Maude Elizabeth Seymour Abbott "She was an institution"





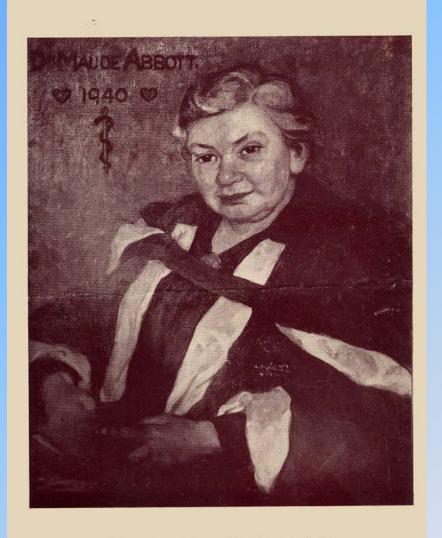
Born on March 18, 1869 at St Andrews, a small village on the north shore of the Ottawa River.

Higher Education for Women ??

In the minds of many if not most men, women were considered incapable of the increased mental strain which higher education demanded.

MAUDE ABBOTT

A Memoir



H. E. MacDERMOT

Her Early Steps

Class Valedictorian Winner of the Lord Stanley Gold Medal

But what about medicine??

Maude set her mind on McGill, and petitioned the Registrar who replied,
"I am sorry to inform you that the Faculty of Medicine can hold out no hope of being able to comply with your request."

Enlightened Opinions

Dr. F.J. Shepherd, "Introduction of women amongst medical students would be nothing short of a calamity."

Dr. G. E. Fenwick, Professor of Surgery, "I will resign if women are allowed to take the medical course.

Dr. F. W. Campbell argued that, "In the difficult work of surgery, they would not have the nerve. Can you think of a patient in a critical case, waiting while the medical lady fixes her bonnet or adjusts her bustle?"



Maude graduated from Bishop's in June 1894 with brilliant honors. Vienna was the medical Mecca, so she immediately planned her post graduate work in Europe. One of the greatest medical opportunities in Vienna was pathology with Kolisko and Albrecht, an experience that made possible Maude's later work.

And she traveled--London, Heidelberg, Zurich--exposing herself to the best minds in contemporary medicine. These years of study and travel were the happiest in her professional life.

But what Maude wanted above everything else was to be on the staff of the McGill Medical Faculty

Osler's Advice to Maude



"I wonder if you realize what an opportunity you have?
That McGill Museum is a great place."

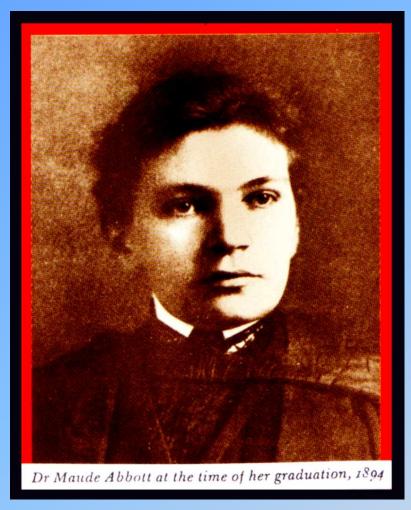
And so he gently dropped a seed that dominated all her future work

Hopeless Futilities

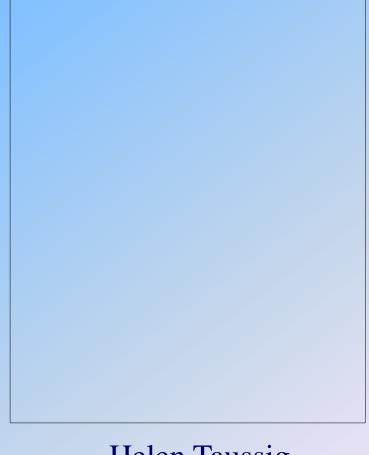
Before World War II, congenital malformations of the heart were regarded as hopeless futilities, an occupation appropriate for the few women in medicine. Maude Abbott was advised by William Osler to occupy herself with the collection of anatomic specimens at McGill, and Helen Taussig was advised to occupy herself with the hopeless futilities in the Harriet Lane children's clinic at Hopkins. Congenital heart disease in adults was an oxymoron.



The Two Few Women in Medicine



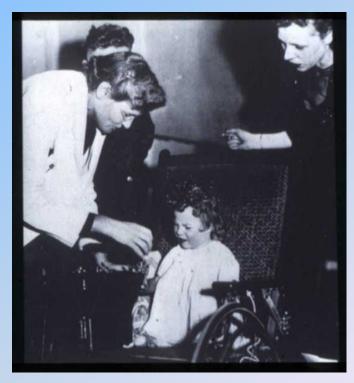
Maude Abbott

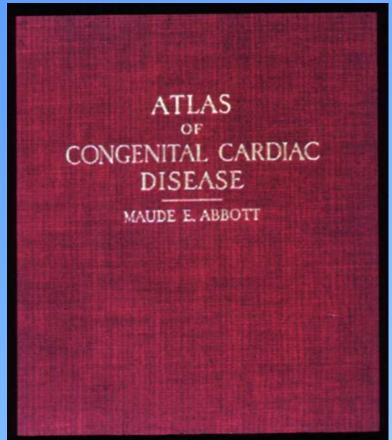


Helen Taussig

Maude and Helen







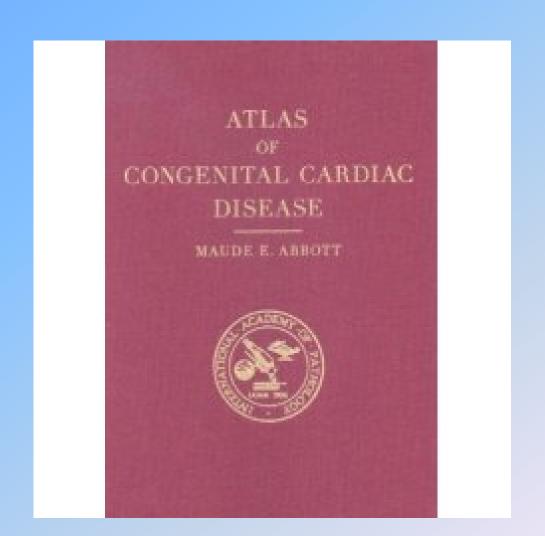
The Osler Library McGill

1936 1000 Cases





2006 Re-publication on the occasion of the 100th Anniversary of the International Association of Medical Museums, now called the International Academy of Pathology



THE SCHOOL OF MEDICINE

aug 27 1931

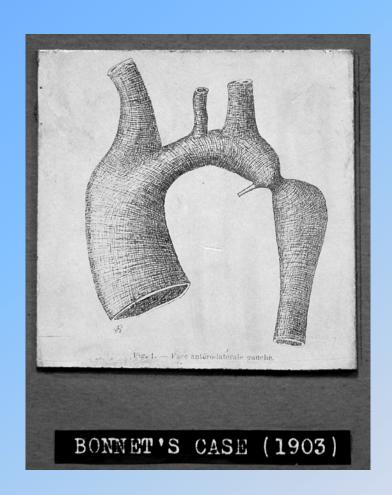
Dr mande E. abboll Ma gill Universely monheal, Canada.

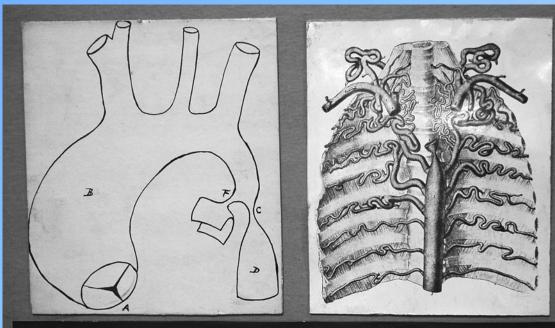
Dear Dr abbott :-

as you requested, Jam, sending you a reprint of my article "A Perastent Oslium alioventiculare commune with deptat Defects in a mongolian Ediat. I am also sending the heart reported in this article. This is being done at the request of Dr Elliott C Culter, Profeson of Surgery, Western Reserve University, Cheveland Ohio. The patient was his daughter and he wishes the heart to be added to the collection in your museum. I want to again thank you

for your advise about this core.

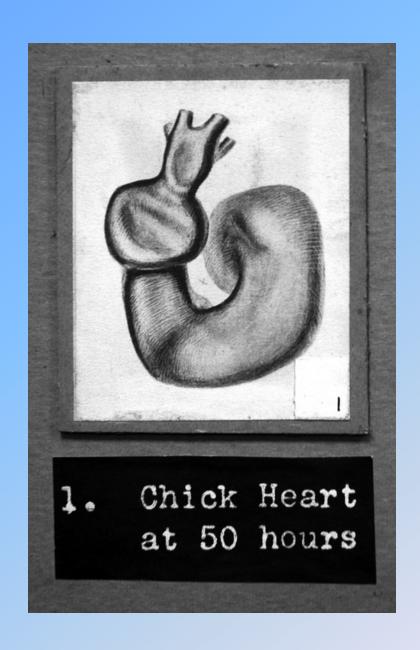
Sincerely Jeorge m. Robson m. I.

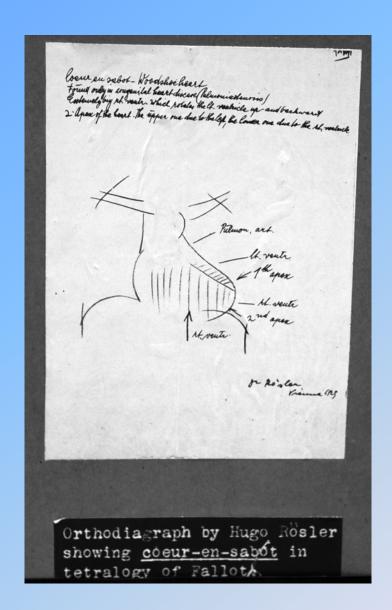


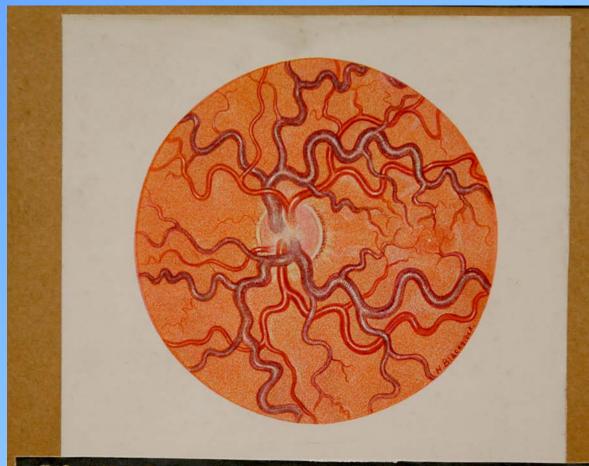


MECKEL'S CASE (1827) OF EXTREME COARCTATION.

Marked development of collateral circulation in a
man aged 35, who died from rupture of right auricle.







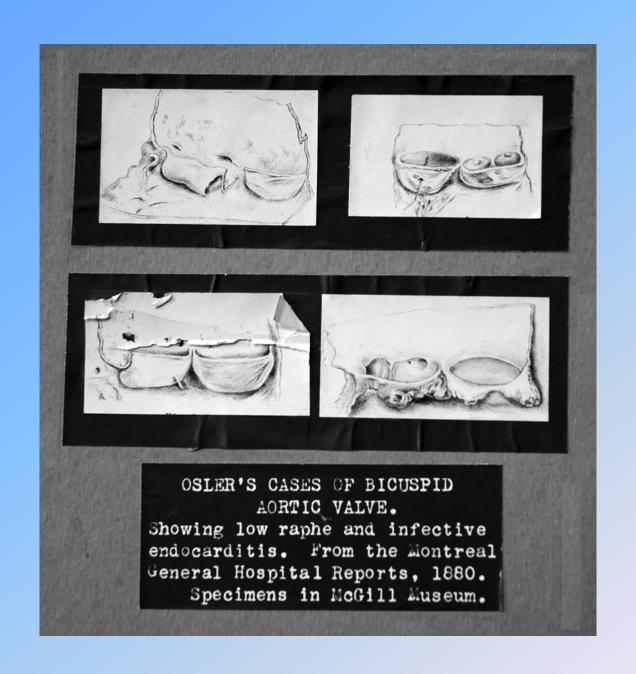
CYANOSIS RETINAE. Eyeground in a cyanotic child aged 3 1/2 yrs.

Painting by Harriet Blackstock

McGill University

CHART I. — STATISTICS OF CONGENITAL CARDIAC DISEASE (1,000 CASES ANALYZED)

Classification of Defects Classification of Defects Assumables of pericardium: 1. Pericardia defects. 2. Divertication. 3. Operation.	Marienum Meirenum Menn Metale Female	Cardiovascular system Cardiovascular system Cardiovascular system Cardiovascular system Cardiovascular system Cardiovascular Car	Amor., pilica- tions.	Per- sonal	Special sympto	Cardiac	Cardio- yasc. Defect	Pre quen
parties of percardium:		D. C.	nbolem se	Per- sonal	Cyano- sis		darteritis	totion
Anomalies of pericardium: 1. Pericardial defects.		t D. A. 4 A. S. 4 A. S. 7 V. S. 1 V	nbolism	attoa				1 3
1. Pericardial defects. 30		Patent D. Defect A. S. Defect A. S. Defect A. S. Defect V. S. Defect A. S. Defect A	In heart In vessels Elsewhere Faradoxical Er Cerebral Absor	Herefikary prediapa Rheumation Tuberculosis Congesital ayphilis Infre. dis., recovery	Silph: Moderate Moderate Moderate Market Terminal Clubbing Dygenes Dygenes Synopic attacks Delayed development	Polycyclenia Procyclenia Procyclenia Procyclenia Procyclenia Procyclenia Procyclenia Procyclenia Procyclenia Procyclenia Procymenia	Sudden Cardiac insufficiency Saliborn Saliborn Cerebral disease Bacteria resionadita or es-	Other causes Number classified as primar Number consultation other
1. Ectopia cordis. 2. "Isolated" Dextrocardia	75 Fetus 45 19 6 52 Adult 45 2 3 15m Fetus 1m 3 3 49 Birth 11 3 4 33 Birth 6 7 2 58 Fetus 8 3 4	1 3 4 5 6 1 1 2 3 2 2 2 2 2 1 1 2 1 1 1 1 2 4 4 1 5 2 4 4 5 6 6 4 1 3 1 1 1 1 1 1 3 4 4 3 4 3 4 3 3 1 3 3 1 3 2 1 2 4 4 6 1 5 4 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6	3 3 4	2 1	2 1 4 3 5 2 1 3 1 0 4	1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1	1 6 2 1 2 4 1 5 4 1 1 1 4 4 2 1	13 30
(a) Without inversion chambers (b) Without inversion chambers (c) Without inversion (c) Without inversion (c) Without inversion (c) Without inversion (c) Destroposition cordin (c) Distribution	41 2 m 26 3		1 2 2	1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1	2 1 4 3 5 2 1 3 1 6 . 4 6 3 . 4 1 0 2 1 1 1 3 1 1 2 1 2 1 1 2 2 2	2 1 - 2	1 2 2	11 3
4. Incomplete heterotary. 5. Destroposition orolls. 1. Biffel apex. 1. Diverticulum. 2. Diverticulum. 3. Compenial that before the control of	1 im Fe to 16d 1 2 4 Birth 10m 6 4 20 Birth 18,11 3 20 18d 7 2 1 48 Birth 2 4 48 Birth 2 4 48 Birth 2 4 49 10m 14 1 2 39 10w 14 1 2 49 24 46 4 1 69 23 36 4 1 69 23 36 4 1 69 23 39 3 2		2 2 10 3 3 2 2 3 6 1 2 3 3 4 1 1	1 1 1 1 1 1 1 1 1 1	3 2 1 1 6 2 1 5 1 1 3 2 1 2 1	2 1 1 1 3 1 3 1 1 1 1 1 3 1 1 1 1 1 1 1	5 4 1 3 1 3 2 1 2	2 10 2 . 4 3 1 1 7 6 6 2 5
Defects of International Conference of International Confe	70 3m 29 1524 64 6m 34 4 5 46 8m 19 8 8 52 16 38 3 2 32h Fetus 18h 2	. 40 . 15 . 112 6 . 314.28291310 10 . 4 . 2 . 1 . 3 . 4 . 9 . 7 . 4 . 4 118 . 11 . 1 . 9 . 5 . 812.1312 . 5	0 11 5 8 6	1.1	1 7 5 1 5	5 419 8 238 3	7 18 2 2 . 1 5 . 4	7 40 2 10 2 2 18 2 5 3
2. Defects elsewhere or multiple. 3. Aneurysms of pars membranacea. Complete defects of cardiac septa: 1. Cor triloculare biventriculare. 2. Cor triloculare histatum. 3. Cor biloculare. 4. Incomplete double heart. 10	49 Fetus 144 21 26 79 4m 23 2 2 60 24 42 6 1 31 17d 6 7 35 Birth 74 7 3 36 78h 31 3 3 44 2m 15 5 2	1 3 4 1 2 1	5 2 3	3 3 6 1 12 2	3. 112 114 3 1 4 1 . 1 . 1 . 1 . 1 3 3 3 2 1 6 5 . 1 3 3 3 2 1 6 5 . 1 2 1 4 1 4 3 . 6 3 30 1 5 7 6 . 3	1 1 1 1 1 2	1 4 4 1 10 31. 1 5 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1	6 7
Defects of aortic septum: 1, Persistent truncus (complete defect). 21. Communication between A. and P. A. 10 3, Congen. aneur. of right aortic sinus. 12. Transposition of arterial trunks: 1, Destroposition of aorta.	25 Birth 4 11 7 48 6d 14 6 3 53 11 28 11 1	11021 1914 1 2 21015 111 1 2 1 2 4 1 2 1 1 1 5 1 4 6 4 1 3 2 1 3 4 310 4 6	1 13 12 5 1 4 2 4 2 1 6 7 2 1	3 1 1	310 1 5 7 6 3	3 1 2 2 2 2 5 2 1 1	2 7 3 1 . 1 1 4 5 1 9	1 3 21 2 10 2 . 12
(a) A. From both ventricles. (b) A. from both ventricles. (c) A. from R. V., double comus. (d) A. from R. V., double comus. (e) Closed V. S. (b) Defect V. S. (c) Dartial transposition. (d) Corrected transposition. (e) Corrected transposition.	39 Birth		7 4 4	3 1 1 4 1 1 3 1	1 1 2 2 2 3 1 2 1 2 1 2 1 2 1 516 4 9 11 3 13 1 5 6 3 3 1 3 4 2 4 4 2 3	1 3 5 2 1 8	12 4 1 2 4 3 4 1 2 3 2 1 3 1	1 1 7 3 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1
	16 6d 2 1 1 3 20 12d 4 8 5 24 9w 11 3 1 45 10 22 5 4 57 4 18 51 60 11d 12 2721 28 11w 12 2112	116 4 4 . 21 7 10 1116 3 6 51 823 19 232 1 1 16 10 20 32 2 4 5 33 33 328 16 21 4 3 2 9 4 12 30 111	2 2 1 1 3 4 4 1 2 4 13 9 10 1 1 1 12 9 4 1 3	2 3 1 6 1 6 4 4 2 8 5 6 1 7 1 9		3 6 3 8 3 2 11 2 1 6 6 6 3 7 2 6 32 2 1 3 6 8 7 9 2 6 31 2	1 3 1 1 2 1 2 1 5 9 7 4 4 2	2 1 9 2 16 8 51 8 51 5 34
Pulmonary atreslat 1. With closed V. S. 2. With closed F. O., defect V. S. 3. With patent F. O., defect V. S. Pulmonary insufficiency or dilatation: 1. Valvular insufficiency. 2. Congenital dilatation P. A. 6	20 6d 11 2 4 29 10w 6 6 3 30 9d 4 9 3 64 22 43 2 59 21m 14 2 3	910 1 5 7 1 4 0 1 5 8 8 12 1 7 8 211 4 1 1 8 7 5 111718 1114 15 3 1 2 11114 1 2 1 1 2 1 1 1 2 2 1 1 0 0	5 5 8 2 1 2 6 8 5	3 1 1 1 1 2 1 1 1 1 1 1 1 1 1 1 1 1 1 1	10 2 3 2 2 1 4 1 1 5 7 7 7 5 1 1 3 1 1 4 2 2 2 3 3 3 3 3 3 3 3 3 3 3 3 3 3 3 3	1 1 1 1 2 5 1 1 1 1 1 1 1 1 1 1 1 1 1 1	1 3 . 1	1 10 3 12 18
Aortic stenoels and atreels: 1. Subsortic stenoels. 2. Aortic stenoels. 3. Aortic atreels. 12. Anomalies of stenoels. 13. Supernumerary cusps (a) of V. V. 2. Reduced no. (a) Bicuspid P. V. (b) Bicuspid P. V. 32	58 2 22 8 8 3 24 31h 31 4 5 15w 2d 2m 8 3 80 11 35 6 1 41 31 36 2	. 1 1 . 2 1 1 . 2 2 6 6 3 5 2 8 4 4 1 1 . 6 1 1 1 . 1 . 1 . 6 1 3 2 9 1011 211		1 1 1 4 2 1	1 1 1	2 5 . 1 1 6 2 2 1 1 2 1	3 2 1 2 3 2 1 1 1 7 2 1 1 1 1 1 1	4 12 11 12 3 8 2 7 32 1 1
2. Reduced no. (a) Bicuspid P. V. (b) Bicuspid A. V. 32 3. Defect (a) of P. V	28 7m 15 1 2 56 6w 51 3 26 2 28 7m 15 1 2 56 6w 51 3 6 27 51h 51 3 3 31 2d 10m 2 2	1 1	18888	1 2 . 3	2 . 1 6 11 1 . 1 1		6 5 3 3 1 1 3	1 1 3 16 6 5
4. Mitral attresis. Anomalies of a-v cusps: 1. Double orifices (a) of T. O. 2. Insuff. or defect (b) of M. V. (b) of M. V. Patent ductus arteriosus:	43 1 1 20 12 15 3 2 14 2d 7 1 1	1 1 1 2 2 4 1 4 2 4 4 3 4 4 1 1 1 1 2 1 1 1 1 2 1 1 1 1 2 1 1 2 1 1 1 1 2 1 1 1 1 2 1 1 1 1 2 1 1 1 1 2 1 1 1 1 2 1 1 1 1 2 1 1 1 1 2 1 1 1 1 2 1 1 1 1 1 2 1	4 2 2	221	2 1 3 1 5 1 2 1 1 3 2 5 37 13 5 19	2 2 3 1 1 1 2 . 2 2	1 2 1	1 1 8 1 5 1 5 2
1. Simple patency, 92 2. So-called aneurym, 13 3. Absence of D. A. Coarctation of the aorta: 1. Adult type. 7 2. Infantile type. 9 3. P. A. forms descending aorta. Hypophasia of the norta. 2	92 21 33 5315 9m Sh 11m 4 2 5i Fetus 1 79 2	S G S 2 S 23 7 41 837132418352551	1 27 14 8	9 5 5 1 8 1 . 1 1	3 2 110 117 1 110 1 3 1 3 1 3 1 1 2 1 1 1 2	4 7 5 61022 4 110	22.25 . 1 6 7 1 . 2 3 . 1 2 1 2 .	2
1. Valvular insufficiency p. A. Aertic atenosis and attrests 1. Insubnovitie stemosis. 1. Acrtic attribution of the property	87 Fetus 38 2 3 61 Fetus 17 3 12 6m 1d 3m 1 2 44 Adult 31 1 1 5 38 26 1 1		1 2 3 2 2 14 4	1	1 i i i 2 i i i i i i i i i i i i i i i	1 3 1	1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1	1 5 3 14 3 1 1 5 1 2
6. Common brachloorgh, trunk Anomalous coronaries 1. Origin from P. A. 2. Origin from P. A. 2. Origin from P. A. 3. Anomalous from P. A. 3. Anomalous pulmonary arteries 1. Origin from P. A. 2. Origin from P. A. 3. Origin from P. A. 3. Anomalous pulmonary veins. 4. Origin from P. A.	61 3 m 36 3 3 54 Adult 65 Birth 14 1 1 1 61 3m 18 4 2 86 7m 35 4 3 3 16 21 3 3	3 1 1 2 4 1 1 1 1 1 8 1 2 2 1 1 3 1 1 1 1 1 2 3 2 1 1 3 1 1 1 1	4 3 2 1		21311	11	3 1 1	1 8 1 1.



F.W. - SUMMARY OF CASE

HISTORY.

WHITE MALE, AGE 45. CHIEF COMPLAINT, WEAKNESS AND DYSPNEA. PAST HISTORY-KNOWN TO HAVE HAD HEART DISEASE FOR 18 YEARS. SIX MONTHS AGO WAS ILL WITH SYMPTOMS OF GRIPPE FROM WHICH HE NEVER RECOVERED. RECENTLY NOTICED PURPURIC SPOTS OVER LEGS.

PHYSICAL EXAMINATION:

THERE WAS FEVER, TACHYCARDIA, ENLARGED SPLEEN, PURPURA OVER LEGS, HYDROTHORAX. HEART: SYSTOLIC AND DIASTOLIC MURMURS AND SYSTOLIC THRILL OVER ADRTIC AREA.









PATHOLOGICAL FINDINGS:

HEART: WEIGHT 480 GRAMS.

ATWO ADRTIC CUSPS; TWO NORMAL COMMISSURES AND ONE FUSED COMMISSURE. CIRCUMFERENCE OF RING 6 CM. LENGTH OF COMBINED CUSP 3.2 CM. (RIGHT AND LEFT ANTERIOR), POSTERIOR CUSP 2.8 CM. BOTH CORONARIES ARISE FROM SINUS OF COMBINED CUSP.

ANTERIOR CUSP IS DIVIDED BY A HIGH SHORT RAPHE FREE MARGIN OF CUSP HAS UNINTERRUPTED SWING. POSTERIOR CUSP AND AORTIC LEAFLET OF MITRAL VALVE ARE THE SEAT OF ULCERATIVE ENDOCARDITIS. SPLEEN SHOWED ON CULTURE STREPTOCOCCUS VIRIDANS. THERE WAS AN EMBOLIC GLOMERULO-NEPHRITIS.

CAUSE OF DEATH.

SUBACUTE BACTERIAL ENDOCARDITIS.

MICROSCOPICAL EXAMINATION:

PARALLEL SERIAL SECTIONS THROUGH RAPHE. WEIGERT'S ELASTICA STAIN.

SERIAL SECTION *10. THE RAPHE IS SEEN AS AN ELEVATION UPON THE SINUS SURFACE OF THE VALVE JUST DISTAL TO ITS ATTACHMENT. THE ANNULUS FIBROSUS ARISES DEEP TO THE AORTIC ELASTIC MEDIA.

THE SUPERFICIAL FURNISHED OF THE STANDARD FURNISHED TO THE MAINTENANCE THAN THE REPRESENDANCE. THE SUPERFICIAL TO THE ELASTIC PROCEEDS FURTHER TOWARD THE VALVE THAN THE DEEP ENDING AT THE WEDGE SHAPED JUNCTION.

2 SERIAL SECTION #29 - APPROXIMATELY .6 MM. ADVANCED. THE CONNECTIVE TISSUE OF THE ANNULUS
NOW LIES SUPERFICIAL TO THE ELASTIC FIBERS.

THERE HAS BEEN A REVERSAL OF THE RELATIONSHIP AT THE ANNULUS-MEDIA JUNCTION. NORMAL COMMISSURAL RELATIONSHIPS.

3 SERIAL SECTION #34-APPROXIMATELY 15 MM. ADVANCED. NORMAL COMMISSURAL RELATIONSHIP OF THE ANNULUS MEDIA JUNCTION STILL MAINTAINED. THE DEEP ENDING OF THE AORTIC ELASTIC HBERS DOES NOT HOWEVER PRO-CEED AS FAR BEHIND THE VALVE AS AT A NORMAL COMMISSURE. NO EVIDENCE OF INFLAMMATORY REACTION. DIAGNOSIS:

PROBABLE CONGENITAL BICUSPID VALVE. THE CRITERIA FOR MICROSCOPIC IDENTIFICATION MAY BE INCORRECT FOR THIS CASE.

J.F.- MEDICAL EXAMINER'S CASE-SUMMARY

HISTORY:

WHITE MALE, AGE 52. PATIENT BROUGHT TO HOSPITAL AND DIED A FEW MINUTES AFTER ARRIVAL.



PATHOLOGICAL FINDINGS:

HEART: 770 GRAMS.

TWO AORTIC CUSPS; TWO NORMAL COMMISSURES. CIRCUMFERENCE OF RING 7.5 CM. (LEFT ANTERIOR AND RIGHT ANTERIOR), POSTERIOR CUSP 3.5 CM. BOTH CORONARIES ARISE FROM SINUS OF THE COMBINED CUSP. SMALL ACCESSORY RIGHT CORONARY. COMBINED CUSP, PARTICULARLY AT ITS FREE MARGIN, SHOWS EXTENSIVE CALCIUM DEPOSITION. NO RAPHE WITHIN CUSPS. UNINTERRUPTED SWING OF FREE EDGE OF VALVES. POSTERIOR CUSP SHOWS AREA OF PERFORATION CLOSE TO COMMISSURE. MODERATE AMOUNT OF CALCIUM AT ROOT OF POSTERIOR CUSP AND UPON AORTIC LEAFLET OF MITRAL VALVE. AORTA IMMEDIATELY ABOVE SMOOTH. ABDOMINAL AORTA ARTERIO - SCLEROTIC. KIDNEYS SHOW NO EMBOLIC GLOMERULAR LESIONS. NO OTHER CONGENITAL ANOMALIES.

CAUSE OF DEATH:

CONGESTIVE HEART FAILURE.

DIAGNOSIS:

CONGENITAL BICUSPID AORTIC VALVE. AORTIC STENOSIS. THE GROSS CRITERIA ARE SUFFICIENTLY CHARACTERISTIC. NO MICROSCOPIC STUDY IN THIS CASE.

