THE RELATIONSHIP OF CLUBBED FINGERS TO CERTAIN TYPES OF CARDIAC DISEASE AND ATROPHIC CIRRHOSIS OF THE LIVER

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DEFINITION OF SUBJECT

This discussion deals with the observation of clubbed fingers set forth in the case records of patients in the Wisconsin General Hospital in whom the primary clinical diagnosis was either coronary heart disease (arteriosclerotic heart disease), hypertensive heart disease, syphilitic heart disease or atrophic (Laennec's) cirrhosis of the liver. Primary diagnosis of arteriosclerosis of the pulmonary arteries (Ayerza's disease) was sought but not found. A review of the literature of the subject is made and the theories of etiology are reviewed including some of experiments made to verify them.

REVIEW OF THE LITERATURE

Historical:

Hippocrates, in the fifth century B.C., writing in "Prognostics, number 17" indicates early clubbing of the fingers in empyema, "the nails of the hands are bent, the fingers are hot especially their extremities." Aretaeus, the Cappadocian (about 100 B.C.), likewise confined his attention to the increased curving of the nails in phthisis", the nails of the fingers are crooked, their pulp are shrivelled and flat, for owing to the loss of flesh they neither retain their tension or rotundity and owing to the same cause the nails are bent, namely, because it is the compact flesh at their points which is intended as a support for them and the tension thereof is like that of the solids. In empyema, he noted, "swellings of the extremities of the feet and fingers of the hands, which at one time abate and at another increase", undoubtedly clubbed fingers.

Caelius Aurelianus, however, first drew attention to the "drum-

stick" or clubbed appearance of the digits as a sign of chronic empyema and phthisis. Pigeaux made the next historically noted observation of clubbed fingers, referring to the occurrence of Hippocratic nails in 167 of 200 phthisical patients. Trousseau described clubbed fingers accurately and pointed out its association with advanced pulmonary tuberculosis, empyema, emphysema, nervous asthma and organic heart disease.

Gay (1871) described clubbed fingers on both hands with bilateral subclavian aneurysm.

The literature is swelled by the interest which von Bamberger aroused by the finding of gross changes in the long bones associated with clubbing of the fingers and toes in two cases of bronchiectasis. Marie gave this full blown pathological syndrome its wordy title, osteo-arthropathie hypertrophiante pneumique (which, unfortunately, has persisted, however translated, added to, or shortened) having observed it as a secondary manifestation of cardiac and pulmonary disease.

The incidence of clubbed fingers in various diseases has been indicated mainly because of its constant association with secondary hypertrophic pulmonary osteoarthropathy. [Omission, see page following, 2b]

MORPHOLOGY

A simple inspection of the patient's outstretched fingers is part of the clinical examination that can provide information of considerable value in diagnosis. The discovery of clubbed fingers is at the same time satisfying to the clinician and condemning to the patient for it is a condition long known to be associated with grave disease.

Locke's "Secondary Hypertrophic Osteoarthropathy and its Relation to Simple Clubbed Fingers" (1915) is the classical review. He recommends the previous reviews of Walters, Massalongo, Thayer, Janeway, Thompson, Wynn, Ebstein and Alexander. Since his time Högler (1920) has been quoted frequently. Witherspoon, Horsfall, and others have stimulated the interest in etiology by suggesting its occurrence on the basis of an inherited tendency. The Comperes and Adams lead a Chicago group, attempting to produce the changes in experimental animals by causing lesions similar to the usual pulmonary human type, whose critical standards and methods are admirable. Sir Thomas Lewis has fostered the inquiry into the pathological physiology.

Clubbing is one of those phenomena with which we are all so familiar that we think we know more about it than we actually do. Clubbing is first seen in the thumb and index finger. The distal phalanges are swollen giving the fingers a drumstick appearance. The enlargement starts at the root of the nail just beyond the interphalangeal joint as a definite firm transverse ridge more obvious on the dorsal aspect of the finger, for the increase in size is more gradual on the palmar side. The overlying skin is smooth and shiny and the patient often complains that the cuticle is growing more rapidly on the nail. The hyperconvexity of the nail was likened to a watch crystal by Lefevre, and may become so greatly curved in both directions, so much thickened and have such marked accentuation of the longitudinal ridges as to offer a close resemblance to a parrot's beak. Vascular turgescence of the nail bed produces a lilac hue and it becomes full, rounded and cyanotic. Color returns slowly after slight pressure and indicates sluggish circulation. With development of deformity the base of the nail becomes elevated so that it soutline may be discerned beneath the skin. When true clubbing of the fingers is present, if the observer places one of his fingers upon the upper edge of the nail at its free margin, i.e. the distal end, and his thumb upon the under surface of the clubbed finger and presses downward, the hard margin of the root of the nail can be distinctly felt if another finger is applied over it. When severe, the nail can be rocked backwards and forwards giving the impression that it is floating on a soft edematous pad. If of long standing, as in congenital heart disease, hyperextension of the distal phalanx develops, as first described by Marie. In early stages of clubbing slight curving of the nails, slight cyanosis and mobility of the nail root occur before the obvious swelling or clubbing. Shininess and smoothness of the skin over the base of the nail, Witherspoon

considers significant early changes.

In the vast majority of cases the development of the clubbing is insidious and slow and usually passes unnoticed by the patient. Rarely it develops quickly. West records two cases in which the clubbing developed in two and four weeks, respectively and Godlee mentions a case in which well marked clubbing developed in two months. Aside from the deformity there are no symptoms, although occasionally pain is complained of in rapidly developing cases. Once established, the deformity is permanent, although some improvement has been noticed after the drainage of an empyema.

MICROSCOPIC MORPHOLOGY

There is no agreement on essential changes in the soft parts by different observers. This adds considerable difficulty in offering a satisfactory explanation of the manner in which clubbing arises, which is not diminished by the extraordinary number of diseases in which the deformity appears. Campbell presented a photomicrograph of a longitudinal section of a clubbed finger and noted a distinct thickening of the tissues between the nail and the phalanx due to the presence of excessive edema. The fibrils of the connective tissue, instead of running in compact bundles as in normal fingers, were widely separated from each other in many places. He noted no increase of the rete mucosum, which Shaw said Buhl described and no increase in the fibrous tissue, although he stated that if the edema persists long enough increase in the connective tissue will result, and no increase in fat. Dr. Norman Moore (Shaw), in support of edema says ~~Shaw states~~ that pressure reduced the finger to normal size. Campbell did not find that as Freytag noted there was dilatation of the capillary loops in the nail bed with engorgement of the interpapillary pro-

cesses without alteration in the skin, fibrous endarteritis obliterans and thickening of the nerve sheaths, although his illustration does show a vascular proliferation. Therese commented on the hypertrophy of the horny layer papillae, and connective tissue of the derma. Thomas described marked dilatation of the venous side of the capillaries of the nail bed and extreme degree of tortuosity of the arterial side. Buzzard saw mainly excess fat. Landis supports this finding, "Two clubbed fingers which I had removed at autopsy and placed in Kaiserling's solution both lost the deformity. Fowler had a similar experience with a clubbed finger preserved in alcohol (though hypertonic solutions would dispose of edema equally well). These are not blind men feeling different parts of an elephant, but they may be describing the clubbed finger in different stages of development by slightly different pathological standards considering their ages and schools, and from preparations made by widely different techniques which alter the appearance of the tissues.

X-RAY APPEARANCE

Distal phalanges in healthy individuals vary much in size and shape. In clubbed fingers, apparently the simple stage, Locke found phalangeal periosteum proliferation in five out of twenty-one cases. In familial enlargements the bony part of the finger tips have been described as ~~spread~~ splayed out, or showing spatulation. The characteristic alterations in the end phalanges, if present, consist of irregular mossy proliferation mostly in the distal half which produce a bur-like appearance, which Locke says may look like the end of a magnet dipped in iron filings.

CHEMICAL EXAMINATION

Thompson reports that chemical examination reveals marked increase in magnesium phosphate at the expense of calcium salts with increase in organic matter.

SYNONYMS

Clubbed fingers are also known as les doigts Hippocratisme, les doigts Hippocratiques, Hippocratic fingers, bulbous fingers, spade fingers, serpent head fingers, trommelschlagel finger, drum stick fingers, acropachy. The peculiarity of the nails has been named (and doubtless has often been used as insufficient name for clubbed fingers) Hippocratic nails, les doigts en ~~l~~baquette de tambour, watch crystal nails, parrot beaked nails, the hyperconvex nail of ~~the~~ hypertrophic osteoarthropathy.

ASSOCIATED CHANGES

Hypertrophic pulmonary osteoarthropathy is the commonest. The relation of hypertrophic pulmonary osteoarthropathy to clubbed fingers is firmly established by the arguments of Locke and it has been well summarized by Holman. Locke defined hypertrophic pulmonary osteoarthropathy as a condition characterized by general and symmetrical clubbing of the fingers and toes often associated with hypertrophy of the long bones of the feet and hands, and less frequently with painful enlargement of the long bones of the forearms and legs. The condition is secondary to some chronic ~~and~~ or, rarely, an acute, disease, most commonly of the lungs. With this syndrome Brooks describes the signs of globular nose tip and malar puffiness, having seen it in fifteen of fifty-seven cases. Clark has seen new bone formation in the diploe of the cranial bones. Others have seen new bone formation in

almost all of the other bones of the body when involvement has been estensive and severe. Cooper and Cosman have observed a peculiar nodular tumor on the hard palate in the midline extending from the horizontal plate of the palate to the foramen of Scarpa situated in the anterior portion of the palate process of the superior maxilla, 45% in actively tuberculous males, 25% in inactively tuberculous males, causing speech changes but no subjective symptoms. They also find hypertrophy of the bony processes of the jaws in about the same proportion of the same kind of cases.

The rationale of the identification of clubbed fingers as a stage in the same process as hypertrophic pulmonary osteoarthropathy is indicated by four points which Landis articulates:

1. "Both simple clubbing and hypertrophic osteoarthropathy are associated with the same general group of diseases.
2. Clubbing of the fingers is one of the characteristic features of hypertrophic osteoarthropathy.
3. The clubbing which occurs in the Hippocratic fingers is identical with that occurring in osteoarthropathy, except perhaps in the degree of development.
4. Osseous changes sometimes occur in simple clubbing, which are precisely the same as those seen in hypertrophic osteoarthropathy; 12 out of 39 cases of simple clubbing studied by Locke showed bony changes."

In this paper, as Holman and Vosquil have done in determining the incidence of clubbed fingers in the primary diseases which more commonly cause hypertrophic pulmonary osteoarthropathy, clubbed fingers are taken

as a stage of osteoarthropathy.

The degrees of development of clubbed fingers and hypertrophic pulmonary osteoarthropathy have been divided by Sternberg into three groups: (1) Pure clubbed fingers, (2) clubbed fingers with transitory periosteal deposits on the distal ends of bones of the extremities (Bamberger's type), (3) the severe picture with high grade changes in most parts of the bony system (Marie's type) which is symptomatic while the first two are usually asymptomatic.

As hypertrophic pulmonary osteoarthropathy has not been diagnosed roentgenologically in any of the cases presented, no great emphasis will be laid upon its pathology as discussed in the literature. Because of hints at etiology and out of deference to its pathological great interest to its investigators its main features will be indicated. Quoting Carr, whose description agrees with the others, "The osseous changes occur most frequently and characteristically in the bones of the extremities. The essential bony change is proliferation of the periosteum. The thickening of the ends of the bones, especially of the lower ends, is especially characteristic of the hypertrophic pulmonary osteoarthropathy, though the proliferations are not confined to these localities. Irregular thickenings, due to this periosteal proliferation, may be found through the length of the bone."

Locke's x-ray examinations are typical. He finds irregular subperiosteal new bone formation which is distinctly layered, occasional osteophytes near the joint margins, and occasionally in cases with joint symptoms, rarefaction of the articular cartilages.

Gross Pathology: Compere and Adams give this description "When

these bones (tibia, fibula and ulna) were cut longitudinally, the new bone was found to be 1 to 5 mm. in thickness and possessed a thin cortex with cancellous bone filling in the space between this and the old cortex which was still intact. The cortex and cancellous portion of the old shafts were grossly normal." Occasionally, as Hall found, the bones are characterized by extreme softness of texture, the post mortem knife cutting them easily.

Microscopic Pathology: In examining microscopically the bone described above, these authors found, "the raised periosteum and the space between the periosteum and the old cortex filled with spongy cancellous bone which has surrounded the shaft, increasing its diameter by 30 to 50 per cent. There is slight absorption of the old cortex."

The legend of their figure 4 gives microscopic detail. "Photomicrograph of a section of the distal end of the tibia. A. left. The outer portion of the new bone is slightly denser than that near the old cortex and a definite new cortex is forming. There is some smooth absorption of the old cortex on both the medullary and outer surface with areas showing indentation and an occasional giant cell and lacunar absorption. There are very few cells of any kind and no infiltration of round cells. The type of new bone which has been formed is that which is usually seen at the site of an injury to bone. B. A higher power, shows both absorption of the bone trabeculae throughout the cancellous portion of the old shaft and definite new bone formation. The extracortical new bone has been formed in irregular trabeculae and the spongy bone is highly vascular, particularly just outside the old cortex." In figure 5, B, they point out, "higher power shows a brown zone of amorphous or gelatin-like material which may be fibrin or

jelled serum paralleling the old cortex and present on both the inner and outer surfaces. Whether this is the product of bone absorption cannot be determined under the high powered microscope. Fine vacuoles are noted in this brown zone.".

In discussing the microscopic pathology, they add, "At first the layers of periosteal bone are distinctly separate from each other; they may accumulate until as many as six or more layers are present and indistinguishable in the roentgenograms." They affirm its softness, vascularity and friability and that the old shaft retains its form, but is less dense, due to loss of lime salts and an increase in fat content.

Synonyms: The variety of names which has been given hypertrophic pulmonary osteoarthropathy is even greater than that given clubbed fingers. Toxicogenic osteoperiostitis ossificans (Sternberg), hyperplastic periostitis, secondary hyperplastic periostitis, toxic periostitis, hyperplastic osteoarthritis, ossifying periostitis and secondary ossifying periostitis are objected to by Hëgler as signifying an inflammatory process. He denies that such is present. He says, "Roentgenologic examination shows that the shaft of the bone is intact and that the thickening rests exclusively on the periosteal deposits described. These deposits are hyperplastic and not inflammatory." Others are osteoarthropathie hypertrophiante pneumique, sekundare hyperplastisches Ostitis, osteo-arthropatia ipertroficas secondarie, Marie's syndrome, Marie's sign-group, von Bamberger's disease, Bamberger-Marie syndrome, generalized osteophytosis, hypertrophic osteo-arthropathy, hyperplastic osteoarthropathy, secondary hypertrophic osteoarthropathy, secondary pulmonary osteoarthropathy, tuberculous polyarthritis and akroelephantiasis. Acropachy, suggested by Hëgler, is recommended by Thomas

for the condition of new bone formation as it avoids pathological descriptive terms, chiefly the term which invites the idea that there may be an inflammation of the periosteum present, which is incorrect. It also precludes terms suggesting etiology, which may be erroneous. Acropachy is a term which is short, convenient to handle and is non-committal as far as the pathology or etiology is concerned. Analyzed as Voltaire did "Holy Roman Empire" which he found neither holy, roman nor an empire, many of the terms indicating acropachy appear similarly anomalous.

Differential Diagnosis: Brooks in reviewing the literature of hypertrophic osteoarthropathy rules out mistaken diagnosis of; rheumatic arthritis complicating rachitis, congenital syphilis with luetic arthritis, arthritis deformans or incompletely developed acromegaly, acromegaly with long standing intestinal toxemia and suppurative multiple arthritis.

Clubbed fingers ~~in~~ themselves are to be differentiated from Heberden's nodes, chronic paronychia and occupational effects on the finger tips. Locke points out that acromegaly is a primary disease, characterized by face changes (such as coarsening of the features and prognathism), central nervous system symptoms, small nails, no similar change in the bones by x-ray, some irregularity of outline especially in the epiphysis and accentuation of muscle and tendon attachments. Landis adds, enlargement of the whole of the hands and feet, of the tongue and genitals and also of the cartilages of the ear, nose, eyelids and epiglottis. In arthritis Locke calls attention to the deformity and inflammation at the articulations.

Heberden's nodes of osteoarthritis are osteophytic growths on the dorsal surfaces of the finger, distal to the interphalangeal joint at the

edge of the joint cartilage, and occur in elderly people. Chronic paronychia produce swelling of soft tissue at the base of the nail, not of the nail bed. The chronic joints of infective arthritis are characterized by fusiform periarticular swelling. In these first three there is no change in the nail bed. Subungual whitlow is painful and occurs in one finger only. Occupationally induced hypertrophy causing broadening and thickening of the distal phalanges occurs among bootmakers, sailmakers and saddlers. Lovibond graphically illustrates a profile sign in the clubbed fingers derived from a profile silhouette of the nail. It is worthy of reproduction, but in lieu of it, in the normal finger the angle made by the top surface of the nail and the dorsum of the distal phalanx is about 160°, whereas in the clubbed fingers the angle is obliterated and may be greater than 180°. Clubbing is usually symmetrical. Close inspection of the toes, allowing for the usual distortion, will reveal it there also.

INCIDENCE IN VARIOUS DISEASES

Clubbed fingers, or hypertrophic osteoarthropathy, have been observed in a great variety of diseases. For the purpose of comparing the investigations of others with the investigation presented in this paper, the primary diagnoses have been grouped and the available data which is comparable is made into tables.

(There is a large group of cases in which the incidence of acropachy has been described, but in which the primary diagnosis has not been given in the title of the article. There is a long list of the names of authors who described clubbed fingers, but whose cases Locke did not summarize because hypertrophic pulmonary osteoarthropathy was not demonstrated. With it goes a list of authors who have reported cases of clubbed fingers or acropachy since his compilation who have not named the primary disease in their titles and whose case records have not been investigated. The reference in most cases is available).

The groups are:

A. Diseases of the respiratory system (Table I)

B. Diseases of the circulatory system.
Six of these are incorporated in Table II.

C. Diseases of the alimentary tract.

D. Miscellaneous

E. Diseases of the central nervous system

F. Environmental conditions

G. Congenital (Primary. No primary lesion demonstrable).

The first four groups are those which Locke made. Other cases have necessitated more groups and have lengthened the list in the original groups.

TABLE I A. RESPIRATORY DISEASES IN WHICH CLUBBED FINGERS ARE FOUND

Disease	Authority	Incidence %	Aver. age years	Sex %		Aver. Pulse	Aver. Vital Capacity %	Aver. R.B.C. millions	Aver Hb.	Aver. Blood Pressure	
				M	F					Syst.	Diast.
1. Lung abscess	Holman	24.5 (14 of 57)	34	64.9	35.1	102.5	54.2 (4 cases)	4.1	63.6	107.9	62
2. Bronchiectasis	Holman, Clubbed fingers ~~including~~ including watch crystal nails Shaw	31.4 (74 of 217) 43. (94 of 217) 67 (38 of 57)	28.5	59.4	40.6	94.4	47.5 (18 cases)	4.49	70.9	117.9	71.8
3. Silicosis	Holman	9.1 (9 of 99)	48.2	100	0	84	71.1	4.8	81.5	131.7	86.6
4. Pulmonary tuberculosis	Holman. Clubbing ~~including~~ Including down-curved nails.	8.52 ~~1.75~~ (of 855 cases)	36.1	64.8	35.2	99.5	45.90 (39 cases)	5.057	67.8	116	74
	Pollack (Shaw)	M.29, F.23									
	Shaw (Brompton Hospital)	69.6 M. 66.4 F.									
	Kessel (simple clubbing; clubbing with bone changes Clubbing with long bone changes	32 (of ~~100~~ cases) 17 10 (6 had globular nose tip and malar thickening)									
	Corper and Cosman Hyperconvex nails	75-95,90 in active 55 in inactive			less than M						
	Landis, Curving of nails (Phipps Institute) Clubbing	38.9 (1382 of 3551) 21.7 (772 of 3551)									

5. Lung cyst
6. Chronic bronchitis
7. Empyema,
8. Malignant diseases of the lung, pleura and mediastinum
   a. Primary
   b. Metastatic
9. Pneumonia
10. Pleurisy with effusion
11. Asthma
12. Emphysema
13. Streptothrix of lungs
14. Unresolved pneumonia
15. Mediastinitis
16. Blastomycosis of chest wall.

TABLE I B. [Continuation of p.14 at same line level]

Wassermann %	Fever %	Dyspnoea %	Cyanosis %	Emphysema %	Sinusitis * %	Av.Dur's of Dis. in Years	Onset of Clubbing	Av.Ven. Pressure cm.H_2O	Cardio-vascular Lesions %	Rheu.H.D. Mitral stenosis & insuff.	Coronary Disease	Peripheral Sclerosis
									Cardio-vascular lesions			
									% of C-V lesions			
0 in all	85.7	50	28.6	14.3	28.6	3.4	-		14.3			
1.3 (1 in 74)	52.7	67.5	32.5	25.6	62.2	6.	6 Mo.Dis 4 years	10.5	16.3	40	60% 3 Decomp.	2.8
0	0	100	55.5	77.7	22.2	2.2	-	-	33.3		11.1 F.C. IIA	11.1
1.3	78.5	89.2	37.8	89.2	8.1	4.4	4 yrs Dis. 5 yrs	10 2 cases	33.8	5 cases MI,1 MS	4 of 10 had ***	
	80	80				2.6			Toxemia 100%	Cough 100%	Expectoration 100%	Stage of Disease 4 / 1
	94	68				3.62			81	100	100	14 / 3
	80	60				3.4			70	100	100	10

*Diagnosed by x-ray or antrum puncture

**Symptoms began how many years ago, is the question answered.

***Myocardial degeneration, aortitis, relative mitral insufficiency, functional capacity III.

Facts Observed		Rheumatic Heart Disease	Subacute bacterial Endocarditis	Congenital heart Disease
Cases of clubbing		70 (11% of 653)	19 (29% of 65)	10 (17% of 58)
Sex		54%M, 46% F	9 M and 10 F	6 M and 4 F
Average age (Years)		31.3 M & 28.6 F	39.3 M & 27.4 F	12.7 M & 19 F
Average duration of disease		10 years	Cardiac 16 years H.P.I. 5.2 months	-
Average onset of clubbing		(Before being seen) 2 yrs. (6 cases)	-	-
Vital capacity		59% (13 cases)	-	-
Functional Capacity	~~Compensated~~ Compensated	23%	52%	66%
	Grade I	9%	1 case	-
	Grade IIA	11%	-	2 cases
	Grade IIB	17%	2 cases	1 case
	Grade III	40%	31%	-
Emphysema		-	-	-
Dyspnoea		80%	90%	78%
Cyanosis		51%	48%	78%
Pulse (Average)		89	99	84
Blood Pressure	Aver. Systolic	129	112	107
	Aver. Diastolic	75	66	74
Average venous pressure (cm. of water)		13 (29 cases)	14 (2 cases)	-
Valvular Involvement	Tricuspid Insufficiency	17%	2 cases	
	Tricuspid Stenosis	-	-	-
	Pulmonic Insufficiency	-	-	-
	Pulmonic Stenosis	1 case	-	44%
	Mitral Insufficiency	91%	100%	-
	Mitral Stenosis	80%	74%	1 case
	Aortic Insufficiency	33%	15% (3 cases)	-
	Aortic Stenosis	5%	-	-
Interventricular septal defect		-	-	80%
Patent Ductus Arteriosus		-	-	1 case
Dextra position Aorta		-	-	1 case
Enlargement		77%	90%	-
Myocardial Degeneration		-	-	-
Adhesive Pericarditis		14%	2 cases	-
Rhythm Disturbance	Tachycardia	2 cases	-	-
	Auricular fibrillation	29%	-	-
	Extrasystoles	5%	-	-
	Gallop Rhythm	2 cases	-	-
	Heart block	-	-	-
Aortitis		7%	1 case	-
History of attacks of Rheumatic fever or chorea		74%	1 case S.F. 84% 1 case tons.	-
Dilatation of aorta		-	-	-
Fever		47%	100%	1 case
Red blood cells, millions per cc. blood		4.7	3.6	6.4
Hemoglobin % or Gm. per 100 cc.		69%	54%	96%
Wassermann Reaction		-	-	-
Blood Calcium mg./100 cc.		6.75 to 8.59	Comperes and	In H.P.O, 2 cases
Blood Phosphates mg. /100 cc.		3.69 to 6.33	Adams	

Key: - no observation
0 no occurrence
—— Joint occurrence of lesions.

e bacterial rditis	Congenital heart Disease	Coronary Disease	Hypertensive Heart Disease	Syphilitic Heart Disease
of 65)	10 (17% of 58)	14 (1,6% of ~~845~~ 843)	14 (1.2% of 313)	2 (2.5% of 80)
10 F	6 M and 4 F	9 M and 5 F.	3 M and 1 F	2 Male
& 27.4 F	12.7 M & 19 F	57.7 M & 66.2 F	52.6 M & 52 F	47.5
16 years 5.2 months	-	10 mos.in 4 cases, HPI 3 yrs.hyperten- sion, 1 case	M. 2 years F. 14 years	11.5 years
-	-	At 54.3 yrs in 17 mos: (3 cases)	65 ? (1 case)	-
-	-	30.7 (1 case)	47% (1 case)	-
	66%	-	-	-
-	- 2 cases 1 case -	1 case 29% 36% 2 cases	1 case 1 case 1 case 2 cases	- 1 case 1 case
-	-	64%	1 case	
	78%	79%	100%	2 cases
	78%	57%	75%	2 cases
	84	89	86	82
	107 74	138 88	188 114	120 56
ses)	-	14 (1 case)	13.2 (3 cases)	11 (1 case)
	-	2 cases 2 cases (Rel.)	1 case (Rel.)	1 case 1 case (Rel.)
	- - 44% -	- - 2 (rel.),7 Rel.,2 cases	- - 1 case(rel.),1 (rel)	- - - 1case(rel.),1case
es)	1 case - -	- -	- -	1 case 2 cases -
	80%	-	-	-
	1 case	-	-	-
	1 case	-	-	-
	-	57%	100%	2 cases
	-	36%	2 cases	2 cases
	-	-	-	-
	- - - -	1 case 2 cases 2 cases 1 case -	- 1 case - - 1 case	- 1 case - - 1 case
se S.F. se tons.	-	86%	100%	2 cases
	-	-	1 case	-
	-	-	1 case	-
	1 case	-	-	-
	6.4	4.92	5.21	5.48
	96%	14 Gm.(9 cases) 69% (5 cases)	77%	72%
-	-	0	0	1 case +
	In H.P.0,2 cases	11.2 (1 case)	-	-
		3.8 (1 case)	-	-

of lesions.

FACTS LISTED IN TABLE II
NOTED BY VARIOUS OTHER AUTHORS

~~Noted by various authors.~~

French noted clubbing in pericardial adhesions and in pericarditis.

Harrison found clubbing associated with cor pulmonale when pulmonic stenosis, tricuspid insufficiency and right sided hypertrophy and dilatation were present.

Jones found clubbing in fifty per cent of cases of subacute bacterial endocarditis.

Brooks states that clubbing occurs with coronary disease.

Locke mentions the importance of interventricular septal defect and pulmonic stenosis.

95472	+	-	67	F	-	-	-	IIB	0	+	0	69-112	86	164/80	180/90				
97793	+	-	65	M	-	-	-	IIB	+	+	+		100	124/96	130/96				
98004	Slight	-	75	F	-	-	-	IIB	+	+	+	48-112	90	170/90	188/110				
98816	Slight	-	63	F	-	63,4 Mos. prev.	-	IIA	+	+	+	64-100	84	140/90	160/94			128/84	
98981	Slight	-	57	M	-	-	-	IIB	+	+	+	68-112	82	130/106	142/108				
63202	+	-	56	F	Hypert. 3 yrs. prev.	Not. obs. 3 yrs. p	-	IIA to IIB	+	-	-	60-112	90	150/98	134/84-	228/120			
75992	Slight	+	44	M	-	-	-	I	0	0	0	-	110	96/76	-				
85858	+	Gangrene	70	M	-	-	-	IIA	+	+	0	-	84	124/70	-				
80716	+	-	47	M	10 mos.	46 ?	-	IIA	-	slt	slt.	68-90	84	136/86	128/72				
84016	Marked	+	54	M	5 mos.?	-	-	IIA-IIB	+	+	+	60-102	70	180/104	150/94				
80966	Some +	+	70	F	4 months	-	-	IIB	+	+	0	64/108	88	140/78	156/100	136/78-			
14 Cases	8,+ 5 Slight 1, marked 9, not noticed	4,+ 1, gangrene	FV. 60.7	M,9 F,5	Av.10.0mo in 4 Hypertension 3 yrs. in 1	54.3 11 not notice 30.7 in 1 12mos, case 4 mos, 36mos, duration	1, I 4,IIA 2,IIA 5,IIB 2,III	1 not men- tioned. -IIB 9 +	1,0 1 not 1, not ment	5,0 1, no ment	Aver. 59-109 Range 28-120	Range 70- 110 Range 89	Av. 138/88 96/70- 180/106 96/66- 120	Av. 128/82- 150/98 Range 130/80					
HYPERTENSIVE HEART DISEASE																			
95303	+	-	67	M	2 years	65 ?	47%	III	+	+	+	52-124	80	194/114	220/140	130/80			
86484	+	-	46	M	1 + yr.	-	-	I to IIA	0	+	+	60-88	84	190/110	220/150	180/110			
80828	+	-	52	F	14 + yrs	-	-	IIB	0	+	0	48-92	80	185/100	204/130	158/98			
81122	+	-	45	M	2 +	-	-	III	0	+	+	102		182/130	240/144	144/98-			
4 Cases	+, 4	4, not mention	52.6 M 3 F 1	Av.6.25 F 52 F 14 + Rng: 45-67	65 ? 3 not ment.	1:47% 3, not ment.	I, I to IIA: 1 IIB: 2 III	+,1	+,4	+,3 0,3 0,1	Aver. 48-124 Av.54 (66 with 102)	86 Range 182-194 100-130	Aver. 188/114	154/94 Range 240/142	Av. 13 Ra 9. 16. Ran 8-2				
SYPHILITIC HEART DISEASE																			
64016	+	-	49	M	15 +	-	-	IIB	+	+	+	46-120	84	130/62	85/38	140/100			
55019	+	-	46	M	8	-	2700 cc	IIA	0	+	+	60-100	80	110/50	95/40- 120/60	12 14			
2 Cases	+:2	-:2	47.5	M2: F 0	11.5	-:2	1:2700	IIA 1 IIB 1:	0:1 +:1	+:2	+:2	Aver. 54-100 Range 46-120	82	120/56	Av. 90-130 85-140	Av 11 Ra 38 80 Range 38 160 6-1			

0	+	0	69-112	86	164/80	180/90	-	Rel.mit insuff	Aur. fib.	+	+	4.25	10.3	0	11.8 10.6	4.3 3.2
+	+	+		100	124/96	130/96	-	Rel.mit insuff	(Aur.Fibr.) usually Regular	+	+	-	16.8	0	-	-
+	+	+	48-112	90	170/90	150/66- 188/110	-	Rel. mit. insuff	-	0 II 6 0III 7.5 0 IV 9 3V 12.5	+	4.68	13.8	0	-	-
+	+	+	64-100	84	140/90	128/84 160/94	-	Rel. mitral insuff	-	-	+	3.88	13.4	0	-	-
+	+	+	68-112	82	130/106	142/108	-	Rel. mitral insuff	Extra- sys- tolic	~~II~~ ~~III~~ V 11.5	+	6.19 4.19	15.6 16.6	0	-	-
-	-	-	60-112	90	150/98	134/84- 228/120	-	-	-	0	+	4.5	68%	-	-	-
0	0	0	-	110	96/76	-	-	-	-	-	-	4.8	68%	0	-	-
+	+	0	-	84	124/70	-	-	Mit. insuff	Extra- syst.	+	+	4.6	68%	0	-	-
-	+ slt	+ slt.	68-90	84	136/86	128/72	-	Rel. mit.& tricus insuff	Prema ture contra tion	+	+	6.52	88%	0	-	-
+	+	+	60-102	70	180/104	150/94	-	-	-	Slight	+	4.95	65%	0	-	-
+	+	0	64/108	88	140/78	136/78- 156/100	-	Rel. mitral insuff	-	-	+	4.34	58%	0	-	-
1 not men- tionedment 4 0 9 +	1,0 1not ment 1,++ 11,+	5,0 1 no ment 1slt 7,+	Aver. 59-109 Range 28-120	Range Av. 70- 110 Av. 89	Av. 138/88 Range 96/70- 180/106	Av. 128/82- 150/98 Range 96/66- ~~228~~/120	14 in one case	2, 3, not ment. RMI, 7 RM&TI, 2, MI, 2, 3	aur. fib. 9, no ment. Ex. sys. 1, proto- diast. gallop	4, no mention 2, 0 7, + 1, slt.	1, no men 1,0 12+	Aver 4.92 Range 3.88 6.52	Aver 14gm Range 10.3 16.8 Aver 69% Range 58- 88%	14,0	1, as above	

RTENSIVE HEART DISEASE

+	+	+	52-124	80	194/114	130/80 220/140	8- 16	Rel. mitral insuff	-	+	+	4.5 4.8	80% 15.6 Gm.	0	-	-
0	+	+	60-88	84	190/110	180/110 220/150	10	-	-	Ortho- diagram +1.5II 4.0 3III4.5 2.8IV 6 3.6V9.5	+	5.	75%	0	-	-
θ	+	0	48-92	80	185/100	158/98 204/130	-	-	-	+	+	6.6	75	0	-	-
0	+	+		102	182/130	144/98- 240/144	24- 11	Rel. mitral & tri. insuff	Aur. fib.to compl. heart block	3 II 5 3.5 III 9 0IV 13	+	5.49 4.81	55 70	0	-	-
+,1 0,3	+,4	+,3 0,1	Range 48-124 Av.54 ~~(66with 102)~~	86	Aver. 188/ 114 Range 182-194 100-130	154/94 Av. 142/142	Av. 13.2 Range 9.7- 16.7 Range 8-24	2 not ment. RMI,1 RMI & RTI, 1	as above	+,4	+ 4	Av. 5.21 Range 4.5- 6.46	Av. 70% Range 55- 80%	0 4	-,4	-,4

SYPHILITIC HEART DISEASE

+	+	+	46-120	84	130/62	85/38 140/100	6	Mit. sten.& insuff Aortic tricus insuff	-	3II2.1 4III5.3 6 IV 9 5.8 V 10	+	5.66	80	4+	-	-
0	†	+	60-100	80	110/50	95/40- 120/60	12- 14	Aortic & tri. & rel. mit.ins	-	0 II 4 4III 6 4.5 IV 12.5 0 V 14	+	5.49 4.81	55 70	Bl.0 0	-	-
0:1 +:1	+:2	+:2	Aver. 54-100 Range 46-120	82	120/56	Av. 90-130 38 80 Range 85-140 38 160	Av. 11 Range 6-14	MS&MI 1 A& TI 1 RTI 1 RMI 1	-:2	+:2	+,2 ~~12~~	Av. 5.32 Range 4.81 5.66	Av. 68.3 Range 55- 80	4+:1 0:1	-:0	-:0

Case Number	Clubbed fingers	Clubbed toes	Age	Sex	Duration of the Primary clubb	Age of onset	Vital Cap. %	Funo. Cap. %	Emphysema	Dyspnoea	Cyanosis	Pulse Range	Pulse Aver	Blood Pressure Aver.	Blood Pressure Range	Ven. Press cm H_2O	Valv. Invol	Rhythm	Enlarg	Aorti-tis	RBC mil-lion	Hb. % or Gm.	Wass erman	Calcium mg./100 cc.	Phos. mg.per 100 cc. blood
202988	+	+	62	M	-	-	-	IIA	+	+	+	-	100	140/90	-	-	Rel. mitral Insuff	-	0	+	4.9	13	0	-	-
202624	Slight	-	57	M	-	-	-	III	0	++	0	64-120	90	126/90 130/100	104/72-	-	Rel. mit.& trious insuff	Proto-dias- tolic gallop	3 II 4: 4III 6: 5 IV 9: V 15: VI 15:	0	4.85	15.4	0	-	-
99506	+	-	64	M	21 mos.	-	30.7	III	0	+	+	28-118	90	128/80	150/110	14	Mit. insuff	Aur. fib.	-	+	6.5	13.8	0	-	-
95472	+	-	67	F	-	-	-	IIB	0	+	0	69-112	86	164/80	180/90	-	Rel.mit insuff	Aur. fib.	+	+	4.25	10.3	0	11.8 10.6	4.3 3.2
																	Rel.mit (Aur. Fibr.)								

Associated Pathology or Additional Diagnoses	Additional Observations	Laboratory data of Significance	Past Medical History
202988 None			
202624 Generalized anasarca, bilateral hydrothorax, Benign prostatic hypertrophy, and myocardial fibrosis.	Subicteric tint Generalized pallor Clubbed fingers observed bystudent, not referred	NPN 45,52,43, PSP 75 Total protein 5.2; 5.1 Albumin 4.5, Globulin .7;3.9 AG 6.4; 3.2. Urea Clearance 68%, Mantoux.01,++	
99506 Bilateral hydrothorax; no organisms cultured from it. Rt. hydrocele. Rheumatoid arthritis 6 years previously.		Albumen cleared up PSP 60%	
95472 Peripheral sclerosis, Hypertrophic arthritis of dorsal spine. Cystocele, rectocele, procedentia uteri.	Trophic changes in skin. Chronic osteoporosis No free acid in stomach ECG showed temporary spasm or infarction		Menopause 23 yrs. before. Measles, whooping cough ch. Typhoid age 63
97793 Generalized arteriosclerosis, kyphoscoliosis. Atrophic arthritis. Bilateral Dupuytren's contracture. Hypertrophic arthritis Left indirect inguinal hernia.			
98004 Generalized arteriosclerosis.	Family history. Father died 70 of heart trouble.	PSP 40%	Abd. gyn. surgery. Abd. surgery for adhesions. Stroke 2 yrs. previously.
98816 Toxic adenoma of thyroid. Chronic cholecystitis. Chorio-retinitis of left eye. Osteoarthritis of spine.	8-26 & 10-20-37 cyanosis of nail beds. 1-4-38 watch crystal nails and clubbing.	BMR-8-27-37 +30 10-21-37 +22 1-5-38 +25. PSP 65%	

Associated Pathology or Additional Diagnoses	Additional Observations	Laboratory data of Significance	Past Medical History
63202 Peripheral sclerosis. Myocardial degeneration. Hypertension. Atony of colon. Secondary anemia.	*Dense opacities of both lung bases;had had broncho-pneumonia 3 weeks previously,but clubbing had been noticed previously.	Aspiration of fluid produced cough reflex Esophageal varices at orifice by bronchoscopy.	Trachoma;no clubbing 3 yrs.previously. Hypertension. Late involutional neurosis.
98981 Moderate myocardial fibrosis. Possible cardiac asthma Onychogryphosis great toe nails.	*Healed tuberculosis,right apical scar,increased hilar markings on rt.Healed many yrs.ago. Clubbing was noticed only recently.	No B.tuberculosis in sputum or gastric contents.	
75992 Chronic alcoholic addiction.	(Record incomplete, no summary)		
85858 Arteriosclerotic gangrene of rt.great toe; dental caries;pyorrhea alveolaris. Benign prostatic hypertrophy. Fibroma left forearm.			
80716 Myocardial fibrosis.Corneal opacities, Chronic conjunctivitis. Recent occlusion. Chronic orchitis.Nasal obstruction. Generalized arteriosclerosis. Middle ear deafness. Non-toxic adenoma of thyroid	No edema	Urine: Albumin .06%, Hyaline casts.	
84016 Secondary anemia.Achlorhydria. Benign prostatic hypertrophy. Alveolar abscesses. Pyorrhea alveolaris.		PSP 85%	

*Lung findings not thought of relevant nature if history can be believed.

Associated Pathology or Additional Diagnoses	Additional Observations	Laboratory Data of significance	Past Medical History
80966 None			
95303 Chronic passive congestion of lungs. Ascites. Right hydrothorax. Obesity. Generalized arteriosclerosis.	Bilateral hydrocele and Bilat.inguinal hernia Arteriosclerotic nephrosclerosis	Wood workers and common laborer.	
86484 Essential hypertension. Myocardial degeneration.Dilatation of aorta.	34 lb.wt.loss in 1 year.Right side of body numb for 10 days 3 yrs.before. Productive morning cough;chest plate negative. ECG consistent with early myocardial or hypertensive changes.	NPN 42 PSP 65	A.M.alcohol consumption.Duration ? When ?
80828 Thrombosis of left lenticulo-striate artery with hemiplegia and dysarthria Hypertrophic arthritis of spine.	Chronic cervicitis,bilateral sacro-iliac arthritis.	.05% albumin	Stroke 4 yrs. previously.Influenza & pneumonia not dated
81122 Myocardial degeneration. Anasarca. Generalized arteriosclerosis	1 2 3 4 5 Paracenteses of vol.cc.3600 950 2000 1300 1800 Venesection Sp. Gr.1017 1015 500 cc.ECG partial A-V block Blood:Color index .7	Urine Alb.06% 1-10 hyaline casts per HPF 4-8 granular casts per HPF	Acute & recurrent rheumatic fever 20 years before,without heart lesions.Influenza,recurrent. Brother died 58 of dropsy.

Hypertensive Heart Disease

Associated Pathology or Additional Diagnoses	Additional Observations	Laboratory data of Significance	Past Medical History
64016			
Myocardial degeneration Generalized arteriosclerosis. Right indirect inguinal hernia Varicosities of lower extremities. Coronary sclerosis.	Orthodiagram: flattening of left auricular salient. Spinal fluid sugar 68.2 Blood sugar 93	Post Mortem Examination: Cardiac hypertrophy and dilatation. Coronary and aortic sclerosis. L.coronary opening almost closed. Syphilitic aortitis. Mitral valve:Marked fibrosis, with small injected area containing nodule of 1 mm. diameter. *	Syphilitic Heart Disease.
55019			
Myocardial degeneration Chronic passive congestion lungs and liver. Beginning arteriosclerosis. Dental caries.	2nd Admission: Coronary occlusion,5 months after previous discharge. Terminal. No post mortem examination		

*Post mortem findings continued:
Aortic valve:Marked thickening and widening.
Myocardium:Chronic fibrous myocarditis.
Indeterminate acute endocarditis
Extreme chronic passive congestion of liver, spleen and kidneys
Pulmonary congestion and edema
Bronchial pneumonia
Chronic cholecystitis.

B. Circulatory Diseases.

1. to 6. given in Table II, p. 17.

The types of cardiac disease under six headings by etiology does not comprise the great variety of primary conditions possible by taking various lesions given in Table ~~II~~, as they occur singly or in the possible combinations. For instance, ~~the asterisks on Table II labeled French indicate the following:~~ ~~In~~ in the differential diagnosis of enlargement of the heart in children, French finds clubbed fingers useful in distinguishing the morbus caeruleus of pulmonary stenosis combined with patent interventricular septum with clubbing from either patent ductus arteriosus alone or pure pulmonary stenosis alone without clubbing. Also he grades up the incidence of clubbing in pulmonary stenosis as favoring its diagnosis in cases with precordial thrill over either patent ductus arteriosus or intraventricular septal defect alone.

Other lesions are:

Fatty heart - Brooks

Cor pulmonale - Harrison

Brown atrophy of heart - Brooks.

Extracardiac:	Authority
Vascular:	
Systemic	
7. Aneurysms	
←Unilateral	
Aortic	Lovibond
Aorta and its branches.	Shaw, Canton, Ogle, Smith, Osler, Beclere, Groedel.
Subclavian	Carr, Recklinghausen (Cotton)
Axillary	Brooks
←Bilateral: subclavian	Gay (Campbell)
8. Acrocyanosis	Planas, Bricall, Pang.
Pulmonary	
Arteriosclerosis (?)	

9. Ayerza's Disease (N.B. see etiology) — Weber, Paine and Platt. Landis

Hematopoietic.

10. Polycythemia Erythemia — Carr. Cushing

11. Purpura — Mangelsdorf, quoted by Shaw.

C. Diseases of the Alimentary Tract.

1. Liver abscess

2. Hypertrophic biliary cirrhosis

3. Chronic enteritis

4. Chronic jaundice

5. Sprue

6. Amoebic dysentery. (Intestinal amoebiasis)

7. Nonspecific ulcerative colitis

8. Amoebic hepatitis

9. Cicatricial pyloric stenosis.

10. Splenomegalic cirrhosis.

11. Laennec's cirrhosis

(One case of Laennec's cirrhosis was not entirely uncomplicated. There were multiple subcutaneous hemangiomata, multiple lymphangi-endotheliomata of the intestinal tract and multiple polypi of the stomach undergoing malignant changes; associated with vascular sclerosis and cirrhosis of the liver. The lungs, post mortem, showed sclerosis of the pulmonary arteries in all sizes of their branches and pleuritic adhesions. The heart was normal.) [Winternitz and Boggs]

12. Inflammatory stricture of the rectum and infectious arthritis.

13. Polyposis of the colon.

Colonic polyposis.

[19, 20, 21. are omitted by misnumbering]

14. Hydatid (echinococcus) cysts of the liver

15. Cavernous angioma of the tongue

16. Obstructive jaundice

17. Carcinoma of the esophagus

18. Dysentery

19. Carcinoma of the stomach.

D. Miscellaneous.

1. Abscess of salivary gland.

(The case of abscess of the axilla and salivary gland which Locke had in this group was deleted because of the presence of pulmonary tuberculosis).

2. Carcinoma of breast, neck and larynx.

(The extent of this lesion raises the question of lung metastases, in view of which a pulmonary mechanism in etiology is more likely).

3. Lues, congenital, with jaundice.

4. Polyuria.

(French, in discussing albuminuria points out the differential value of clubbed fingers in establishing the albuminuria as due to bronchiectasis with fibroid lung).

5. Post-thyroidectomy myxedema.

6. Pyonephritis.

7. Alcoholism

8. Malaria
Malarial cachexia.

9. Enlargement of spleen.

10. Chronic edema of nephrotic type and chronic gastro-enteritis.

11. Pyelonephrosis, Pyelonephritis .

12. Cystitis

13. Leprosy

14. Rickets

15. Influenza

16. Chronic nephritis.

8 (The report of Higier does not indicate clubbed fingers nor give x-ray evidence of subperiosteal new bone formation. His complete title indicates the use of osteoarthropathy as a general and not a specific term).

17. Avitaminosis.

E. Diseases of the Central Nervous System.

1. Syringomyelia

2. Neuritis.

F. Environmental Conditions.

1. High altitudes

G. Congenital ("Primary", "No primary lesion demonstrable").

of congenital clubbing
Cases have been reported by Brooks (Case V); Crouzon and Gutman; Campbell; Sacasa and Camp; Decloux and Lipplman; von Eiselberg; Fraentzel; Freund; Freytag; Horsfall; Israelski and Pollak; Kayn; Lowry; Locke; Mangelsdorf; Mendlowitz; Neurath; Newton and Mercellis; Odassky and Schirsner; Ragins and Frelich; Seaton; Weber; West; Witherspoon; and others.

The case reports on some of these examples have been stripped to the pertinent positive findings. Brooks found no evidence of definite or primary pulmonary or mediastinal lesion in a 24 year old Russian laborer. Campbell, Sacasa and Camp found no apparent cause for pronounced clubbing and considered the patient's appearance acromegalic-like. Freund's case, male, 57, was emphysematous and plethoric. In Horsfall's first case he found no sufficient cause for clubbing, although the patient had cirrhosis producing ascites and bleeding esophageal varices, a palpable spleen, red blood cells of 1,48 million, hemoglobin 20%, left ventricular preponderance by electrocardiogram, pleural thickening at right base, mild thickening of the larger bronchi. In this case a speculative and theoretically possible hidden common pathology might be found in tricuspid insufficiency which Vaquez associates with clubbing and which Moschcowitz thinks is associated with cirrhosis. The size of the left heart probably points to a mitral insufficiency which Vosquil indicates in 91% of clubbing in rheumatic heart disease. Horsfall does not give a negative rheumatic history for this case.

In Horsfall's second case he found no sufficient cause for clubbing in a 56 year old Italian laborer who had mild chronic pharyngitis, moderate pleural thickening at left base, early peripheral arteriosclerosis, moderate left ventricle hypertrophy (by orthodiagram?), chronic rheumatoid arthritis of right wrist, elbows, knees and ankles, left ventricular preponderance by

electrocardiogram, and 5,580,000 red cells.

Horsfall's third, fourth and fifth cases, descendents of case two, had no gross pathology evident. Horsfall's sixth case had a blood pressure of 120/60.

Israelski and Pollack had a case with a right sided abdominal tumor mass without any of the usual forms of primary disease. Kayn's case had 5,200,000 red cells, no other counts were exhibited, and whether this count was ever higher is not stated. Mendlowitz presented a case of congenital clubbing in which there was essential hypertension. Odassky and Schirsner's patient had right sided catarrhal pneumonia and cardiosclerosis with precordial pain, cardiac enlargement, dyspnea, palpitation and laboratory evidence in the form of sedimentation of red cells to the extent of 35 mm. per hour. They thought the "Generalized Ossifying Periostitis" accidental to the occurrence of lung disease and did not consider the cardiac condition. Ragins and Freilich give no detail of cardiac or pulmonary investigations stating only that the patient had mild gastro-intestinal complaints. Seaton's patient had blood pressure of 230/150 and left ventricular hypertrophy. Others in the family had chronic cough as well as clubbed fingers. Witherspoon found no abnormality in his 47 year old negro.

The cases of congenital clubbing reported have not yet been freed of suspicion of internal cause by reports of the autopsies on which basis Rentschler objects to Locke's five cases. Montuschi objects to Seaton's case not having an x-ray report, for in a similar case in which one of the usual lesions which cause clubbed fingers was not clinically apparent, he found a congenital lung cyst by x-ray.

Horsfall finds twenty cases of clubbing in three families in a year at Royal Victoria Hospital, Montreal. Though he does not find sufficient cause for clubbing in some of his cases in face of the familiar dictum, "The extent of the clubbing is not proportional to the seriousness of the primary lesion", he comments, "Despite its usual serious significance, clubbing of the fingers is not always a pathognomonic sign of severe visceral disease. Congenital clubbing is not the rare abnormality it has been considered, but is simply an unusual congenital characteristic which has no indirect diagnostic or prognostic importance." His comment gives added stimulus to careful consideration of finding a common mechanism causing clubbing. On the basis of his findings, the observation in older patients of only isolated instances of clubbed fingers in conditions similar to those which frequently cause clubbed fingers in younger individuals, requires that a congenital incidence be ruled out.

CLINICAL SIGNIFICANCE

In some types of disease clubbed fingers are an aid in differential diagnosis. In discussing enlargement of the heart and morbus caeruleus, French points out that when due to patent interventricular septum and pulmonary stenosis, clubbed fingers are present but that no, or less frequent, clubbing is found when it is due to patent ductus arteriosus alone, and pulmonary stenosis alone. French says that with precordial thrills in the pulmonary area, the presence of clubbed fingers favors pulmonary stenosis alone, over either patent ductus arteriosus or interventricular septal defect alone. The ancient pathognomonic import of clubbing is lost in the diversity of conditions in which it is found, its doleful prognosis is usually given later than many more readily available and more accurate modern diagnostic aids, and it is often

averted by modern therapy.

Once a pathognomonic sign, then a medical curiosity, the phenomenon now is a problem of etiology weighted heavily by the great disturbance of physiology it represents which is concurrently taking its toll in other tissues.

ETIOLOGY

The great variety of the previously enumerated primary conditions in which clubbed fingers has been found has put a great strain on the tenability of theories of various simple, or a single common, pathological processes or physiological aberrations to which the pathogenesis of clubbed fingers could be ascribed.

Before discussing the specific theories of etiology, one looks for general predisposing factors conducive to clubbed fingers. To many investigators the question has come, why does not one individual develop these proliferative changes when his condition is just as acute in the factors which seem to be conducive to clubbing, as another whose finger tips are bulbous. The factor of individual susceptibility seems to be strongly represented, and in very few modern reports do a majority of patients exhibit clubbing, no matter which one of these diseases afflicts them. Race, age and susceptibility to certain diseases are others which seem likely.

Brooks found that in 43 cases of osteoarthropathy the average age of onset was 17.55 years. The average age of onset in tuberculous patients was 29.6 years. In neoplasm of lung cases it was 40.5 years. Excluding tuberculous and neoplastic disease the average age was 12.67 years which, he says, "would then place the disease as definitely of juvenile origin." Kennedy

presented a case of acropachy in a $7\frac{1}{2}$ months infant. Brooks also calls attention to the fact of its occurrence in what he calls proliferative diseases characterized, as are tuberculosis and neoplasms, by tissue hyperplasia.

The role of race is hard to estimate in determining the likelihood of acropachy to develop, but we can point to the few cases reported and the exultation with which Witherspoon greeted what he called the first case of congenital clubbed fingers in a negro. So that it seems that they usually are not heir to it. Which nationalities of the white race are more susceptible seems reflected again, like the great white plagues on North Sea Countries.

The following theories have been advanced as concisely summarized by the Comperes and Adams in 1935.

1. Marie, Bamberger and Symes-Thompson thought that a toxin was produced, probably by bacterial decomposition and absorbed into the blood stream to produce the changes in the tissues characteristic of the disease.
2. Thayer considered it part of the picture of amyloid degeneration.
3. Massalongo believed that it occurred only in cases of arthritic diathesis and in this he was supported by Symes-Thompson.
4. Thorburn called attention to the striking similarity between the periosteal new bone formation in osteoarthropathy and that which may be seen along the shafts of bones adjacent to tuberculous joints. He pointed out that post mortem examinations frequently confirmed the diagnosis of tuberculosis in patients showing these peripheral changes where tuberculosis had not been found in the course of clinical examinations, and, on a

basis of these findings, he advanced the theory that all cases of osteoarthropathy were a form of tuberculosis which affected principally the long bones and the joints.

5. Berent suggested that the syndrome was due to a nerve disease and compared it to changes which are sometimes noted in syringomyelia.

6. Davis, Brooks, Kessel, Hyman and Herrick, Bryan and Stephens have offered arguments and some evidence to support the theory that the changes in osteoarthropathy may be explained entirely on a mechanical factor by blood stasis. Campbell states, "clubbing of the fingers is the result of defective oxidation in the tissues of the extremities, whether produced mechanically by obstruction of venous return, or as the result of a general lowering of the oxygen tension of the blood affecting parts of the body where normally the circulation is slow.

7. Bryan, Kleinberg, Marcus and Phemister have expressed the opinion that at least two factors were necessary to produce hypertrophic pulmonary osteoarthropathy: First, a toxemia from a long standing disease in the chest and, second, circulatory disturbances resulting from either cardiac or pulmonary involvement.

8. Harter thought that the condition was due to lack of oxygen and called attention to the somewhat similar changes found living in high altitudes where oxygen is rare.

In addition to this compilation may be added:

9. Witherspoon contends that there is a congenital diathesis which may be evident at birth or appear later. ~~(Described by Baker).~~

10. Elevation of the periosteum by release of nitrogen gas (Described by Baker) is mentioned by Lorio in discussing the presentation of a case of hypertrophic pulmonary osteoarthropathy by Williams.

In the following discussion additional comment is made concerning some of the theories so that some may be disposed of and that others may be combined, and also to point out that some one or more common fundamental conditions may be produced by the various mechanisms.

Regarding the toxic theory the Comperes and Admas say, "The concomitant occurrence of infection and usually of suppuration in the thorax has led to the theory that absorbed toxins were at least in part responsible for the more peripheral changes in the bones and soft tissues. Reports of cases with well advanced hypertrophic osteoarthropathy secondary to malignant growths such as sarcoma or carcinoma of the lung without abscess formation or other chronic infections discredits the toxin theory."

Thomas' questions are also asked in a manner suggesting a negative answer: "Why, for instance, should an abscess of the lungs produce such a toxin and abscesses elsewhere in the body not produce it? Why should it occur in its most advanced form in Hodgkin's disease which involves the glands of the mediastinum, and not occur with Hodgkin's disease confined to glands elsewhere in the body? Why should it occur with carcinoma of a bronchus and not with carcinoma in other parts of the body? Why should this toxin be liberated by pulmonary tuberculosis and not by tuberculosis of other

organs? Others, e.g. Paterson, think that toxemia is effective in some cases, and not in others. Shaw gives this summary: "Beclere has modified the toxic conception of the causation of clubbing: instead of adopting the view that diseases of the lung whether primary or secondary to heart disease, or of such organs as the liver, produce a toxin which causes clubbing, he considers that venous blood maturally contains substances which provoke changes in the fingers, and that if during its passage through the lungs this substance be not removed from the blood, clubbing results."

No one has yet identified the toxic substance nor described the mechanism of its action. However, the substances which the lungs are supposed to clear, CO_2 and O_2, are not ruled out. Concerning the toxicity of excess of CO_2 and deficiency of O_2, there is no question except in the matter of specificity in the production of the lesions of hypertrophic pulmonary osteoarthropathy. It is Campbell's view that, "it is unnecessary to invoke the conception of a special toxin, unless the metabolites produced in the extremities under conditions of imperfect oxidation can be regarded in this light."

Thayer's, Massalongo's and Thorburn's disciples have been neither recent, numerous nor particularly vocal, no doubt because of the limited applicability of their theories in view of the wide variety of primary conditions since enumerated. Berent, Sahli, Hirschfeld and others have not had nerve changes corroborated by such investigators as Alexander and Buzzard, nor have trophic changes been described in clubbed fingers of the order produced by local neurotrophic hypo- or hypersthenicity.

Mechanical blood stasis is dramatically affirmed by Thomas' example of acropachy in a case of hypothyroidism with myxedema following sub-total

thyroidectomy of a toxic adenoma. He rules out an acromegalic hook-up* on an interglandular basis and noted a decrease in new bone formation with administration of thyroid substance. "In my case, he writes, as in those presented by Högler, a persistent edema-like swelling of the lower extremities is strong evidence in favor of local congestion. One is led to wonder whether the common factor in all of these cases may not be some change in the circulation. Such a change occurs in cases of jaundice with bradycardia and might also occur if a fraction of the lung substance ceased to act properly in its function of aerating the blood. Mechanical pressure from a mediastinal mass does interfere with the blood flow in the great vessels and the disappearance of the bone changes in such a case following deep roentgen therapy to the mediastinum may be accounted for by the removal of such pressure. It would seem that the circulatory change must be abrupt, and that its effect is most striking when it occurs in patients whose osseous system is still in the process of adolescent growth." Then, after raising the questions damaging to the toxic theory, he also raises the logical objections to the theory of an altered blood flow, though he does not load a very heavy charge against them. "Why does one not see acropachy in the many cases of unilateral thrombophlebitis? So far as I know, subperiosteal new bone may occur in these cases without ever having been noticed. If an insufficient oxygen supply is an important feature, why has acropachy not been found in long-standing cases of severe anemia? It may be, I think, that the tissue adjustment to anemia is gradual, whereas in acropachy time for adjustment is lacking."

*No involvement of the pituitary gland was demonstrable by roentgenological examination of the sella turcica, by determination of the visual fields, or by sugar tolerance tests. A skeletal deformity quite different from acromegaly was encountered. He also considers the possibility of thymus and parathyroid involvement and rules them out.

In some cases of pronounced secondary anemia in patients with pulmonary tuberculosis, clubbed fingers occur (Dr. Coon's clinical demonstrations to fourth year medical students at Wisconsin State Tuberculosis Sanatorium). Holman's series has some cases which are not exactly plethoric, e.g. Case 7505 with moderate clubbing in a tuberculous female age 18 with 2,350,000 red cells and 25% hemoglobin. Though this is mentioned perhaps too irrelevantly emphasizes the observation that anemia does not preclude clubbing in the presence of one of the conditions favoring incomplete aeration of the blood.

The common association and slight excess over that in similar age groups of secondary pulmonary arteriosclerosis in chronic lung and cardiac disease is pointed out by Brenner. Rather than ascribe the clubbing of the fingers to the secondary, he attributes their pathogenesis to the primary and the chronic cyanosis. He does not tell of clubbed fingers in two cases which he considered primary arteriosclerosis of the pulmonary arteries.

The description of periosteum elevation by nitrogen gas was not discovered and examined, nor has other comment been found. This theory can probably be included with the stasis theory along with the toxic theory and toxin combined with stasis theory, for the physical, toxic, physiologic and chemical mechanisms of the stasis theory has not been explained. (over, please) Text book opinion likewise has been altered as reflected in Norris and Landis'. In the 1938 edition is the statement, "clubbing of the fingers indicates long-standing stasis of the pulmonary circulation resulting from chronic cardiac or pulmonary disease." Whereas in the 1936 edition it was said, "the most probable cause seems to be that the changes are in some way dependent on toxic absorption." The modern theory of stasis (which Campbell dates to Pigeaux,

The opinions regarding congenital occurence are largely uncritical, and the presence of hidden lesions awaits the verdict of autopsy. Discussion was given in the section on "Incidence". For instance, as Vaquez' observ~~ation of~~ functional tricuspid insufficiency may be one of the hidden lesions. Moschowitz has found at autopsy that cirrhosis of the liver is associated with the same lesion, which might be found in Horsfall's first case.

1832) yet requires plausible explanation of the physiochemical reaction in the tissues affected.

EXPERIMENTAL STUDIES

Herz produced clubbed fingers by producing experimental widening of the capillaries of the nail beds ~~in two cases which he considered primary arteriosclerosis of the pulmonary arteries.~~ but this does not elucidate etiology, though the result simulates the stage of capillary dilatation in pathogenesis. The Comperes and Adams give this summary of the experimental work previous to theirs:

> "Attempts to produce in experimental animals changes characteristic of the clinical syndrome of hypertrophic osteoarthropathy have been uniformly unsuccessful.
>
> Bamberger, believing a toxin to be the cause of the bone changes injected into the rectum of three young rabbits twice daily a bronchiectatic secretion from one of his patients, but did not produce any of the changes.
>
> Phemister injected cultures of various organisms including streptococcus viridans and streptococcus hemolyticus into rabbits for a period of six weeks without producing any bone changes.
>
> Stephens tried to support his theory that all of the changes noted could be explained on the basis of peripheral blood stasis. He produced mechanical interference with the venous flow of blood in dogs, creating a venous congestion in the extremities, but was not successful in producing a proliferative periostitis.
>
> Harter and Churchill sought to produce the changes by ligating and sectioning the bronchus in cats and monkeys and in other ways tried to create a lowered oxygen content in the peripheral blood, but they were not successful in producing any changes in the bones, nails, or capillaries of the nail beds."

The Camperes and Adams were unable to produce hypertrophic pulmonary osteoarthropathy in dogs: (1) "By pressure upon a lung from a foreign body in the pleural cavity, (2) stenosis of a primary or secondary bronchus, (3) collapse or total absence of a lobe or of an entire lung, (4) pleurisy with

effusion, (5) empyema and solitary lung abscess." They say, "In the human subject any of the above intrathoracic complication might be expected to produce respiratory embarrassment with varying degrees of dyspnoea, cyanosis and venous engorgement in the tissues most distant from the heart. None of these changes were noted in the experimental animals which seemed to be able to compensate for loss of functioning lung to a surprising degree, even when forced to vigorous exercises, such as continuous swimming for an hour or longer." They report two terminal cases with proliferative bone changes, one of blastomycosis of the chest wall with secondary infection and empyema, the other of carcinoma of the lung with involvement of the bronchi and larger blood vessels but with no intrathoracic suppuration. The conditions common to both cases were dyspnoea and cyanosis, and a disturbance of the acid base equilibrium of the peripheral blood. Their findings and running comment are interesting though no comparable figures are available and most investigators have found the blood levels of calcium and inorganic phosphates normal and did not report CO_2 levels nor pH.

In the first case, "blood chemistry studies were made at intervals from March 9, 1929 to March 21, 1929, the day before death occurred, and these showed blood calcium from 7.9 to 8.5 mgs. per cent, figures which are at least twenty per cent below normal for an adult, and inorganic phosphates of 3.69 to 4.42 milligrams per cent, slightly higher than the normal adult average. There was a slight increase in the carbon dioxide content from 66.9 per cent to 71.16 per cent and a corresponding shift of the hydrogen-ion concentration toward the acid phase from an initial reading of 7.62 (definitely more alkaline than normal) to 7.49. During the period of this blood study, the patient was dyspnoeic but there was no hyperpnea and the

changes from normal indicate that there was inadequate aeration of the blood and hence accumulation of carbon dioxide in the tissues in spite of the fact that the patient was kept at rest. This lack of adequate oxygen intake and accumulation of acid products in the blood and tissues has been suggested as the etiological factor in the production of the bone changes. Renal function was not seriously impaired and the nonprotein nitrogen, urea nitrogen and blood chlorides were normal, rising only slightly on the day preceding death, March 21, 1929".

In the second case, "blood chemistry studies were made. Changes from normal were similar to those noted in Case I. Blood calcium was 6.75 and inorganic phosphates were 5.29. The carbon dioxide content was definitely increased to 67 per cent and the hydrogen-ion concentration was alkaline, 7.61. The basal metabolic rate was +41, but since the body temperature was 100 to 101 degrees, this was discounted. Respirations averaged 30 per minute and during the last two days these increased to 40 per minute."

Investigations into the pathological physiology of clubbed fingers have been meager and inconclusive. Mendlowitz investigated disturbances in the circulation of the distal phalanx by means of thermoelectric measurement of skin temperature, heat elimination from the finger tip, digital arterial pressure and brachial-digital arterial gradient. From the latter three he estimated change in blood flow. Besides groups of normals, sick patients without clubbed fingers, ~~and~~ sick patients with clubbed fingers, and two congenital cases of clubbed fingers (one of whom had benign essential hypertension), he studied four cases of unilateral clubbing (one of which had a pulmonary basis for its development with acceleration of the process on the right so that it was recognizable as being due to right upper lobe bronchus carcinoma

which had caused pathological sympathectomy). The summary of Mendlowitz's results is:

1. "The response of the blood vessels in clubbed fingers to environmental temperature changes is qualitatively normal, and the maximum heat eliminations of the hands of patients with clubbed fingers are within normal limits.
2. The maximum heat elimination and hence the blood flow of the distal phalanges of clubbed fingers secondary to lung or congenital heart disease is usually increased." (It was not measured in congenital clubbed fingers).
3. The digital arterial pressure is increased and the brachail digital arterial gradient decreased in clubbing secondary to lung or congenital heart disease. In hereditary clubbing these gradients and pressures are normal.
4. The blood flow of the unilaterally clubbed finger tip, as indicated by maximum heat elimination may be increased or decreased. No significant change was found in the blood pressure gradient except for a bilateral decrease in a case interpreted to be bilateral clubbing with acceleration of the process in the fingers of one hand due to a sympathetic nerve lesion."

Mendlowitz's findings are somewhat confused by inclusion of hereditary clubbed fingers. With the normal mechanism of changing peripheral blood intact (Summary "1" & "2" together) this may be taken to mean that the distal phalanx presents the variation in response to the underlying pathology, producing secondary clubbing, if, for instance, it be local anoxemia arising from chronic stasis,

acting on the peripheral circulation. Wilkins, Doupe and Newman state that most of the change in flow, as well as quantity of flow" in a finger (as reflected in the change in volume) is in the distal phalanx. It is probably not great enough even with clubbing to increase the heat elimination for the whole hand appreciably. ~~(Summary 1 and 2).~~ Summary 2 may be taken to represent dilatation of the vessels in the distal phalanx. The findings given in Summary 3 may have this interpretation. There may be a hidden pathology in the constitution of the individual with congenital clubbed fingers for which there has been compensation with a return toward normal. The underlying pathology producing secondary clubbing may still be active so as to cause dilatation in the clubbed fingers. The local status may be indicated in the blood pressure determinations. Furthermore the local pathology in the congenital clubbed finger may not exactly simulate that of the secondary. The congenital might be in a fibrotic stage while the secondary might be in an edematous stage of development. He gives the following discussion of the indeterminate results (in 4 above) which, he says, are the same as in cases reported by Groedel, Loncaides and Ogle to whom he refers. "There are three possibilities for this difference between unilateral and bilateral clubbing. First, it is possible that the changes observed in the bilateral cases are due to chance and are not significant. The number of cases and the constancy of the results make this very unlikely. Second, it may be that the unilateral cases differ fundamentally in mechanism from the bilateral. Third, it is possible that the factors operating in unilateral and bilateral clubbing are initially the same, but that in unilateral cases complicating factors subsequently obscure the original ones. Some of these complicating factors are arterial obstruction by thrombi, venous and lymphatic obstruction, and nerve lesions. Only further work, especially on

the early course of these rare cases, can decide this issue."

PRESENTATION OF DATA

The case records of 1304 patients at Wisconsin General Hospital in which the primary diagnosis was coronary disease (arteriosclerotic heart disease), hypertensive heart disease, syphilitic heart disease, or atrophic (Laennec's) cirrhosis of the liver were examined for the observation of clubbed fingers. No case was found in which Ayerza's disease or primary sclerosis of the pulmonary arteries was diagnosed. One case of secondary sclerosis of the pulmonary arteries was diagnosed clinically, but clubbed fingers were not observed. If the observer were in doubt as to what he saw, it was not taken to be clubbed fingers. Duplicates which numbered 38 were carefully eliminated by comparison with a check list.

Thirty-two cases of hypertensive heart disease developed coronary disease (arteriosclerotic heart disease) as diagnosed on a later admission. Clubbed fingers were ascribed to the primary condition diagnosed at the time they were observed. If clubbed fingers had been observed, the complete diagnosis and history was checked for mention of some disease condition, or significant sign thereof, which is a primary condition more frequently associated with clubbed fingers, and if present the case was not included in this series unless the relationship was very remote, as with cases #98981 and #63202. No case of atrophic cirrhosis with clubbed fingers was found without such a complication. A list of such cases in each group is appended along with those in which only nail changes were noted.

Tabular presentation is made of the heart diseases in the section on incidence of clubbed fingers in heart disease where comparison is made

directly with Vosquil's and others results. The tabulated findings in the individual cases are appended along with the list of cases of clubbing discarded because of the presence of disease more likely to cause clubbing than the primary disease in these groups.

None of these cases was seen by the compilator. Coronary (arteriosclerotic heart disease) disease and hypertensive heart disease figures are compiled from the case records of Wisconsin General Hospital over the five year period, 1934 through 1938, inclusive. Syphilitic heart disease and atrophic cirrhosis of the liver figures are compiled from the case records over the period 1925-1938, inclusive.

Coronary Disease (Arteriosclerotic heart disease). In 843 cases, 14 were found to have clubbed fingers which is 1.6% of the total. In 8 the degree of clubbing was not indicated. In 5 it was slight. In 1 it was marked. In 4 cases clubbing of the toes was reported. In another gangrene was found. No mention of the toes was made in 9.

The average age was 60.7 years. The youngest was a male, aged 44. The oldest was a female, aged 75. 9, or 64%, were male and 5, or 36%, were female. The average age of the males was 57.7 years. The average age of the females was 66.2 years.

The duration of heart disease symptoms was not given in 9 cases. In four the duration averaged ten months. In one hypertensive heart disease had been diagnosed three years previously.

The onset of clubbing was noticed in three cases. The average age of onset was 54.3 years. The clubbing was noticed on an average of seventeen

months previous to the diagnosis of coronary disease.

Vital capacity was determined in only one case - 31.7%. Functional capacity was I in one case, four, or 29%, had functional capacity IIA, five or 36% had functional capacity IIB, two or 14% had functional capacity IIA-B and two or 14% had functional capacity III. Emphysema was present in nine, or 64%. Dyspnoea was complained of in eleven or 79%. Cyanosis was observed in eight, or 57%. The average pulse was 89. The range of individual averages was 59 to 109. The extreme range of pulse was 28 to 120. Average systolic blood pressure was 138 and average diastolic blood pressure was 88. The range of average systolic pressure was 96 to 180 and the range of diastolic pressure was 70 to 106. The average range of systolic pressure was 128 to150. The average extreme range of diastolic pressure was 82 to 98. The absolute range of systolic pressure was 96 to 228. The absolute range of diastolic pressure was 66 to 120. Pressure is mm. Hg. Venous pressure was 14 cms. of water in one case. In thirteen it was not taken.

Valvular involvement was diagnosed in 11 or 79%. In three, or 21%, it was not. Two cases had tricuspid insufficiency. Two cases had relative insufficiency of both the mitral and the tricuspid valves. Seven had relative mitral insufficiency, ~~and~~ Two had mitral insufficiency. Cardiac enlargement was present in eight, or 57%. Myocardial degeneration was present in five, or 36%. Rhythm disturbance was noted in six cases. One had tachycardia, two had auricular fibrillation, two had extrasystoles and one had proto-diastolic gallop rhythm. Aortitis was present in twelve, or 86%. No dilatation of the aorta was diagnosed. No history of attacks of rheumatic fever was given. Fever was not present in any case. The average red blood cell count was 4,920,000 and the range was 3,880,000 to 6,520,000. The average

hemoglobin in eight was 14 grams, the range was 10.35 to 16.8 grams. The average hemoglobin in six was 68 per cent, the range was 58 to 88 per cent.

The blood Wassermann reaction was negative in all fourteen cases. Blood calcium in one patient was 11.2 It was not taken in thirteen. Blood inorganic phosphates were 3.8 in the same case. They were not determined in thirteen. No cases died under observation.

Hypertensive Heart Disease. Clubbed fingers were observed in four, or 1.2% of 313 patients. On one case the clubbing was called slight. In three the degree of clubbing was not designated. In none was clubbing of the toes mentioned. Three were male and one was female. The male averaged 52.6 years of age, and the women 52 years. The youngest was a man of 45 and the oldest a man of 67. The other man was 46. In the three men the duration of symptoms of heart disease was two years; the woman had symptoms for fourteen years.

In the man of 67 the onset of clubbing was set, with some question, at 65, two years before observed and at about the onset of symptoms. In the other three its onset was not noticed. Vital capacity in the 67 year old male was 47%. In the other three it was not taken. In one case functional capacity was set between I and IIA, in one case it was IIB and in two cases it was III. Emphysema was present in one case, male age 67. In three it was not considered present. Dyspnoea was present in four, or 100%. Cyanosis was present in three, or 75%; it was not considered present in the 52 year old woman. The average pulse was 86. The range of average pulse was 66 to 102. The extreme range of pulse was 48 to 124. The average systolic blood pressure was 188 and the average diastolic blood pressure was 114. The range of average systolic blood pressure was 182 to 194 and of diastolic

blood pressure was 100 to 130. The extreme range of systolic pressure was 154 to 222. The extreme range of diastolic blood pressure was 96 to 142.

Valvular involvement was present in two cases, or 50%. No lesion was diagnosed in two. In one relative mitral insufficiency was present. In the other relative insufficiency of both mitral and tricuspid valves occurred. Cardiac enlargement was present in four cases, or 100%. Myocardial degeneration was diagnosed in two. One case disturbance of rhythm consisted of auricular fibrillation at times and lapsed into complete heart block at others. Aortitis was present in four, or 100%. Dilatation of the aorta was present in one case. In one case acute and recurrent rheumatic fever had been diagnosed twenty years before and no heart lesions were ~~siad~~ said to be present them. The current valve lesions were diagnosed relative insufficiency, mitral and tricuspid. Red cells averaged 5,210,000. The range was from 4.5 to 6,460,000. Hemoglobin averaged 70% and the range was 55 to 80%. The Wassermann reaction was negative in four cases, or 100%. No cases died under observation.

Syphilitic heart disease. In eighty cases, two, or 2.5%, presented clubbed fingers. Clubbed toes were not mentioned. Both cases were male. Their ages were 46 and 49 years, average 47.5 years. The duration of the disease was 14 and 8 years, average 11. Onset of clubbing was not mentioned in either case. Vital capacity of one case was 2700 cc. Functional capacity in one case was IIA and in the other IIB. Emphysema was not observed. Dyspnoea was present in both cases. Cyahosis was present in both cases. The average pulse rate was 82, the range of average pulse was 80 to 84 and the extreme range of pulse was 46 to 120.

The average systolic blood pressure was 120, and diastolic, 56. The

average systolic pressure was 110 to 130. The range of average diastolic pressure was 50 to 62. The gross range of systolic pressure was 85 to 140 and diastolic 38 to 100. Venous pressure was 6 cm. of water in one case and 12 and 14 in the other. The average was 9.5

Valvular involvement was present in both cases. One case had relative insufficiency in the tricuspid and mitral valves. The other had relative insufficiency in the mitral valve. In both cases myocardial degeneration was present. In one case coronary occlusion was fatal on the second admission five months after the first discharge. The other had coronary sclerosis. In both cases enlargement of the heart was found. In one case lengthening of the left auricular salient was demonstrated by orthodiagram indicating left auricular dilatation. In both cases the heart was enlarged to the right. In one case auricular fibrillation developed into heart block and in the other no disturbance of rhythm was found.

Red cells averaged 5,480,000 and the range was 4,~~80~~91,000 to 5,660,000. Hemoglobin averaged 72% and the range was 55 to 80%. Both cases died under observation. Autopsy was done on one, confirming the diagnosis. A brief of the report is appended.

DISCUSSION

With this small incidence of clubbing, 1.2% to 1.6%, one might suspect that the element of chance in the form of a congenital factor were entering. However, considering how loosely many of that classification have been selected and pointing to the obvious signs of respiratory and cardiac distress in these cases, there is enough to make one think that the factors active in the other conditions operate also in these. If these diseases characterized by degenerative changes are considered as diseases of age and representing the lesions of wear and tear on the organism, the incidence of acropachy takes on more significance, for it illustrates the fact that an extremely powerful stimulus is being brought to bear on an organism not only not building up but taking definite steps toward falling apart.

The youngest case presented is 44 and the largest group's average age is 60.7 years. In the first place those susceptible to these changes may have already been claimed in youth. Again, the matter of susceptibility itself may have been much altered by the physiological age of these patients in whom proliferation, for example, bone mending, to which the bone changes have been likened, is very slow.

None of these groups is large enough from which to draw any statistical conclusions, but the incidence of the lesions upon the sexes seems to fall quite pat even in the start of this series. Men in their fifties, women in their sixties get coronary disease, 9 to 5. The hypertensives, 3 to 1, occupy a bracket just beneath then, close enough to reach up and take their place and the syphilitics, both male, come along as far again below. As diseases of wear and tear, nerve strain, and worldliness, the sex incidence is again typical. As cases of clubbing, too, they are struck less

equally than the younger cardiacs or the respiratory groups.

The history of cardiac symptoms as well as that of onset of clubbing is indefinite, reflecting not much more than the intelligence of the patient, and no correlation is attempted with the I.Q. No facetious remark, even with a lack of history will cover the fact of the exceptionally long cardiac history of patients with rheumatic heart disease or subacute bacterial endocarditis, with which these diseases must be compared.

The lack of vital capacity figures is very disconcerting. Not only is a study like this deficient in what the status of other non-clubbed finger patients, is which blocked Holman at this point, but even as Vosquil doubted the adequacy of observation of clubbed fingers, the limitations of the work done by medical students is shown by the paucity of vital capacity records. Those offered in these records are insufficient in number from which to draw conclusions.

Functional capacity is less variable in range than in the heart disease of younger groups. All have some encroachment upon their reserve, but only two sclerotics were fit to be propped up in bed whereas in equal proportion six rheumatics were.

Emphysema becomes apparent in the 60 year old cardiacs. In most cases it was labeled senile. In only one case it was not associated with dyspnea. Though it may be only chance the non-emphysematous were those with functional capacity of III. The effect of strenuous work may be shown here, or of an attempt to compensate for poor circulation, the deficient respiration abetting it now in producing more anoxemia and stasis to favor clubbing. Dyspnœa is prominent in higher average percentage than the younger car-

diacs and except for silicosis than the respiratory group. Less efficient exchange is represented in the alveoli and in the tissues as well as with the atmosphere as occasioned by emphysema. Cyanosis reflects again the failing compensation of respiratory and circulatory systems and on the average there is higher incidence than in any of the younger groups, even congenital. The resemblance in the levels of these last four factors as well as age, pulse and sex and the probable amount of sclerosis in the pulmonary vessels is borne out in the silicosis group. One would suspect the more frequent clubbing were developed earlier in the fibrotic lung group. Pulse is comparable to the others in all respects except perhaps its great range. Blood pressure is typical of the lesions and the age. That of the sclerotics compares with that of the silicotics again. Except for this group among the lung diseases where respiratory embarrassment is so great and the syphilitic males who are on the way out, this factor seems to be the main mechanism keeping up the compensation of peripheral blood flow among the older cardiacs. This factor may explain why an adequate rate of flow of not too anoxemic blood to the extremities prevents clubbing from developing in the rest of these patients.

The venous pressures obtained would again be more useful if there were a few more of the same to work with. The average level is not very remarkable even with a few.

The incidence of valvular involvement, particularly that associated with dilatation, is considerable in this group. All but five, 75%, have some involvement. Vaquez's association of clubbed fingers with relative tricuspid insufficiency is borne out. The incidence (30%) of a lesion in this valve is higher than in the younger cardiac group. Mitral insufficiency is the

most frequent lesion and is present in all cases with valvular involvement in this series. This frequency carries over into the younger cardiac group and even across into silicosis again where Holman noted it once. This lesion was, of course, also present in all cases with chronic passive congestion, ascites, hydrothorax or edema. It was the only valve lesion diagnosed in two such cases and was associated with aortic insufficiency or tricuspid insufficiency in the others.

Mitral stenosis occurred in only one case, that of syphilitic heart disease in which there was found considerable fibrosis and a nodular lesion [Yet the etiology of this lesion could not be luetic] at autopsy. Cotton and Locke argue for the importance of this lesion in clubbing. It is not to be expected as a lesion in this type of heart disease unless a complication, usually an earlier disease in a different category (which is rare) has occurred. It is, of course, prominent in the diseases producing endocarditis and bears out the contentions of Locke and Cotton with the much higher incidence of clubbing associated with them. This is probably another cardiac reason why clubbing is less frequent in this group of diseases.

Again, the acquired heart diseases, rarely and certainly not in this group, have the inducement to clubbing furnished by the congenital defects. #Enlargement seems to be less in this group (65%) than in the infectious group #but again comparison is hampered by the size of the sample. #Myocardial degeneration was present in the two cases each in the syphilitic and in the hypertensive categories as well as being diagnosed in 36% of the coronary disease cases. This is 45% of the total. With the incidence of coronary occlusion and other accidents peripherally this lesion again must be considered as cutting short the time in which circulatory inefficiency can operate

to produce clubbing.

In the infectious group there was about 12% of pericardial involvement which is much more common and the typical adhesive nature of the process is much more conducive to production of chronic circulatory embarrassment than the chance and transient effects of effusion which was not diagnosed in this series.

Though aortitis was present in a total of 85% of the cases in the older group, the only lesion of the aortic valve was stenotic in nature. In the infectious group insufficiency and stenosis were much more common. However, the size of the sample is against conclusions or flights of fancy and the significance of aortitis again militates against the chronicity which favors clubbing and also indicates an altered response in the individual.

The hypertensive case with the rheumatic history showed lesions in both sides of the heart consistent with dilatation not fibrosis, namely insufficiency of the tricuspid and mitral valve.

The blood studies show an average level of red cells exceeded only by congenital heart disease and a hemoglobin level in coronary disease exceeded only by silicosis and congenital heart disease. No more than two red cell studies were done on any one case, but no excessive erythropoiesis was shown. Furthermore, the blood was hypochromic in all cases. This does not show the response to anoxemia which the congenital cases do, nor those elusive cases of pulmonary vascular disease which are linked up with Ayerza's name.

The only positive blood Wassermann was in one of the cases of syphilitic heart disease. Specimens from the Wassermann negative case showed

typical syphilitic lesions in the aorta, but spirochetes were not found in the heart. Again the sampling is reflected upon for only 20% of syphilitic heart disease cases are supposed to be Wassermann negative. And also, larger series of cases reflect the experience at Wisconsin General Hospital of 1 to 2 per cent. Holman found 1.3 per cent in his tuberculous and bronchiectatic ~~and~~ clubbed finger cases.

Blood chemistry studies were insufficient and inconclusive. In Adams and the Comperes' studies the patients were moribund imminently or actually. The status of the blood content in cases in which clubbed fingers are developing or are established has yet to be determined. It is in such cases that a more relevant appraisal of the mechanisms of compensation for respiratory and circulatory embarrassment are yet to be made.

The speculation which arises in connection with description of periosteum elevation by nitrogen gas may be proposed as to whether one might not look for the development of acropachy in caisson workers.

SUMMARY

1. The incidence of clubbed fingers in 843 consecutive cases of coronary disease (arteriosclerotic heart disease) was found to be 1.6%, in 313 cases of hypertensive heart disease 1.2%, in 80 cases of syphilitic heart disease 2.5%.

2. Conditions in these diseases, compared with conditions found in diseases in which there is a more common association of clubbed fingers, which do not seem to favor clubbing of the fingers and toes were found to be: High blood pressure except perhaps in syphilitic heart disease; infrequent occurrence of mitral stenosis; the incidence of other diseases more likely to cause clubbing; the age of the patient; relatively rapid onset of symptoms; more general and rapid encroachment on cardiac reserve.

3. Conditions which seem to favor the development of clubbed fingers were found to be: Fairly high incidence of tricuspid insufficiency; perhaps, the almost universal incidence of mitral insufficiency in patients with valvular lesions; signs of chronic passive congestion; high incidence of dyspnoea, cyanosis and emphysema; and probable decrease of vital capacity and increase of sclerosis of pulmonary vessels.

APPENDIX A

I. Coronary Disease (arteriosclerotic heart disease).

Fourteen cases in which nail changes were noted:

	Degree or Description	Observer	Associated lesion
52405		Musser	
97967			
88392			
80702	Slight	Burns	
80361			
82024			
81685			
81833		Behr	
69752	Slight curving on admission	Finney	
69027		Burns	
80046		Burns	
69008		Burns	
71629		Lee	
84310	Slight	Brown.	

Nine cases in which clubbed fingers were observed, but, because of the presence of a more common primary condition associated with clubbing, were not included in this series, or because of doubtful identification:

Case Number	Associated Lesions
91545	Chronic sinusitis reactivated pulmonary tuberculosis
62547	Chronic bronchitis
90072	Silicosis
81185	Chronic bronchitis (silicosis debated) Marked emphysema
205237	Previous rheumatic heart disease, chronic adhesive pericarditis.
91953	Interlobar empyema found at autopsy
91534	Pleurisy with effusion in childhood.
89894	Chronic adhesive pericarditis
82187	Not agreed upon by various observers.

II. Hypertensive heart disease.

Six cases in which typical nail changes were noted.

Three cases in which atypical nail changes were noted.

Case Number	Degree or Description	Observer	Associated Lesion
200918	Downcurving	Allin	
20047	Curved	Baird	
90608	--	Ingersoll	
87076	Watch crystal nails 1936 (not observed in 1934,1935)	Gordon	
80675	Downcurving	Burns	
83937	--		Silicosis
90795	Slight spoon curving	Rein	
87471	Tendency toward spooning	Pohle	
66302	Spoonshaped	Midelfart	

Clubbed fingers rejected:

107621	--		Generalized peri-bronchitis of chronic bronchitis.
81122	Rheumatic history.		

III. Syphilitic heart disease.

Four cases in which nail changes were noted:

95248	Musser	
68601	Angus and Burns	
53206		Chronic empyema
64326		Met. carcinoma of gastric fundus. Old tuberculous scars. Old rheu. ht. lesions

1 case clubbed fingers, 64326, observer not convinced.

IV. Atrophic cirrhosis of the liver.

2[illegible] cases in which typical nail changes were noted.

Number	Associated Lesions
62854	
64840	
5 cases in which clubbed fingers were observed	
67369	Pleural and pericardial adhesions
66848	Coronary disease, rheumatic fever history, pleurisy, compensatory emphysema.
67329	Coronary disease with relative mitral insufficiency, chronic bronchitis, bronchial asthma.
92722 92722	Generalized arteriosclerosis, terminal peritonitis, pleural adhesions.
204234	Bronchial asthma, coronary disease, functional capacity IIA, enlargement, ascites, secondary anemia.

APPENDIX B

Data on individual cases presented.

Part I - common features taken for comparison, p. 55

Part II - Associated pathology or additional diagnoses, pp. 56, 57, 58, 59. additional observations, laboratory data of significance, past medical history, autopsy report.

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