4 frame were often ridiculed mercilessly, and detractors in newspapers of the day referred to him with such epithets as "ape," "hideous baboon" and the "Illinois Beast." Even cartoonists friendly to the 16th president pictured him as an ungainly giant with long, swinging arms and spindly legs.

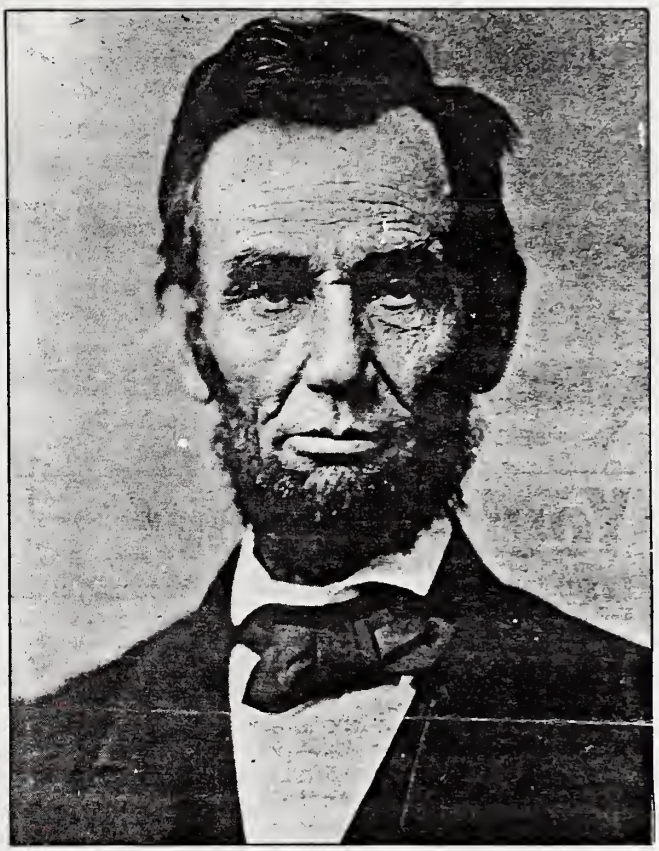
Lincoln had stated not long before his assassination at age 56 that he was not well, had cold hands and feet, and perhaps ought to be in bed. This, along with his poor eyesight and his build, has led to speculation that Lincoln may have had Marfan's.

But it wasn't until 1896, 31 years after Lincoln's assassination, that French pediatrician Antonin Marfan first described the syndrome that bears his name. One of the leading experts on Marfan's, Dr. Reed E. Pyeritz, a medical geneticist and internist at Johns Hopkins University Medical School, was called as an expert witness not long ago by the Organization of American Historians to comment on a formal presentation by the large academic group on Lincoln and Marfan's syndrome. "I happen to agree with the historian who presented the paper that we'll never know for sure," said Pyeritz. "There certainly are things to support the contention, and there are things not so typical. But there is no direct evidence and no full autopsy was done."

In Stephen Ciccariello's case, the weakened aorta suddenly burst. "Stephen went to the college infirmary on the weekend feeling nauseous and experiencing chest pains. When the doctor came in on Monday, he was sent to a hospital. But the aorta ruptured on the table," said his mother. "At the time, the surgeon who operated was shocked that we had never been told about Marfan's syndrome by our pediatrician, our medical doctor, or the heart specialist who operated on my husband in 1961 for what they described as a congenital defect."

Gerard Ciccariello had five operations at hospitals on Long Island and in Texas before he died of complications of Marfan's syndrome, his wife said. He had been in the Army Air Corps in England during World War II and was 55 years old before he even knew the name of the condition that would kill him four years later in 1974, said Mrs. Ciccariello. "At 6-foot-1, he had been the tallest member of his family until Steve grew taller," she said.

"There's a Marfan look," she said. "Every place there's connective tissue, they're that much longer — chin, nose, ears, fingers, toes. Peter is 6-foot-5," she said, "and John is 6-foot-2. The eyes are quite almond-shaped. My oldest surviving son, Peter, who is 34, has two children with the condition, a 9-year-old boy and a girl who is 4. Peter, who was two years younger than Steve, has had two open-heart surgery operations, the first in 1974, and he had to have a valve transplant in 1981. My youngest son, John, is 24. He's fortunate. He hasn't needed surgery." John and Peter, who are commercial artists on the South Fork, both take drugs intended to reduce the work load on their hearts.



This photo of Lincoln, taken late in his life, shows the long ears and large nose that are often seen in people with Marfan's syndrome.

Their mother became active in, and is now chairman of, the National Marfan Foundation, located in Port Washington, where she lives and works as a reference librarian. Mrs. Ciccariello says other families with Marfan's syndrome have had experiences similar to hers. While the disease can be fatal, it varies widely in severity, symptoms and age of onset. The official statistics are sketchy, with the National Center for Health Statistics reporting 251 Marfan's deaths in 1980 and at least 9,000 hospitalizations of children with Marfan's in 1977.

But Marfan's expert Pyeritz of Johns Hopkins, who has a doctorate in molecu-

—*Continued on Page 9*

Treating Marfan's patients with life-saving new surgery

—Continued from Page 7

lar biology in addition to an M.D. degree, said that the incidence of the disease is approximately one in every 20,000 people — without regard to sex, ethnic and racial background, or geography. A large proportion of the country's Marfan's patients are treated at the Moore Clinic at Johns Hopkins, where Pyeritz, Victor A. McKusick, one of the world's leading medical geneticists, and their colleagues diagnose and treat Marfan's. "It's clear that previous estimates of one in 100,000 are too low," Pyeritz said, adding that even one in 20,000 may be conservative.

Marfan's syndrome is inherited as a dominant trait caused by an abnormal gene. "If one parent is affected, then the chance of any offspring having the gene and the condition are 50-50," said Pyeritz. "We have never seen an example of having the gene but not expressing it. There are no skipped generations. We don't have any examples of both parents having Marfan's syndrome," he said. But there are cases, he said, where a parent without the syndrome has had a spontaneous change or mutation in his sperm or her egg and has produced a child with the syndrome. But this happens far less frequently, he added.

"There's a great deal of variability [of symptoms] even within a family," said Pyeritz. But sooner or later, most people with Marfan's will develop a problem with their aorta; the age that happens will vary. It does not necessarily progress rapidly in all patients, so that the age range on whom we've done surgery has been quite wide. The youngest, I think, was 14, and the oldest is 56." But there are infants who also need the surgery, the physician added.

Most researchers, Pyeritz continued, believe that the Marfan gene produces a change in one of the proteins that provide strength to connective tissue, a protein such as collagen. Connective tissue is the support tissue of the body. "Connective tissue is both the scaffolding and the glue that holds all of our organs together," he said. Bone, ligaments, tendons and cartilage are pure connective tissue, Pyeritz said, "but, in addition, connective tissue permeates all of the organs and provides the strength and elasticity of blood vessels, in particular the aorta."

The primary Marfan's problem and the one that is most often fatal, Pyeritz said, is the progressive enlargement of the first 2 to 4 inches of the ascending aorta closest to the heart, and the aortic valve. "The tissue is stretching like a balloon being blown up," the physician said, "the artery wall is getting thinner at the same time, and the stress on the aorta increases. The big danger is tearing or rupture. And when the aorta gets big enough, the aortic valve is no longer sufficient to cover the opening, and it leaks. Blood that's been pumped out leaks back into the heart."

Through the mid-1970s, Pyeritz said, surgeons would either replace or put a tuck in the part of the aorta that had enlarged, or, often in a separate operation, replace the aortic valve. "The rate of people dying on the table was 30 per cent, and the long-term success was very poor," the doctor said.

Now, however, surgeons at Johns Hopkins are using a British procedure that involves replacing the enlarged section of the aorta and aortic valve with a composite graft of Dacron tubing and a metal valve.

"This is preventive surgery," Pyeritz said. "We've done over 40 of these, and we've had no deaths at all. I think it's pretty clear that the majority of the people who have had the surgery here would have died within a year or two if they hadn't had the surgery." Pyeritz said that in many cases the new surgery will do away with the need for multiple heart operations. People with Marfan's syndrome "need to be followed carefully to insure that they don't get to a point where surgery might be particularly hazardous, or that they just die of a ruptured aorta without any warning," he said.

Unless there are significant complications, the disease is painless. Other large hospital centers that have much experience with this surgical procedure for Marfan's syndrome are the Stanford Medical Center and Cedars-Sinai Medical Center, both in California and the Texas Heart Institute and Baylor College of Medicine, both in Houston. In the New York area, New York Hospital-Cornell Medical Center has the most experience with diagnosis and treatment of Marfan's, Pyeritz said.

Peter Ciccariello said that current Marfan's research "kind of gives you a lot of hope for the future, especially when you have children with the condition. It came too late for Steve and my father, and I'm on the top of the crest, but hopefully by the time my children are older they'll have found better ways to deal with these problems. I would like to emphasize, though, that there's a lot more for a person with this condition to be hopeful about now. It's certainly possible to live relatively normal and fulfilling lives. What's most important for a person with this condition is to seek out a medical center that knows how to treat these related problems."

For more information, write to the National Marfan Foundation, 54 Irma Ave., Port Washington, N.Y. 11050, or call 883-8712. /II

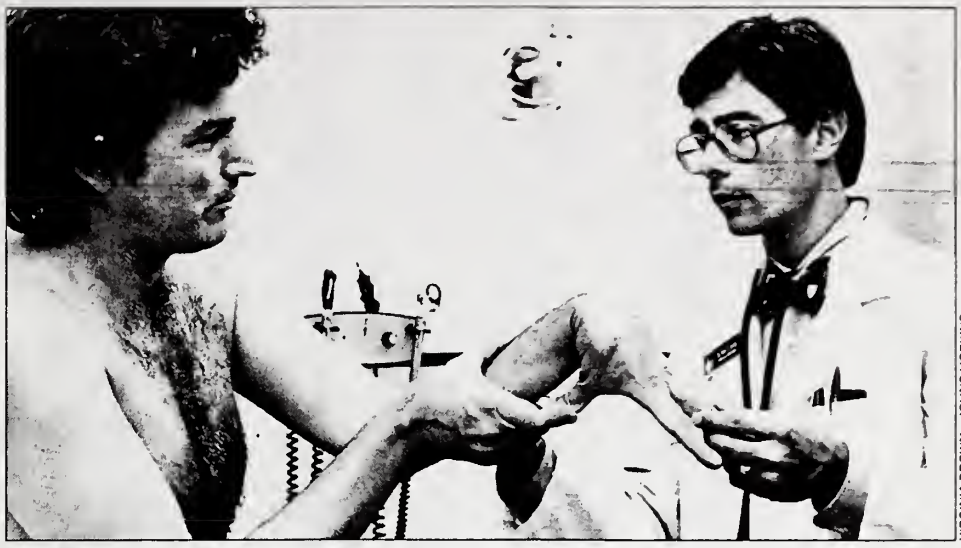
somebody to wail at or to giggle with."

Starting life in 1944, with tickets pegged to $2.50 top (against today's $35), the City Opera has always had to cope with the image of a "poor man's Met." Yet the differences between the companies cut far deeper than the similarities. While Met officials freely own up to a conception of their company as a museum for the masterpieces of the past, both Sills and music director Keene shrug off any such suggestion about their own company, and rightly so. From its beginning, to cite one major difference, the City Opera has steadfastly refused to honor the notion of a split between serious grand opera and musical drama created for commercial theater. Stephen Sondheim's "Sweeney Todd," a work of intimidating seriousness and substance, assumes its rightful place this fall in the City Opera repertory. Previous Broadway shows, notably Leonard Bernstein's "Candide" and Kurt Weill's "Street Scene," enjoyed far greater success in their City Opera reincarnations.

Spellbinder: Despite the obvious differences between the companies, the City Opera's arrival at Lincoln Center in 1966 was viewed with some hostility by the Met's then general manager, Rudolf Bing. For years the Met frowned on sharing singers with its next-door neighbor. Now that antagonism has vanished. Faced with the current worldwide shortage of operatic glamour pusses, the Met and the City Opera seem all too delighted to pool resources. The career of superstar bass Samuel Ramey is a case in point. One of the countless young Americans nurtured at the City Opera and then sent on to worldwide fame—at La Scala, in Ramey's case, and in a stunning debut last season at the Met—Ramey still maintains his City Opera ties, in the archetypal spellbinder Boito's "Mefistofele" this season, and in a new production created for him of Massenet's "Don Quichotte" in the future.

Never a believer in tradition for its own sake, Sills speaks excitedly of the insertion of simultaneous English translations on a screen above the stage, a recent innovation that she sees as a way to break down the communication barrier between opera and audiences. She has also proven herself at least moderately receptive to stage directors with a less orthodox—if not downright radical—approach to staging a traditional opera, as with Frank Corsaro's recent modern-dress "Carmen," or Andrei Serban's mirrored walls and other disco trappings in last year's company première of Handel's "Alcina." "All right," says Sills, ever so lightly on the defensive, "so we had alligators crawling up the walls in some of the scenes. But we also had Carol Vaness in some of the best Handelian singing I've heard in years. It wasn't Serban who was the star that night; it wasn't even Vaness or the rest of the cast; it was the music. So long as it doesn't get to the point where you don't remember whose opera you're listening to, I'm willing to experiment."

ALAN RICH



VIRGINIA BROWN—JOHNS HOPKINS

Symptom: Ciccariello demonstrates his loose-jointedness for Hopkins's Pyeritz

The Lincoln Syndrome

Abraham Lincoln's distinctive long narrow face, deep-set eyes, sunken chest and lanky limbs were irresistible to the political cartoonists of his era, who easily transformed them into comical caricatures. But what may have caused Lincoln's unusual appearance is no laughing matter. Many physicians today believe he was a victim of Marfan syndrome, an inherited disorder of connective tissue that affects about 20,000 Americans—making it as common as hemophilia and more prevalent than cystic fibrosis.

Until just a few years ago, a diagnosis of Marfan syndrome usually meant an early death: many victims died in their 20s and 30s. But at a recent meeting of Marfan researchers, physicians from Johns Hopkins University School of Medicine reported evidence that early diagnosis and aggressive treatment with drugs and surgery can improve the quality and extend the length of many Marfan patients' lives.

Because connective tissue supports structures and organs throughout the body, Marfan syndrome affects several biological systems, with widely varying severity. Skeletal abnormalities—including loose-jointedness and elongated limbs—are common, as are eye problems. The most life-threatening impact of Marfan syndrome, however, is on the cardiovascular system. The disease may damage the aortic valve, for example, allowing blood to leak back into the heart. But what kills many victims is an aneurysm, a dangerous ballooning out of the weakened aortic wall produced by the force of the heart's pumping action. An aneurysm can rupture without warning, often as a result of physical stress such as athletic activity or lifting heavy objects; the patient usually dies within a few hours.

Until the mid-1970s, doctors seldom operated on Marfan patients until aortic damage was extensive. But now physicians at Johns Hopkins and elsewhere believe that surgery should be performed as soon as the aorta has enlarged to a diameter of six centimeters—twice the normal size. "We really think these folks, if they do not have surgery, are disasters waiting to happen," says Dr. Reed Pyeritz, director of the medical-genetics clinic at Johns Hopkins.

Success: In surgery, the stretched portion of the aorta and its damaged valve are replaced with a Dacron tube attached to a mechanical valve. Of 47 Marfan patients who have undergone this procedure at Johns Hopkins since 1976, 41 are still alive; the survival rate is highest among those who had the procedure on an elective basis, before symptoms developed.

Another promising preventive therapy involves the class of drugs called beta blockers, which reduce the strength and frequency of heart contractions. Over a seven-year period, reports Pyeritz, a group of newly diagnosed Marfan patients who were given beta blockers experienced less aortic enlargement than a control group who got a placebo. "The sooner you start using a beta blocker, the better," he declares.

One beneficiary of the new research is Peter Ciccariello, 35, a graphic designer who lives in East Hampton, N.Y. Ciccariello is a Marfan victim whose father and older brother died of the disease; another brother is also afflicted. After two operations on his aorta, Ciccariello now takes the beta blocker propranolol and must undergo periodic monitoring of the condition of his aorta. Still, he remains optimistic. "I think about dying a lot," he admits, "but my father had archaic surgery. There's a lot more to be positive about now than before."

JEAN SELIGMANN with
DEBORAH WITHERSPOON

Contents

The Courier-Journal MAGAZINE
Louisville, Kentucky 40202
February 9, 1986

NEXT WEEK:

The down-to-earth Sky Walker

James S. Pope Jr., Editor
Stephen Lee, Associate Editor
William Norton, Associate Editor
Stephen Sebree, Art Director

The LINCOLN-DIAGNOSIS *Debate*

Left, Lincoln at age 51, during his 1860 presidential campaign. Above, an older and wearier-looking Lincoln in 1865, shortly before he was assassinated.

BY ELINOR J. BRECHER

DID A RARE GENETIC MALADY MAKE THE GREAT EMANCIPATOR INTO THE GREAT, GANGLING MAN THAT HE WAS?

Pam Spaulding

Louisville's Dr. Abraham Gordon, above, who first speculated that Abraham Lincoln had Marfan's syndrome, believes that Lincoln may have inherited the gene responsible for the condition through his mother, Nancy Hanks. No picture of Lincoln's mother exists, but the portrait above right is based on descriptions of her appearance

"I have before me a photograph of him taken at Pittsfield, Ill., during the campaign of 1858. It looks as I have seen him a hundred times, his lantern jaws and large mouth and solid nose firmly set, his sunken eyes looking at nothing yet not unexpressive, his wrinkled and retreating forehead cut off by a mass of tousled hair, with a shadow of melancholy drawn like a veil over his whole face."

— Horace White, a long-time friend of Abraham Lincoln

"Mr. Lincoln stands six feet twelve in his socks, which he changes every 10 years. His anatomy is composed mostly of bones, and when walking he resembles the offspring of a happy marriage between a derrick and a windmill. . . . His head is shaped something like a ruta-bago and his complexion is like that of a Saratoga trunk. His hands and feet are plenty large enough, and in society he has the air of having too many of them."

— A satirical pamphlet from the 1864 presidential campaign

"He was tall and thin, with enormously long loose arms and big hands, and long legs ending with such feet as I never saw before; one of his shoes might have served Commodore Nutt as a boat. . . . He had very large ears standing off a little, and when he was in good humor I always expected him to flap with them like a good natured elephant."

— Princess Salm-Salm, a Washington visitor during the 1860s

Sad. Brooding. Melancholy. Spidery. Shambling. Cadaverous. These are some of the adjectives that history has attached to the 16th president of the United States, who for four years carried on his bony shoulders the crushing burdens of a nation at war. It is often said that Abraham Lincoln seemed to sag under the emotional weight, his long, angular face shrouded in clouds of gloom and anguish.

But almost four decades ago, a Louisville physician and history buff found himself drawn inexorably toward a startling conclusion about the true nature of Lincoln's mournful demeanor, gangling 6-foot-4 frame and

ELINOR J. BRECHER is on the Magazine staff

what the doctor later would call "unique traits of mind and spirit."

In 1947, shortly after he returned from World War II service as an Army physician in Europe, Dr. Abraham Gordon was treating a patient who suffered from Marfan's syndrome, an inherited disorder of the connective tissues that affects bones, muscles, eyes and the cardiovascular system. It also has been linked to high intelligence and sharp wit.

At the same time Gordon, an internist, was reading a biography of the Kentucky-born president, and he realized that what he'd seen in the examining room bore inescapable similarities to the man called not only Honest Abe, Uncle Abe and Old Abe but also, by political satirists of the day, Long Abe.

"It clicked," said Gordon, 71. "I was concerned with what made Lincoln Lincoln." Reading Lincoln's own words — his inspiring

Continued

speeches and clever asides — he wondered, "Is that Lincoln speaking or the Marfan syndrome? I think that his mind and his uniqueness are related to the Marfan syndrome."

And so he launched a 14-year odyssey through history books and medical journals. His research culminated in a presentation before the Kentucky chapter of the American College of Physicians in September 1961, the centennial year of the Civil War. Published six months later in the Journal of the Kentucky State Medical Association, his work "shocked the medical and historical establishments," wrote Civil War author Harold Holzer in the February 1983 issue of MD, a monthly magazine for medical professionals.

Gordon, a past chief of Jewish Hospital's medical staff, received considerable national attention. His findings generated a flood of contacts from purported Lincoln descendants, including a family of Kentuckians claiming descent from Lincoln's maternal grandmother. It also provoked or prolonged controversies that refuse to fade. Having tampered with the image of a historical giant — indeed, having cast the stigma of disease on one of the most revered Americans of all time (not to mention publicly embracing a theory that Lincoln's mother was illegitimate) — Gordon often has found himself on the receiving end of outrage.

Two weeks after Newsweek magazine published an interview with him in the June 11, 1962, issue, a reader replied, "The shocking description of our beloved Abraham Lincoln, buttressed by a medical diagnosis made *97 years after his death*, does little to damage the image of the great emancipator. . . . Marfan's syndrome my eye! Lincoln's understanding, compassion, humanity and leadership — are these the harvest of a loathesome disease?"

In 1983, a Lincoln scholar and his brother, a pathologist, labeled Gordon's theory, "the newest Lincoln myth." And in an interview last month, a New York physican and Lincoln researcher said that the hypothesis is "simply not true."

But Gordon remains unmoved by attempts to debunk his work. As time has gone on, he said, more people have been accepting it. And although he admits the lack of hard medical data leaves much to conjecture, he continues to stand by his original suppositions. "I made the diagnosis based on a mosaic [of evidence] that forms the outline of Abraham Lincoln," Gordon said. "There are missing pieces. We can't *get* all the facts."

After challenges to his theory in Holzer's article, he said he would welcome something of a verbal duel with his detractors. "My gun is loaded, and I'm ready to fire it," he replied to the magazine. He asked that "judges be appointed and that this issue once and for all be laid to rest." No such action has been taken.

So, says Dr. Reed Pyeritz, director of the medical-genetics clinic at Johns Hopkins University School of Medicine in Baltimore and founder of the National Marfan Foundation, "Every year on February 12, we're going to hear about it." Pyeritz has neither endorsed nor rejected Gordon's findings. "It remains an intriguing possibility. The evidence raised makes for fascinating cocktail-party conversation," he said. Pyeritz said his clinic treats 500-600 Marfan patients a year and that "any number of [them], if you saw them, you'd say, 'Gee, he looks a lot like Abraham Lincoln.' "

Gordon's theory has, in fact, gradually come into general use. A 1972 medical textbook by a respected Johns Hopkins professor, Dr. Victor A. McKusick, cites it at some length. And Newsweek magazine titled an October 1984 article about Pyeritz's clinic, "The Lincoln Syndrome." It said that Lincoln's "distinctive long, narrow face, deep-set eyes, sunken chest and lanky limbs were irresistible to the political cartoonists of his era, who easily transformed them into comical caricatures. But what may have caused Lincoln's unusual appearance is no laughing matter. Many physicians today believe he was a victim of Marfan's syndrome, an inherited disorder . . . that affects [at least] 20,000 Americans, making it as common as hemophilia and more prevalent than cystic fibrosis."

First described by a French pediatrics professor, Antoine Bernard-Jean Marfan, in 1896, 31 years after Lincoln was killed by an assassin's bullet, the syndrome, which shows many variations, manifests itself in elongation of the bones, curvature of the spine, loose joints, asymmetrical facial features, "pigeon breast" or sunken chest and other skeletal abnormalities. Marfan patients are longer from pubis to toes than from pubis to crown. They often have high voices because of high-arched or "gothic" palates, and suffer vision problems, including detached retinas and flat corneas.

But the most threatening feature of the condition is cardiovascular dysfunction. "The usual cause of death is ruptured aortic aneurysm, at an average age of 32 years," according to Consultant, another medical magazine. Some doctors and Lincoln buffs — though not Gordon — believe that had not John Wilkes Booth ended the 56-year-old president's life on April 14, 1865, Marfan's syndrome surely would have not long after. (Pyeritz adds, "If he had not been shot and the data are correct, he likely would have died of it.")

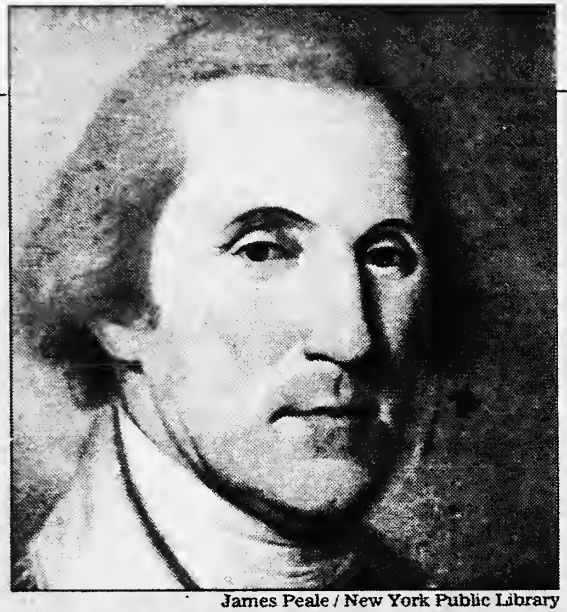
Gordon says photographs and descriptions by casual and close observers can only support his conclusions about the Great Emancipator. In his "Medical Appraisal of Abraham Lincoln," he lists 11 Marfan traits he feels certain that Lincoln — who probably wore size 16½ shoes by today's measures — exhibited. In his paper, Gordon leans heavily on a description by William Herndon, Lincoln's close friend, biographer and law partner, who called Lincoln's expression "woe-struck":

"He was thin, sinewy, raw-boned, thin across the breast to the back, and narrow across the shoulders; standing he leaned forward — was what [may be] called stoop shouldered, inclining to the consumptive build. His usual weight was 180 pounds. . . . His structure was loose and leathery. . . . The whole man, body and mind, worked slowly, as if it needed oiling . . . [his] long arms and giant hands swung down by his side. . . . His legs and arms were abnormally, unnaturally long and in undue proportion to the remainder of his body. It is only when he stood up that he loomed over other men. Mr. Lincoln's head was long and tall from the base of the brain and from the eyebrows. . . . His cheekbones were high, sharp and prominent, his nose large, long, blunt and a little awry toward the right eye. His chin was sharp and curved upward . . . his ears large, and ran down almost at right angles from his head."

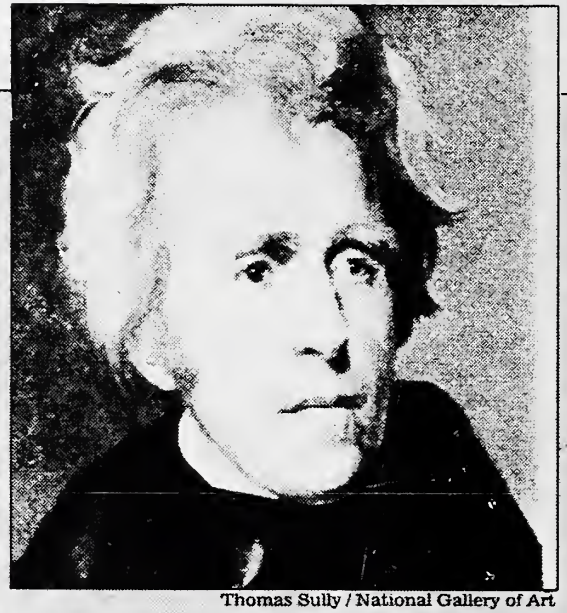
Photographs of Lincoln often are used to illustrate articles about the syndrome, its surgical and drug-based treatments. And whenever such a reference is made, it's sure to stir someone's ire. Priscilla Ciccariello, president of the Long Island-based National Marfan Foundation — widow, mother and grandmother of Marfan sufferers — said that last year, after Newsday published an article about Marfan's illustrated with a picture of Lincoln, her group received some angry mail from a Civil War association that "took great exception to it." The group insisted that Lincoln was a strong, robust man who couldn't possibly have such a crippling, debilitating disease. "That shows how little they know" about a condition that does not always include frailty, said Mrs. Ciccariello, a librarian. Stephen Ciccariello, one of three sons with the condition, died at 22, while running track at college, she said. The other two are living.

(In October, The Washington Post published a story about a University of Maryland basketball star suddenly sidelined by heart problems. The following month, The New York Post ran a similar story about a Temple University player who met the same fate. Both young men, whose fitness programs included weight-lifting and other strenuous activities, were diagnosed as having Marfan's syndrome.)

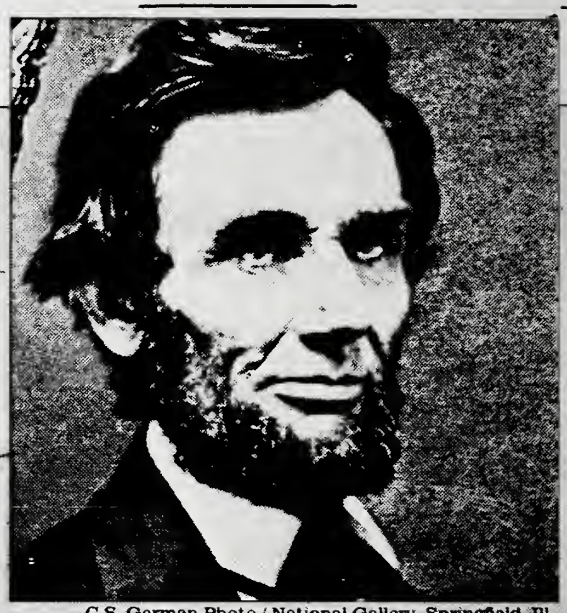
But two years after he published his findings, Gordon received support from a California doctor writing for the Journal of the American Medical Association. Dr. Harold Schwartz not only concurred with his theory, but also expanded it. Gordon and Schwartz, however, disagree on many salient points, and



James Peale / New York Public Library



Thomas Sully / National Gallery of Art



C.S. German Photo / National Gallery, Springfield, Ill.

George Washington suffered from TB and toothache; Andrew Jackson had hives and dysentery; Abraham Lincoln is thought to have had a fatal genetic skeletal disorder.

Our Robust Presidents: Their Image Belied Poor Health

Some of the greatest leaders to occupy the White House have also been the sickest. George Washington, Andrew Jackson and Abraham Lincoln all had a vigorous image. Washington was taller than the average man of his times, and he had the macho general image — as did "Old Hickory." Lincoln, too, was tall and had the image of the strong rail splitter. But . . .

George Washington: He was a hypochondriac, but with good reason. He suffered from tuberculosis, his face was deeply cratered by smallpox. He had a tumor removed from his thigh without anesthesia. Ill-fitting wooden dentures gave him a constant toothache. He also suffered frequent bouts of fevers, chills and pain and was disabled 109 days during his first year in office.

Andrew Jackson: Born a "drooler," he was always spitting and dribbling, and he constantly itched from head to toe with chronic urticaria — hives. His teeth were so bad he couldn't eat solid food, he nearly died from smallpox, and a bout with dysentery left him with cramps and bleeding for the rest of his life. He was shot in the chest in a duel, the bullet lodging in the left lung, causing him to cough up blood and pus until he died at age 78.

Abraham Lincoln: He apparently suffered severe depression, perhaps due to a childhood concussion. With his long, spidery legs and bulging eyes, he is thought by a number of specialists to have had Marfan's syndrome, a fatal genetic disorder that affects the heart, eyes and skeleton. Some medical experts think it's unlikely he would have lived out the year had he not been assassinated on April 14, 1865. —The Washington Post

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MARFAN'S SYNDROME: WAS LINCOLN AFFLICTED WITH THIS FATAL DISEASE?

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Abraham Lincoln and the Marfan Controversy

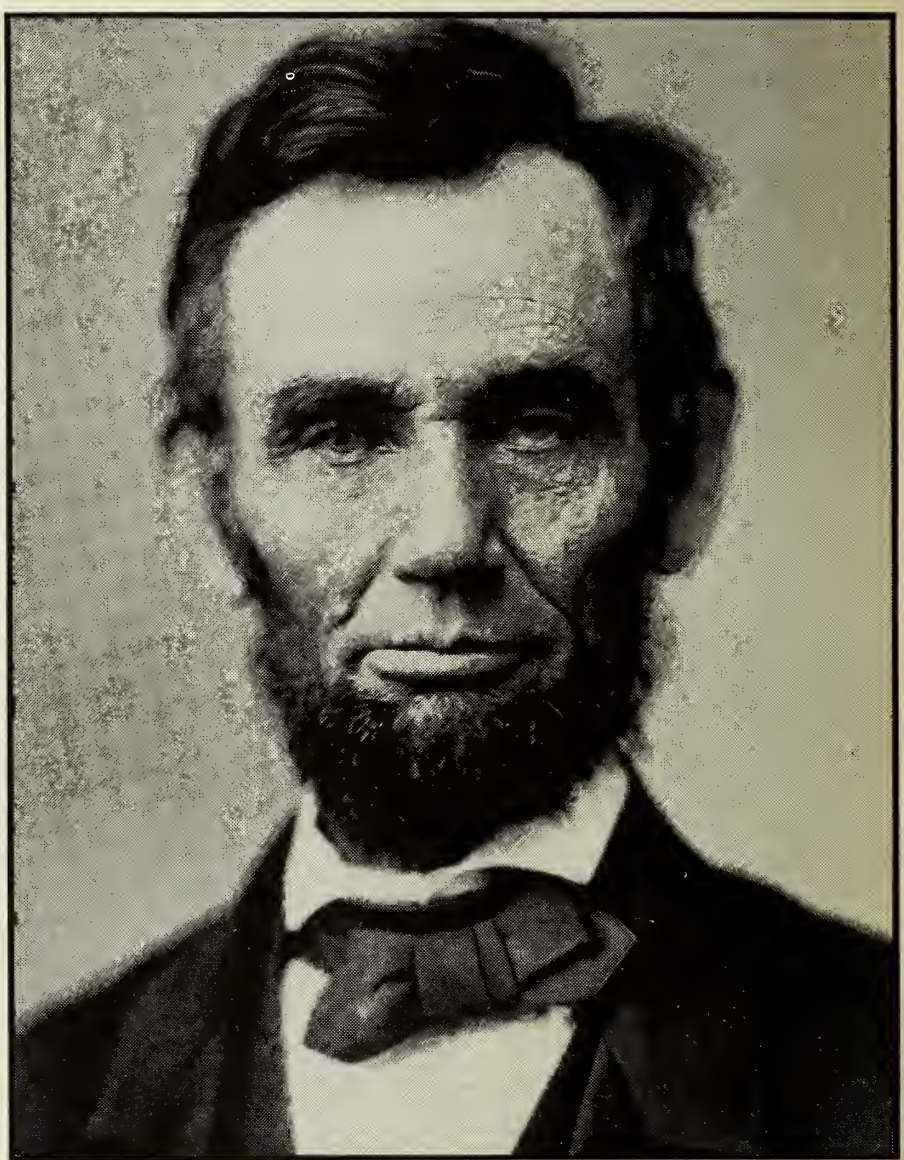
Was our 16th president a sufferer of Marfan's Syndrome? Would he have died prematurely had he not been assassinated? Doctors have been arguing the "case" for 28 years, but still don't agree.

By Alanna Nash

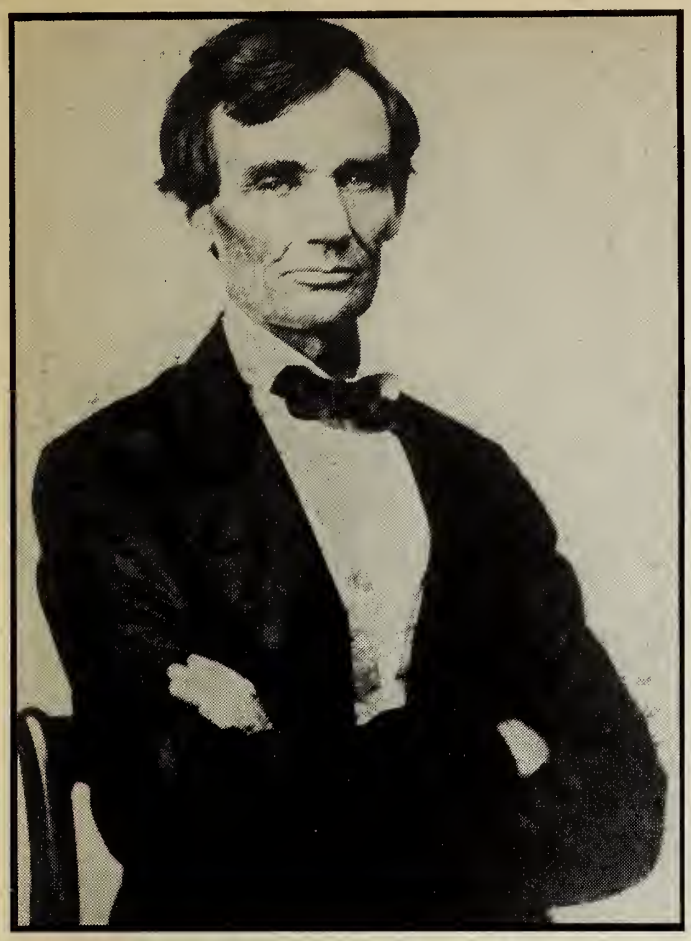
In the late 1940s, shortly after he returned from World War II service as an Army physician in Paris, Dr. Abraham M. Gordon, a Louisville, Kentucky, internist, began treating a patient who exhibited symptoms of Marfan's syndrome, a hereditary degenerative disorder of the connective tissue. While the disorder may result in abnormalities of the eyes, heart, kidneys, and lungs—and some say contribute to superior intelligence and inordinate wit—most physicians first suspect Marfans from their gross skeletal abnormalities and appearances—excessively long bones, spider-like limbs, chest deformities, and a loose-jointed, almost comical gait.

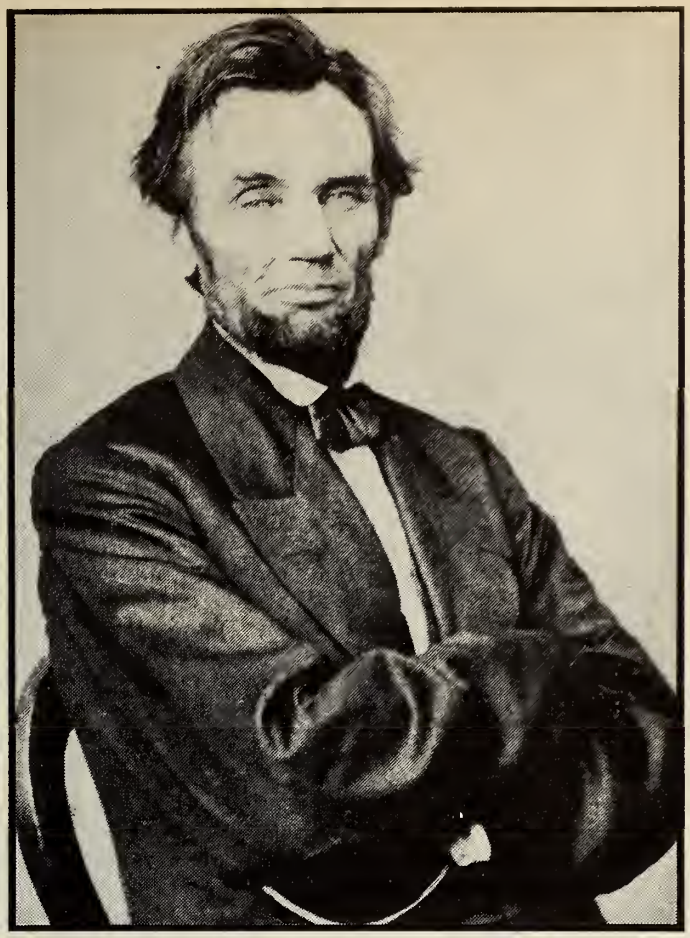
At the same time, Gordon happened to be reading a biography of Abraham Lincoln, the nation's 16th president, whose peculiar physical appearance was described by Lincoln's friend and biographer William Herndon:

> Mr. Lincoln was six feet, four inches high . . . thin, sinewy, raw-boned, thin through the breast to the back, and narrow across the shoulders; standing he leaned forward—was what may be called stoop-shouldered, inclining to the consumptive build. His usual weight was 180 pounds. . . . His structure was loose and leathery . . . . He had dark skin, dark hair and looked woe-struck. . . . When he walked he moved cautiously but firmly; his long arms and giant hands swung down by his side. . . He put his whole foot flat down on



Lincoln's "roving" left eye is fodder for some physicians' belief that the president suffered from Marfan's syndrome, but Dr. John K. Lattimer believes that a mild muscular weakness, and not fragile connective tissue supports, caused the eye to move. *PHOTO BY ALEXANDER GARDNER, COURTESY LLOYD OSTENDORF COLLECTION.*





At 51 (left), Lincoln began his term as president, looking energetic and youthful. *AMBROTYPE BY PRESTON BUTLER.* **Three years later (right), he had aged considerably.** *PHOTOGRAPH BY LEWIS E. WALKER.* **Was it the strain of a country at war with itself and his severe family problems, or the progression of Marfan's, a genetic disorder?** *PHOTOS COURTESY LLOYD OSTENDORF COLLECTION.*

the ground at once, not landing on the heel; he likewise lifted his foot all at once, not rising from the toe. . . . His legs and arms were abnormally, unnaturally long and in undue proportion to the remainder of his body. It was only when he stood up that he loomed over other men. Mr. Lincoln's head was long and tall. . . . His long sallow face was wrinkled and dry . . . his ears large, and ran down almost at right angles from his head; his lower lip was thick, hanging and undercurved, while his chin reached for the lip, upcurved.

"Something just clicked," remembers Gordon, now 75 and still in active medical practice. For the next 14 years, the physician and history buff would study the man to whom he was inexorably drawn—who shared both his given name and his Kentucky upbringing, and who now seemed to harbor a genetic secret behind his sad, brooding eyes.

In the course of that study, Gordon began to see many of the Great Emancipator's famous characteristics in an entirely new light. For nearly a century, historians had written about how Lincoln's angular face sagged with the emotional exhaustion of his years in the White House, a time during which his distinctive visage withered a decade for every year the nation was at war.

"I wanted to find how much of Lincoln was Lincoln," Gordon explains, "and how much was Marfan's syndrome."

But how much of that weariness reflected Lincoln's real anguish over his bloodied homeland and a host of personal problems—the premature deaths of sons Eddie and Willie and his wife's irreversible spiral into madness—and how much was genetic destiny, since a sad, melancholy countenance is now known to be yet another feature of Marfan's syndrome? Had his shoulders become bony from the terrible burdens of the wartime presidency, or were other physical factors to blame? And if he indeed suffered from the syndrome, had it also played a role in determining his intelligence and his unique traits of "mind and spirit," expression and wit?

"I wanted to find how much of Lincoln was Lincoln," Gordon explains, sitting in the study of his suburban Louisville home, "and how much was Marfan's syndrome."

And so in 1961, the centennial of the Civil War, Gordon shocked the medical and historical community alike with a speech before the Kentucky Chapter of

the American College of Physicians, in which he identified Lincoln as perhaps the most famous of Marfan sufferers, citing 11 traits he believed Lincoln exhibited, including a high-pitched voice, "almost girlish in quality." But the following year, when the text of his speech, titled "Abraham Lincoln—A Medical Appraisal," was reprinted in the *Journal of the Kentucky State Medical Association,* Gordon was unprepared for the publicity the assertion would generate both nationally and internationally, and for the hostility and outrage he would encounter.

Shortly after *Newsweek* published an interview with Gordon in the June 11, 1962, issue, Herman Blum, a Lincoln enthusiast and the director of a Lincoln memorial gallery in Philadelphia, wrote an angry letter to the magazine, equating Gordon's theory with an attempt at character assassination. "The shocking description of our beloved Abraham Lincoln, buttressed by a medical diagnosis made *97 years after his death,*" Blum sniffed, "does little damage to the image of the great emancipator. . . . Marfan's syndrome my eye! Lincoln's understanding, compassion, humanity and leadership—are these the harvest of a loathsome disease?"

Twenty-eight years later, the controversy still simmers. A New York urologist and Lincoln scholar, Dr. John K. Lattimer, is perhaps Gordon's greatest critic, saying, "You feel like a dog taking this kudo away from the Marfan's people, because Lincoln is a great hero. But the truth is the truth. I have great respect for Dr. Gordon, but when you find all the evidence and examine it closely, it's obvious that Lincoln couldn't have been a Marfan's."

"Our view is that you don't have to have a strange disease to be great," says Boritt.

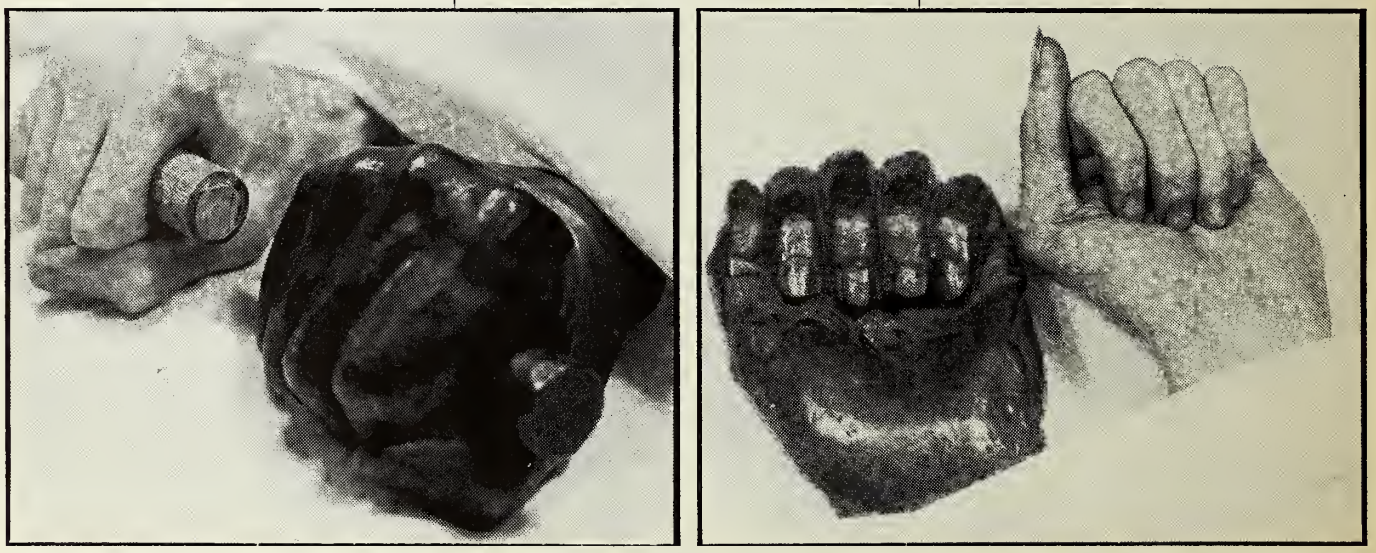
And another Lincoln expert, Gabor S. Boritt, Ph.D., and his brother Adam, a pathologist, called Gordon's label of Marfan's syndrome—a disorder first identified 31 years after Lincoln's death by an obscure French pediatrician, Antoine Bernard-Jean Marfan—"the newest Lincoln myth. . . . Our view is that you don't have to have a strange disease to be great."

If the stigma of disease was enough to rile Lincoln's most passionate researchers, Gordon was soon to incur further wrath when he stirred the embers of one of the oldest of Lincoln quarrels—the alleged illegitimacy of the president's mother, Nancy Hanks. All along, Gordon suspected that Lincoln inherited the disorder—a dominant trait caused by an abnormal gene, with no skipped generations—from his mother, said to be a tall, gangly (5'8", 130 lbs.), sad-faced woman.

Herndon wrote that Lincoln's mother was "above the ordinary height in stature, weighed about 130 pounds, was slenderly built, and had much the appearance of one inclined to consumption. Her skin was dark; hair dark brown; eyes gray and small; forehead prominent, face sharp and angular, with a marked expression of melancholy which fixed itself in the memory of everyone who ever saw or knew her."

Gordon became resolute in his opinion after examining members of the Sparrow family of Anderson County, Kentucky, descendants of Lucy Hanks, Lincoln's grandmother, who married Henry Sparrow after Nancy's birth.

At first, Gordon thought that Nancy Hanks received the defective gene from her father, whose identity is still in ques-



John Lattimer, M.D., argues that Lincoln could not have had Marfan's syndrome, because he had great muscular strength and lack of symptoms that suggest connective-tissue deficiency. He supports his view by comparing a cast of Lincoln's right hand clutching a piece of broomstick (left) with the hand of a man of Lincoln's height grasping a similar piece of broomstick. (Right) A cast of Lincoln's left hand, next to the hand of a man of the same height, with Marfan's syndrome, shows Lincoln's fingers are thick and muscular, while the Marfan fingers are excessively slender and muscularly deficient. The Marfan's syndrome thumb is excessively long, while Lincoln's thumb is of normal length. Abraham Gordon, M.D., and others argue that many Marfan's syndrome patients do not exhibit all the symptoms of the disease. *PHOTOS PUBLISHED COURTESY OF JOHN K. LATTIMER, M.D. ScD., COLUMBIA UNIVERSITY.*

tion, especially since Lincoln once said to biographer Herndon, "Billy, I'll tell you something.... My mother was a bastard, the daughter of a nobleman so-called of Virginia.... My mother inherited his qualities, and I hers." But when Gordon found distinct Marfan characteristics in several members of the Sparrow clan who contacted him after publication of his original findings, he considered the possibility that Nancy inherited the gene from her mother. Still, he leans toward the original theory.

"Marfan's syndrome appears as a mutation in about 25 percent of the cases," he says. "But in diseases of genetic origin, illegitimacy is far commoner than is mutation. I believe this was true in Lincoln's case."

Either way, however, Gordon theorizes that Nancy Hanks Lincoln died of something other than "milk sickness," as history dutifully reports. "If cattle ate a certain wild-growing herb, it appeared in their milk, which is toxic for humans. But why wouldn't her two children become ill? Because children drink milk more than adults do. So I question that explanation of her death."

The famous "big foot" photograph by Alexander Gardner shows both the size of the president's feet and the blurriness of the front foot – which could be caused by Marfan's syndrome, or by the foot being out of the camera's focus range. *PHOTO COURTESY LLOYD OSTENDORF COLLECTION.*

Did Nancy Hanks Lincoln die at the age of 34 of Marfan's syndrome? The answer is unknown. But the disease can be fatal, varying widely in severity, symptoms and age of onset. Marfan's syndrome affects the connective tissue, normally thought of as the "scaffolding and the glue that holds all of our organs together," as Dr. Reed E. Pyeritz, a Marfan expert and internist at John Hopkins University Medical School puts it. Connective tissue appears in all of the organs, and provides the strength and elasticity of blood vessels, in particular the aorta. The Marfan's sufferer's biggest life-threatening problems are the damage to the aortic valve, allowing blood to leak back into the heart, and the progressive ballooning of the first two to four inches of the ascending aorta closest to the heart, where tearing or rupturing can occur. Such was the case with Olympic volleyball player Flo Hyman, who died at 31 in 1986 when her aorta burst during vigorous exercise. She was thought to be in peak condition.

One complication in detecting the disorder, according to Gordon, "is that you have no symptoms to speak of at all until this catastrophic illness hits you. You can have some characteristics, but you don't have to have them all. And I think many individuals don't have."

Despite confusion in this area—even among his fellow physicians—Gordon has come to see his theory adopted into general use. *The New York Times*, *Newsday*, *Time*, and *Pfizer Spectrum* are but a sampling of the periodicals which have reported the link between the president and Marfan's disease. *Newsweek* even headlined an Oct. 8, 1984, article, "The Lincoln Syndrome." The magazines have cited many of the syndrome's characteristics, such as long, slender fingers ("arachnodactyly," from the Latin word for "spider," which the violinist Niccolo Paganini displayed), arm spans that sometimes exceed the patient's height, protruding or sunken chests, blurred or distorted vision, rugged facial features, gothic or "arched" (sometimes cleft) palates, curvature of the spine, pronasis of the chin, and the devastating impact on the cardiovascular system. The disorder is thought to affect one in 100,000 (although Pyeritz believes that number is too low), and to be as common as hemophilia and more prevalent than

cystic fibrosis.

But the Lincoln/Marfan argument was to lose credibility in some circles when Dr. Harold Schwartz, an instructor at the University of Southern California School of Medicine, stepped into the arena. The late doctor first gained publicity two years after Gordon published his findings in 1962. He went on to become an especially vociferous and enthusiastic proponent of the Lincoln/Marfan debate. Schwartz agreed with most of Gordon's assertions, but he insisted that he, too, had examined descendants of the doomed president (beginning with a 7-year-old Marfanoid boy who was said to be descended from Mordecai Lincoln II, the president's paternal great-great-grandfather), and concluded that the Marfan gene had come up through Lincoln's *father*, not his mother, as Gordon believed.

Schwartz announced with much fanfare that had John Wilkes Booth not fired the fatal shot the president would have died during his term in office.

Furthermore, he announced with much fanfare, including a full-page interview in the May 22, 1978, issue of *Time*, that had John Wilkes Booth not fired the fatal shot on April 14, 1865, the 56-year-old president would have died during his term of office.

"He was already in heart failure at the time he was assassinated," Schwartz told the Associated Press in 1982. "He could not have lived more than six to twelve months."

That claim, in particular, outrages Gordon, who has sought to distance himself from Schwartz in every way. The Kentucky physician believes that Lincoln's young sons may have died of complications from Marfan's syndrome, especially 18-year-old Tad, who expired in 1871 from what was then called "dropsy of the chest," but which Gordon thinks was probably congestive heart failure with cardiac asthma. But Gordon says that Schwartz's speculation that the president suffered from aortic insufficiency or threatened to experience dissecting aneurysm is "ridiculous, and basically dishonest. There's nothing to back it up. That is the conclusion of a fertile brain and a feverish imagination."

Schwartz based his "evidence" for imminent death on two points. The first was Lincoln's own description of his health. In 1866, his friend Joshua Speed recounted that seven weeks before the assassination, he overheard two women make an impassioned plea for Lincoln to pardon several draft resisters. After they left, Speed remarked to the president, "It's a wonder that such scenes as this don't kill you." To which Lincoln is supposed to have responded, "I am very unwell—my feet and hands are always cold—I suppose I ought to be in bed."

This Schwartz attributed to serious circulatory insufficiency, labeling Lincoln a "Class II" cardiac patient. The

Nancy Hanks Lincoln died at age 34 in 1818. No photographs exist of her. But she was tall, lanky, and angular, and described as having a melancholy expression. Dr. Gordon believes the president inherited Marfan's syndrome from her. *PAINTING BY LLOYD OSTENDORF*

doctor, who wrote three papers on the subject, also cited a *New York Herald* item from March 13, 1865, "Mr. Lincoln . . . sick today." But critics of this information have countered with reports of Lincoln's renowned hypochondria, adding that a few weeks after the March 13 illness, he was well enough to travel to the conquered rebel capital of Richmond, where he walked the streets and climbed stairs with apparent ease.

The most challenged point of Schwartz's supporting evidence of cardiac involvement, however, revolves around an 1863 photograph of Lincoln taken by Alexander Gardner. In this pose, now referred to as the "big foot" photo, Lincoln's legs are crossed, with the left, or elevated foot—the one nearest the foreground—blurred out of focus.

One day, according to an article in *Civil War History* by Gabor and Adam Boritt, Lincoln was looking at the photograph with his journalist friend, Noah Brooks. "I can understand why that foot should be so enormous," the president reportedly said. "It's a big foot anyway, and it is near the focus of the instrument. But why is the outline of it so indistinct and blurred? I am confident I did not move it." Brooks then suggested that "the throbbing of the leg arteries inside the bend of the knee caused an almost imperceptible motion." Lincoln supposedly then reassumed the pose in the photograph, and declared, "That's it! That's it! Now, that's very curious, isn't it?"

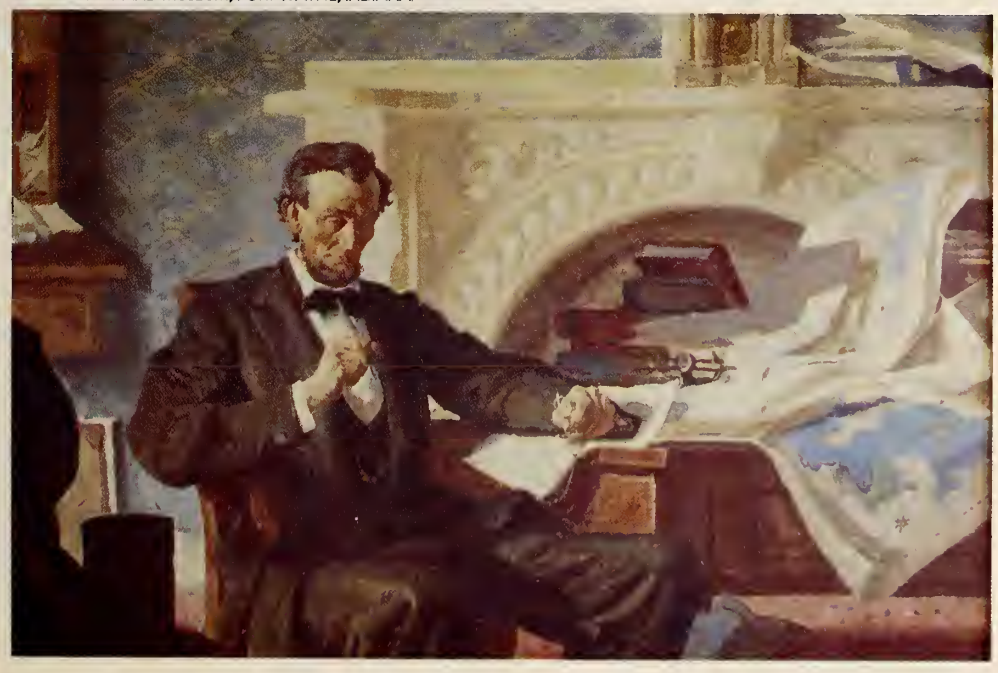
Schwartz interpreted the shaking leg as indication of excessive pulse pressure from "aortic regurgitation." That, and the severe headaches Lincoln allegedly suffered were, in Schwartz's view, the result of "a decompensating left ventricle which was the undiagnosed or concealed cause of the president's failing health."

The assertions came under heavy attack from several sources, including Lincoln historian Harold Holzer, who pointed out in the February 1983 issue of *MD* magazine ("The Health of Abraham Lincoln: How Sick Was He? Or Was He Sick at All?") that "Lincoln was probably right the first time about the photo: His foot *was* too close to 'the focus of the instrument'—so close that it probably distorted the depth of field, a condition exaggerated by the primitive camera, which required up to 15 seconds to complete an exposure. Many Lincoln photos of the period show blurriness in the foreground details."

Others saw Schwartz as little more than an opportunist, or perhaps a scientific and historical charlatan. Lincoln expert Lattimer, a Columbia University professor and chairman emeritus, says that Schwartz "just went overboard. That kind of nonsense is inexcusable." He further mentions that Schwartz's own Lincoln family diagram indicates that the syndrome skipped generations, and scientists know that the condition occurs in successive generations. Furthermore, the Boritts note in their article that "we cannot be certain about the Lincoln ancestry of Schwartz's young patient because, in the name of confidentiality of doctor-patient relationship, the evidence is not made available to historians [and] claims of Lincoln kinship are altogether too numerous in this country."

And Gordon, who has examined some three dozen Marfan sufferers over a period of 40 years, claims that the California physician simply "muddied the

(Above) Before becoming president, Lincoln's features were circulated on campaign lithographs such as this 1860 Currier and Ives. (Below) Presidential acts were recorded in paintings, such as *Lincoln Proclaims Thanksgiving Day* by Dean Cornwell. *LOUIS A. WARREN LINCOLN LIBRARY AND MUSEUM, FORT WAYNE, INDIANA.*



water, added nothing new, and tried to take credit for the whole thing." Furthermore, he says, "I can never remember seeing anyone with aortic insufficiency who had a spontaneous movement of his foot. Never. The patient's systolic pressure goes up, and the diastolic pressure goes down, and if you feel the femoral arteries, it's sharp, and goes up and down very quickly. But it doesn't move the leg."

Who then is correct? And was Lincoln one of the finest physical specimens to ever assume the presidency—doctors remarked about his muscularity as he lay on the autopsy table—or one of the most seriously ill? Was he a man just past the prime of life, or a walking time bomb? And how accurate can any diagnosis from a distance be?

Gordon readily admits that "there are missing pieces. We can't *get* all the facts." But Lattimer, who is also a forensic expert, author (*Kennedy and Lincoln: Medical and Ballistic Comparisons of Their Assassinations*, Harcourt, Brace, Jovanovich 1980) and collector of Lincoln artifacts (the blood-stained collar cut off of the dying president at Ford's Theatre, the handles of his coffin), is absolute. In November 1981, the *New York State Journal of Medicine* published his study, "Lincoln Did Not Have the Marfan Syndrome," and today he frequently lectures on the subject, using photographs and slides to support his stance.

In his much-reprinted study, in his talks, and in personal interviews, Lattimer outlines several main points, among them:

1. Skeletal Structure: "The dimensions of Lincoln's body were within the normal range of those for tall, lanky persons." As proof, he cites the recollection of Dennis Hanks, Lincoln's cousin and childhood playmate, who described the future president as "a powerful boy, round, fat, plump, well-made and well-proportioned." And in an interview, Lattimer mentioned a recently discovered outline of Lincoln's feet, made by shoemaker Peter Kahler, and now in the possession of Gabor S. Boritt. The sketch shows Lincoln to have had the big toe of someone who wears a size 14 shoe (earlier estimates were 16 1/2). Boritt, in an interview, said the sketch doesn't disprove the Marfan theory, since someone can have the disorder without having long toes. "But it does disprove the long toes." Lattimer interprets the sketch to show "the outlines of his feet are perfectly normal. They are not Marfan feet." In addition, Lattimer says, "In Lincoln's day, the maximum life span of a male (who had the disease) was 50. He wouldn't have made it to 56.... Old age is rare, although not impossible."

2. Eyesight: "There is insufficient evidence to support the diagnosis of dislocated lens, or of any other severe ocular troubles in Lincoln." The proof of that, he says, is twofold. "Marfans are

During the war, songs such as "Our National Union March" carried images of the Union's leader. *LOUIS A. WARREN LINCOLN LIBRARY AND MUSEUM, FORT WAYNE, IN*

very nearsighted. I have a pair of his eyeglasses, and they're clearly normal, farsighted glasses." In addition, he says, Lincoln did not have fragile connective-tissue supports for the lenses of his eyes, "as do patients with the classic Marfan syndrome. Lincoln was perfectly able to fire a heavy military rifle with open sights, with great accuracy, as recently as twenty months prior to his death." The head-jolting recoils of these rifles "kick something fierce," Lattimer said in an interview. "And he had shot them a lot, because he was very good at it, when you look at his target. That, and one good trauma such as being kicked in the head by a horse, as Lincoln was (in his youth) will jar those tiny threads of connective tissue. His lenses would have broken loose."

Lattimer terms Lincoln's famous "roving eye," an upward deviation of his left pupil, noticeable in a famous portrait by Alexander Gardner, the result of "a mild muscular weakness rather than . . . a dislocated lens—obviously of minor degree." Others report that Lincoln suffered at least two episodes of double vision.

3. Lincoln the Wrestler: "Lincoln's record of great muscular strength as a champion wrestler and the lack of symptoms that would suggest connective-tissue deficiency make it impossible to pin the diagnosis of the Marfan syndrome on Lincoln himself, as I see it," says Lattimer. In essence, Lattimer says Lincoln couldn't have been a "big, tough, muscular frontiersman" if he were Marfan. "Just before he was assassinated, he put on a display of woodchopping with a heavy axe, making the chips fly in all directions, a feat that amazed the audience of convalescent servicemen at the soldiers' home near Washington."

Furthermore, he says, Lincoln had no joint laxity. "The connective-tissue deficiency of the hands of people with Marfan's would make it impossible for them to grasp a heavy wrestling opponent and hold him down; the opponent could take each finger of the man with Marfan's and bend it backward. In fact, he could bend back the entire wrist and arm and thus escape from any 'hold' at all, even if the man with the Marfan syndrome should succeed in wrestling him to the ground."

Lattimer's greatest evidence for this point is a series of photographs, in which copies of the famous plaster casts of Lincoln's hands, made by Leonard Volk in 1860, are laid alongside the hand of a male Marfan sufferer of the same height. Lincoln's fingers appear thick, and his hand muscular, while the Marfan hand has "excessively slender bones and deficient muscularity," in Lattimer's words.

In summary on this subject, Lattimer says, "There are no very strong

Continued on page 43

"There are no very strong people who are Marfans," says Lattimer.

After Lincoln's death, he was eulogized in popular lithographs such as *In Memory of Abraham Lincoln, the Reward of the Just* (above) and compared to Washington (below)
LOUIS A. WARREN LINCOLN LIBRARY AND MUSEUM, FORT WAYNE, IN

The Tampa Tribune-Times, Sunday, February 10, 1991

Lincoln's legacy could help Marfan's victims

WASHINGTON (AP) — Scientists want to test bone fragments, strands of hair and blood stains from Abraham Lincoln to determine if the 16th president had an inherited condition called Marfan's Syndrome.

Marc S. Micozzi, director of the National Museum of Health and Medicine, said Saturday that new techniques may make it possible to use 126-year-old specimens in his museum to reconstruct Lincoln's complete genetic pattern.

Such studies, he said, could tell much about Abe's aches, pains and health problems.

"There is a lot of potential social value to learning the answers to these questions," Micozzi said.

While settling the historians' debate about whether Lincoln had Marfan's, he said, the studies could also "provide an inspiring perspective" on what people can accomplish despite serious medical problems.

Marfan's is an inherited condition that can have painful and crippling effects. Its most common symptoms include exceptional height and thinness, along with elongated fingers, arms, toes and legs and the effects of the condition can range from mild to very serious heart problems.

> "There could be a very inspiring message from whether or not Lincoln had these medical conditions ... and that it did not prevent him from enormous accomplishment."
>
> — Dr. Marc S. Micozzi

Patients severely affected die at the average age of 32.

Lincoln, assassinated at age 56, had many of the characteristics of Marfan's, but medical data on him isn't detailed enough to determine if he really inherited the disorder.

To settle the question, Darwin J. Prockop of the Jefferson Institute of Molecular Medicine in Philadelphia, has proposed genetic testing of medical specimens collected during Lincoln's autopsy in 1865.

Micozzi said his museum had eight to 10 bone fragments recovered by Army doctors when they performed a post-mortem after Lincoln was assassinated. The president was mortally wounded with a gunshot to the head as he sat in a box at Ford's Theater in Washington.

The bone fragments, which are only fractional inches in size, bear the beveled marks of penetration by a bullet, Micozzi said. The museum also has strands of hair and clothing stained with Lincoln's blood.

Prockop has developed tests that extract DNA, the molecule that contains the genetic pattern, from very small samples of tissue, bone or hair. Micozzi said if Prockop is successful, Lincoln's entire genetic pattern could be determined and his genes preserved for future studies.

The findings, Micozzi said, could enable Lincoln to instruct a whole new generation.

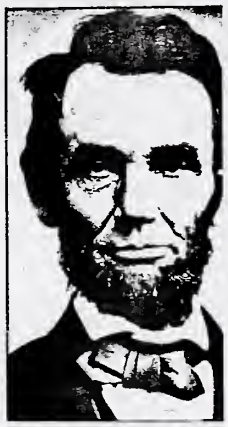
"We live in an era where we are taking a hard look at disease and disability and what it means in people's lives," he said. "There could be a very inspiring message from whether or not Lincoln had these medical conditions and what he overcame and that it did not prevent him from enormous accomplishment."

Asked if he thought Lincoln would approve of the idea of genetically testing specimens from his body, Micozzi said:

"It was very clear that Lincoln was a very strong supporter of all kinds of scientific and medical research. And he was very much interested in his own health."

A committee organized by the National Museum of Health & Medicine Foundation to study Prockop's proposal will meet in May.

Members of the committee will include people from the White House staff.



Abraham Lincoln's features semble symptoms of Marfa

Experts hope to test Lincoln bone bits

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Patients severely affected die at the average age of 32.

Lincoln, assassinated at age 56, had many of the characteristics of Marfan's, but medical data on him isn't detailed enough to determine if he really inherited the disorder.

"He was tall and gaunt and narrow in the chest," Micozzi said. "From some descriptions, it looks like his legs and arms may have been within the range that you see with Marfan. But having gone over all the evidence, I do not have an opinion to any reasonable degree of medical certainty."

To settle the question, Dr. Darwin J. Prockop of the Jefferson Institute of Molecular Medicine in Philadelphia, has proposed genetic testing of medical specimens collected during Lincoln's autopsy in 1865.

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Doctors who performed the autopsy kept the specimens and family members turned them over to the museum in the 1950s.

Prockop has developed tests that extract DNA, the molecule that contains the genetic pattern, from very small samples of tissue, bone or hair. Micozzi said if Prockop is successful, Lincoln's entire genetic pattern could be determined and his genes preserved for future studies. The genetic pattern could bear evidence of Marfan's along with any other disorder Lincoln many have inherited.

"We live in an era where we are taking a hard look at disease and disability and what it means in people's lives," Micozzi said. "There could be a very inspiring message from whether or not Lincoln had these medical conditions and what he overcame and that it did not prevent him from enormous accomplishment."

Heart problems linked to Marfan

Dear Dr. Donohue: Recently, you had an article about Marfan syndrome. Shortly thereafter, my 37-year-old daughter had heart surgery, and the cardiologist mentioned the possibility of the syndrome. I find only vague references in my books, including mention of the fact that President Lincoln had it. Thank you in advance for any help. – D.I.

Marfan syndrome in its full-blown state features a tall, slender body; long fingers; sternum deformities; and lax ligaments. Eye lens dislocation often is part of the syndrome.

Dr. Paul Donohue

It's interesting that you should mention the 16th president. Some medical historians a few years back popularized the Lincoln-Marfan speculation, presumably based on photographs and portraits revealing Lincoln's gaunt frame and long fingers.

But in the same batch of mail with your letter came a note from Dr. A. Lambert, a Grand Rapids, Mich., physician who has made a careful study of Marfan syndrome. He concludes in passing that Lincoln did not have the syndrome.

I'm glad your daughter is doing well. Underlying the visible signs of Marfan syndrome are its dangerous features – specifically heart-valve problems and weakness of the aorta wall. Often, aortic leakage must be repaired to restore normal circulation. Threat of a fatal rupture might call for surgical grafts. For a complete rundown on the syndrome, call the National Marfan Foundation, toll-free, at (800) 862-7326.

Dear Dr. Donohue: I had mononucleosis last year, and I have again tested positive for it. Is it possible to have it twice? Also, what is the difference between mono and the Epstein-Barr virus? – M.L.

It's highly unusual, almost unheard of, to have mononucleosis twice.

The usual case lasts two to four weeks and then is gone, never to return. Some residual fatigue might persist for another month, by which time most patients are back on their feet, spry as ever.

I should note some recent speculation about a relapsing form of mono, an apparent flare after the initial illness has subsided. If it occurs – and some doubt it happens – it would be rare indeed.

What test did you have? Antibody tests remain positive for years, so your current positive result might be but a calling card of last year's infection.

Epstein-Barr is the virus of mono, and it's transmitted in saliva and mucus, a fact that gave mono its other name: "the kissing disease."

Dr. Paul Donohue is a North America Syndicate columnist whose work appears daily. Write to Dr. Donohue, The Journal Gazette, P.O. Box 88, Fort Wayne, IN 46801-0088. 9-11-96