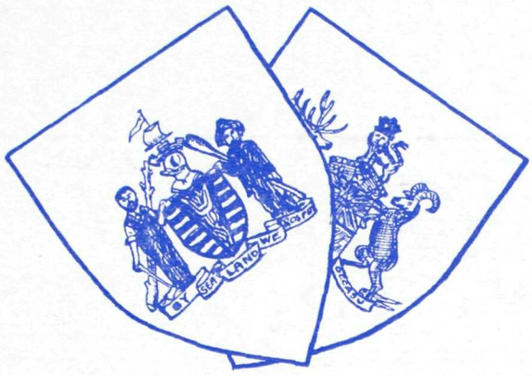
THE BULLETIN



OF THE VANCOUVER MEDICAL ASSOCIATION

Contents

Vol. VII. SEPTEMBER, 1931 No. 12

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THE VANCOUVER MEDICAL ASSOCIATION

BULLETIN

Published Monthly under the Auspices of the Vancouver Medical Association in the Interests of the Medical Profession.

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Vol. VII. SEPTEMBER, 1931 No. 12

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VANCOUVER HEALTH DEPARTMENT

STATISTICS, JULY, 1931

Total Population (Estimated)		242,629
Asiatic Population (Estimated)		14,227
	Rate per 1,000	Population
Total Deaths	180	8.7
Asiatic Deaths	11	9.1
Deaths—Residents only	160	7.8
Birth Registrations	344	16.7
Male 193		
Female 151		
INFANTILE MORTALITY—		
Deaths under one year of age	5	
Death Rate—Per 1000 births	14.5	
Stillbirths (not included in above)	11	

CASES OF CONTAGIOUS DISEASES REPORTED IN CITY

	June, 1931		July, 1931		August 1st to 15th, 1931	
	Cases	Deaths	Cases	Deaths	Cases	Deaths
Smallpox	0	0	0	0	0	0
Scarlet Fever	12	0	3	0	4	0
Diphtheria	3	0	12	1	1	0
Chicken-pox	35	1	14	0	4	0
Measles	4	0	2	0	1	0
Mumps	27	0	2	0	2	0
Whooping-cough	2	0	3	0	0	0
Typhoid Fever	0	0	2	0	1	0
Paratyphoid	0	0	0	0	0	0
Tuberculosis	55	10	52	20	42	—
Poliomyelitis	3	1	3	1	4	0
Meningitis (Epidemic)	0	0	1	0	0	0
Erysipelas	3	0	6	0	4	0
Encephalitis Lethargica	0	0	0	0	0	0

EDITOR'S PAGE

Pictures of the ostrich show him as a bird possessing very few tail-feathers. This is probably due to his habit of burying his head in the sand, whenever danger threatens. He thus puts himself in a position where anyone who wishes to do so, can remove the tail-feathers quickly and easily.

There is a lesson in this parable, for the medical profession. As a profession, we imitate the ostrich to a large degree. We refuse to face the fact that the social order is changing, and that as a result of this change, new conditions are arising ,and have arisen, which gravely threaten our economic security. Since we do not show any evidence of resentment, gradually but steadily, we are being mulcted of rights and privileges, which we seem to have taken for granted would always be ours. Every medical man knows that small but persistent and long-continued haemorrhage is more damaging than the occasional loss of a large amount of blood, and we should be wiser and better-advised, if we faced the situation squarely, made the necessary and proper adjustments, and put ourselves in line with the new state of things.

There are signs, however, of awakening. In Great Britain, an immense amount of valuable and far-reaching work is being done by the British Medical Association, as anyone who follows the supplements of the British Medical Journal will realize. The profession in the Old Land is particularly well-organized and well-led. Their National Association has been fortunate in its General Secretary, Dr. Arthur Cox, who for many years has been a tower of strength—and is soon, we believe, to retire. It will be difficult to fill his place.

We, in Canada, are fortunate, too, in our National Association. It is very much alive, and ably led. Dr. T. C. Routley, the General Secretary, is one of the dynamic men of our time. It is a pleasure to have dealings with him, and one cannot but recognize and acknowledge his energy and alertness.

At the Annual Meeting here this year, an important resolution was passed. Steps are to be taken to organize a Study Group all over Canada ,with headquarters at the Canadian Medical Association offices, which will go into the whole question of health insurance, and be prepared to submit constructive proposals.

This is as it should be. While B. C. is at present furthest ahead in the political management of this question, it is only a matter of time before the rest of Canada will be fully interested, and our relation to it should be on national lines, rather than merely provincial ones. We are glad to see that this step has been taken and hope that it will be productive of result. One of the first problems, we feel, is in the matter of "charity practice" so-called, which has become such a burden, and an unfair one at that, on the medical profession. Space will not permit development of this theme, but it is one that is worthy of our closest and most careful study. It is in this connection more than any other, that the medical profession has allowed itself to be manoeuvred into a position

of grave danger, and one that is very unfair, and the time has come for us to protect ourselves against the rising tide of encroachment in this regard.

* * *

NEWS AND NOTES

We congratulate Dr. and Mrs. U. P. Byrne, of New Westminster, on the birth of a son, on July 25th.

* * *

Dr. H. H. McIntosh, who accompanied our President, Dr. C. W. Prowd, on a trip to the Third International Congress on Radiology in Paris, has returned to work. Dr. Prowd, however, has gone on to Russia, and so far the only news from him is a picture postcard from a Hofbrau-haus, instead of the interesting scientific notes on the Congress which he promised us. However, he will doubtless have something more to say when he gets back to Vancouver.

* * *

Dr. J. C. Haramia, formerly of the Vancouver General Hospital Interne Staff, and who has, for the past two years, been doing locum work around the Province, has opened an office in the Dominion Building.

* * *

We regret to say that Dr. A. P. Proctor, the Registrar of the College of Physicians and Surgeons of B. C., has been ill in the Vancouver General Hospital, under the care of his old friends, Dr. R. E. McKechnie and Dr. J. W. Thomson. The doctor is much improved and should soon be home again.

* * *

A special meeting of the Association is to be held on September 9th, in the Ballroom of the Georgia Hotel, on the occasion of the Autumn Postgraduate Tour. A joint Dinner of the B. C. and Vancouver Medical Associations will take place at 7 p.m., after which Drs. F. H. McKay and C. C. Birchard, both of Montreal, will speak, Dr. McKay on "Poliomyelitis," and Dr. Birchard on "Cardiac Signs and Symptonms." Tickets will be $1.50, and can be obtained at the Association Offices.

* * *

As so many of the members object to the presentation of drafts for the Annual dues in November as laid down in the Constitution and By-laws of the Association, we propose this year to send out a further statement, to each member whose dues are unpaid, during the month of September. Prompt payment on receipt of the statement will obviate the necessity for a draft later on, and will also be an economy, which is the order of the day.

* * *

We regret that in our notes on the addresses given by Drs. Primrose and Robertson, we omitted to give credit to the Staff of the Vancouver General Hospital, who were responsible for the opportunity given us to hear these interesting speakers.

Another appendix gone West. This time the one formerly housed by Dr. Gordon Burke. "An empty house is better than a bad tenant."

* * *

The Library Committee feels constrained to remind members that there is a rule limiting the time for which books may be borrowed from the Library. Should the Committee endeavour to strictly enforce this rule we feel the Association would probably be bankrupt of both postage stamps and members in a very short time. The Committee, however, feels that the ultimate limit has been reached when a book is kept out for two years, more or less. If every member, after reading this month's News and Notes, would look through his office and his home and return everything he finds belonging to the Library, the Committee would take heart again. The custom of members going off for their holidays leaving borrowed books at home, which other members often need, is a bad habit and steps should be taken to correct it. Immediate improvement will, we feel sure, follow the hint.

* * *

Apropos of above will somebody please return Volume Six of Abt's Paediatrics.

* * *

A most enjoyable gathering took place at Jericho Country Club, on the night of August 17th, when the staff of the Vancouver General Hospital met to tender a farewell dinner to Dr. R. A. Seymour. About seventy guests sat down at 7:30. The health of the guest of honour was proposed by Dr. P. A. McLennan in a characteristically felicitous strain the wine of whose speech sparkled with bubbles of unquestionable vintage. He was most satisfactorily seconded by Dr. J. S. Conklin. If the proposer had already stolen the thunder of the seconder, the latter had yet other bolts in his quiver which rocked the assemblage visibly.

Before Dr. Seymour rose to reply, Dr. B. D. Gillies, who ably presided as chairman, presented him, on behalf of the Staff, with a handsome silver service. At the same time Dr. Haywood presented a desk set from the V. H. G. Nursing Staff.

In his brief and feeling response, Dr. Seymour rose nobly to a trying occasion. A pleasant note struck by all the speakers was the assumption that in speeding Dr. Seymour off to his new field of work, a farewell was not being said, but merely an "*a bientot.*"

* * *

The B. C. Provincial Board of Health Laboratories and its staff are, at date of writing, about to be transferred from the Vancouver General Hospital, to 763 Hornby Street, facing the Court House and just back of the new C.N.R. Hotel. The actual date of commencing technical work will be probably about the last week in August.

All the Public Health Laboratory work, heretofore done at the Provincial Board of Health Laboratories, at the Vancouver General Hos-

pital, will hereafter be done at the Hornby Street laboratories. The Hospital Clinical Laboratory examinations will not be made at the Hornby Street laboratories but at the Hospital as heretofore. The Public Health work at the Hornby Street laboratories includes the following:—Kahn(spirochaeta, gonorrhea, diphtheria, animal inoculations, tuberculosis (all forms: urine, spinal fluid, etc.), ringworm, parasites, malaria, typhoid, paratyphoid, Shiga, Flexner, abortus, Vincents, meningococci, etc.—in fact all bacteriological work relating to diagnosis of infection and release of same. Water and milk specimens and diphtheria cultures from schools will also be examined for municipalities outside Vancouver, under special financial agreements in each case.

The new laboratories are established and controlled by the Provincial Board of Health, but will do also the Public Health Laboratory work for the City of Vancouver, which City pays the Provincial Board of Health for its share.

No patients will be received at the Laboratories for the taking of specimens. Specimens of every kind should be sent to the Laboratories completely ready for examination. This will not affect present facilities in this direction, for Dr. H. H. Pitts, now Director of the Vancouver General Hospital Laboratories, has arranged that the few such Public Health specimens, (chiefly Kahn bloods) as hitherto have been taken from patients at the Hospital Laboratories will continue to be taken there, and then forwarded to the new Laboratories.

The staff consists of Dr. H. W. Hill, Director; Donna E. Kerr, M.A., Assistant Director; Mabel M. Malcolm, Chief of Bacteriology; six technical workers of various grades, two clerks, and a resident janitor; all but one having previously been on the Provincial staff at the Vancouver General Hospital.

The Laboratories have been designed, equipped and staffed with the object of rendering the highest forms of Public Health Laboratory service to the Province and the City.

The Provincial Public Health Laboratories work, since its inception in 1920 under Dr. H. E. Young, with Dr. R. H. Mullin as first Director, has been carried on at the Vancouver General Hospital. The recent transfer of this work to Hornby Street was the outcome of friendly negotiations amongst all the parties concerned. It was universally recognized that adequate provision of laboratory space and staff was necessary for the rapidly increasing laboratory work, both Hospital and Public Health, which threatened to completely swamp the available space and staff at the Hospital. Everyone concerned saw the needs of both laboratories, but also that the needs of neither could be met except by radical changes, such as would provide more space and therefore allow a larger staff. The solution agreed to as the only obvious and possible one was that which has now been reached—the separation of the Hospital and the Public Health work, giving to each increased space and independence for the pursuit of its own objects.

Advocated by Dr. F. T. Underhill, as Medical Health Officer of Vancouver, and by Dr. J. W. MacIntosh, his successor, the proposition

was looked upon favorably by Dr. H. E. Young, Provincial Health Officer, and Dr. A. K. Haywood, General Superintendent of the Vancouver General Hospital. Arrangements were made to this end, beginning in November, 1930, and developed between the parties concerned —the Province, the City and the Hospital—during subsequent months.

The Hornby Street buildings now in use were already Provincial property, but required considerable making over for laboratory purposes. Apparatus and equipment were provided, in part already in use at and taken over from the Vancouver General Hospital Laboratories, in part from accumulations of the Provincial Board of Health in Victoria, in part new. The principle of nothing fanciful, but everything the best for its purpose, already firmly established in the Vancouver General Hospital Laboratories, has been followed throughout in the new Laboratories. We believe that space adequate for a time at least, for the rapidly growing increases in work, has been secured and that staff and equipment will be increased by the Government as increases in work develop. On this basis we may look forward to notably serviceable Public Health laboratory work in the near future.

The first year in the new building will necessarily involve many adjustments and adaptations in which it will be the aim of the Provincial Laboratories to achieve the smoothest, most rapid and efficient system. but this will necessarily be a matter of growth rather than of the immediate spresent. "Shaking down" into the new quarters cannot be done all at once; but we hope will be done quickly and well. Thereafter expansion will be achieved as rapidly as circumstances will permit.

* * *

MITRAL STENOSIS

DR. WALLACE WILSON

If you read over the minutes of the meetings of this Association from the time of its inception under the presidency of the late Dr. D. H. Wilson, in 1898, up to the year of our Lord 1931, you will find that a paper on valvular disease of the heart has never been read before the members. Indeed the whole question of cardio-vascular disease appears to have appealed but little to the many men addressing you.

Up to 1913 not a word—then Dr. Pearson discussed "High blood pressure with special reference to Nephritis," and again, in 1915, he spoke on "The treatment of heart disease." Silence again until 1921, when I reported fifty cases of "Dilatation of the Aorta" and finally in the last few years our president has read papers on "Coronary Thrombosis" and "Cardiac Pain," while Dr. Coffen, of Portland, brought us up to date with his resume of "Modern advances in Cardiology." Six papers by four men in 33 years to cover the whole field of cardio-vascular pathology and physiology and not one of them directly concerned with endocarditis!

An address delivered before The Vancouver Medical Association, March, 1931.

It is certainly passing strange that during the period from 1898 to the war—a period when murmurs still held the stage and when the importance and significance of the various physical findings constituted the standard by which heart disease was measured—no discussion of the subject occurred in our Association. Evidently in those days we were not "heart conscious" or we agreed with Trousseau that "the exact seat of a cardiac lesion is a study more interesting than useful."

Therefore, Mr. President, if in discussing stenosis of the mitral valve, I but stir your memories without contributing to the sum of your knowledge I shall still feel justified. I shall feel justified because the lesion is a serious one and the most frequent single form of valvular disease, because it is, to you as an Association, an entirely new subject and, because it should be on record that we, in the space of 33 years, devoted at least part of one evening to some phase of valvular disease of the heart.

The first definite records of cardiac disease that we find in the literature are contained in the writings of the morbid anatomists of the eighteenth century—Vieussens, Lancisi, Morgagni—and to Morgagni goes the priority of describing the mitral valve in cases of advanced stenosis.

In 1749, J. B. de Senac published a treatise dealing especially with heart disease and it was then definitely established that heart disease existed and could be diagnosed. Twelve years later came Auenbrugger with his percussion and in another fifty-five Laennec in 1816, with his mediate auscultation.

For most of the next hundred years the teachings of these men dominated the field of cardiology and medical men bowed down and worshipped at the shrine of murmurs as the be-all and end-all of heart disease. In 1836, Peter Mere Latham, who was the great clinican of his day in London, wrote: "But it is needless to dwell upon the indisputable fact that by the use of a well-disciplined and well-practiced ear we arrive at a readier and surer diagnosis of diseases of the heart than by all the other means of enquiry which clinical observation can command."

But finally came MacKenzie, and then the war, and there followed a great, renewed interest in symptoms and physiological efficiency, with much less importance being attached to the presence or absence of murmurs.

And so today, in discussing stenosis of the mitral valve, we must consider not only the morbid anatomy and the signs produced thereby, but, also what is of more importance, the character of the response of the heart to the lesion present.

Aetiology

The aetiology of mitral stenosis is that of rheumatic fever. We may not know the exact nature of the virus, for, while Coombs has endorsed the work of Poynton and Paine and accepted a member of the streptococcus family as the causal agent, there are many who are not

so converted; nevertheless, be the virus what it may, all reliable evidence points to the fact that true mitral stenosis spells some form of rheumatic fever past or present.

All the sites of the primary entry of the organism into the body are not known but, undoubtedly, one frequent portal is the lymphatic tissue of the pharynx. Once arrived, the invader reaches the valves via the highway of the blood stream. In this connection Poynton and Paine have recorded some experimental observations suggesting that the infection of the valves takes place from the coronary circulation and not from the blood in the heart chambers and that therefore the morbid changes begin within the structure of the valve as they do within the body of the vermiform appendix. On the other hand Ritter, Gross and Kugal in a study of 700 normal hearts found traces of blood vessels in some of the valves in only 14 and concluded these were embryonic vestiges.

Morbid Anatomy

The inflammatory reaction set up within the valve is predominantly productive in character and permanent lesions are the result. Repeated attacks increase the damage and there finally results a diffuse fibrosis which "so shortens and thickens the mitral curtains and their chordae tendineae as to encroach on every side on the mitral opening. This encroachment varies from a stiffening of the edges to the "button-hole mitral" (Coombs). Severe stenosis may result from a single prolonged attack of rheumatic fever as in the case of the little girl reported by Poynton where, in the course of five months at the bedside, he watched the development of all physical signs of mitral stenosis in what had been at the commencement an apparently healthy valve. The child died and the autopsy confirmed the clinical findings. Most frequently, however, fully developed stenosis is the result of a fibrosis engendered by repeated attacks of rheumatism, chorea, or tonsillitis that have occurred over a period of years.

Sex

More frequent in females, but there is no known reason for this.

Age Incidence

If mitral stenosis is spelt by rheumatic fever then the age incidence of the first departure of the mitral valve from normal is that of rheumatic fever and is highest around the tenth year of life. Cases do occur earlier. A mother had rheumatic fever during pregnancy and an autopsy on her two day old infant showed masses of vegetations on the mitral valve. Another child had chorea and mitral disease at two and a half years followed by relapses and mitral stenosis by the fourth year (Poynton). Cases are also found much later in life but probably seventy-five per cent. of the mitral stenosis seen in adults had its origin in childhood around the end of the first decade of life.

Incidence In Rheumatic Endocarditis

Coombs' figures are interesting. In 97 cases of rheumatic endocarditis the mitral valve was involved 97 times, the aortic 57, the tri-

cuspid 35, and the pulmonary twice. From which we may deduce that the mitral valve is the only valve that may become involved while the rest escape injury and that we should hesitate to say that a lesion of any of the other valves is rheumatic in origin if, at the same time, the mitral is found to be free.

Physiology

The functions of the mitral valve are:

1. Prevention of the backward flow of blood into the left auricle during systole.
2. To avoid impeding the flow of blood into the left ventricle during diastole.

If the first function is interfered with we say "mitral regurgitation," if the second "mitral stenosis." Yet a disturbance of only one of the two functions is rarely found. Mitral regurgitation without stenosis is an extremely rare bird, although in heart disease it is one of the commonest diagnoses made. Osler describes this pure lesion as a curling and puckering of the mitral leaflets without a narrowing of the orifice and Cabot goes so far as to say that "it cannot be recognized during life and a diagnosis of mitral regurgitation without mitral stenosis is never justified." When it does occur it is an extremely serious lesion with not a good prognosis. The blood churns up and down from ventricle to auricle and from auricle to ventricle and you get an early dilatation of the whole left heart. When a case has been diagnosed as mitral regurgitation and the patient lives fairly actively and to a good age one can be satisfied that stenosis is also present.

The left auricle makes up the superior and posterior parts of the heart. When stenosis of the mitral valve occurs there is a piling up of blood in this auricle and as a result it enlarges. This enlargement may cause projection upwards, backwards, or to the right of the normal heart outline and as a result certain signs and symptoms may arise.

Signs and Syptoms Due To Enlarged Left Auricle

1. *Dyspnoea*—due to pressure upwards on the left or on the left and right main bronchi. The auricle may press into the angle made by the two main bronchi and cause it to be markedly widened with a narrowing of the bronchial lumina or it may exert sufficient pressure on the left bronchus to cause collapse of the left lung.

2. *Dysphagia*—from pressure backwards on the oesophagus.

3. *Choking sensations and alteration of voice*—resulting from left recurrent, laryngeal palsy caused by the enlarged auricle lifting up the left recurrent laryngeal nerve and crushing it against the under side of the aortic arch.

4. *Inequality of the pupils*—There being contraction of the left due to pressure paralysis of the sympathetic.

5. *Inequality of the pulses*—The left being smaller due to the auricle pressing on the left subclavian.

6. The left auricle may enlarge to the right to such an extent, as in cases reported by Manuel, Batty Shaw, East, Bedford and others, that a visible and palpable pulsation is noted as far out as the right lateral chest wall. In these cases there is considerable compression of the base of the right lung and the auricle has been punctured more than once in the belief that a pleural effusion was present.

It is to be remembered that the above signs and symptoms are uncommon, that they depend entirely on a very considerable enlargement of the left auricle and that they may be absent in the presence of the most extreme degree of mitral stenosis. They do occur, however, and some examples have been observed and put on record as far back as 1838. When present, recognition for what they are will sometimes clear up an otherwise obscure case.

More common symptons—not many.

Early in the history there are usually complaints of some distress on exertion but through a long life there may be no marked symptoms, unless an extra strain, such as a change of occupation to heavier work, increased blood pressure resultant on kidney disease, menopause, etc., or failing heart muscle, makes its presence felt. When extra exertion *does* cause distress that distress is usually the result of dyspnoea or dyspnoea and palpitation. This dyspnoea is of the "simple type" referred to by Rolleston where the cause is increased irritability of the respiratory centre due to an oxygen want that is secondary to diminished blood supply to the centre. This can be the more readily understood when we remember that with stenosis present there is an incomplete filling of the left ventricle.

Nocturnal dyspnoea is a cross that even the advanced sufferer from mitral stenosis rarely has to bear. Those distressing attacks of cardiac asthma and Cheyne-Stokes' breathing, that turn night into hell for so many cardiac cases, are usually reserved for those afflicted with aortic or coronary disease, for they are a sign of a failing left ventricle.

When a failing left auricle, a failing right heart, or both, demands the pooling of blood in the pulmonary vascular bed, then increasing diurnal dyspnoea makes its appearance, cough is troublesome, recurring bronchitis or pneumonia is not uncommon, and frequent haemoptysis is the visible sign of a defeated heart.

Signs

Percussion—may be negative unless you have an enlargement of the left auricle or a displacement to the left (never down) of the apex, due to an enlarged right heart. When this occurs you get a "mitralization" of the cardiac outline with a disappearance of the normal "waistline" of the heart. Rarely, when there is a marked enlargement of the left auricle to the right, percussion reveals the dullness well out to the right of the normal right heart border.

If, after percussion, there is any doubt as to the size of the left auricle the patient should always be examined behind the fluoroscopic

screen, particularly in the right anterior oblique position. Here an enlarged left auricle, that has not been demonstrable by percussion, or has not been seen through the screen in the antero-posterior position, will project backwards from the normal heart outline towards the spine. To further verify the findings the patient should then be given a heavy barium paste to swallow. Normally the oesophagus will be seen to descend in an almost straight line between the heart shadow and the spine and close to the heart. If the left auricle is enlarged, however, the oesophagus will be seen to break into an angle at the upper border of the left auricle and swing back towards the spine in a more or less acute-angled curve.

Palpation

1. *Thrill*—The purring tremor, or palpable thrill of mitral stenosis, was first recognized by Laennec and called by him the "fremissement cataire." He failed, however, to realize that it was presystolic in time and even as late as 1870, Austin Flint maintained that it was due to regurgitation through the mitral valve.

In a deep-voiced man one can often palpate vocal fremitus but in a woman with a high pitched voice it cannot be done. Low-pitched cardiac murmurs of which the presystolic of mitral stenosis is an example can be palpated also. What the hand feels is the vibration set up by the murmur and the "thrill" therefore occurs before the first heart sound. If the presystolic murmur disappears, as in auricular fibrillation, so does the thrill, but it must be remembered that occasionally the murmur may be so low pitched as to be inaudible to the ear and still the palpating hand may feel a thrill.

2. *Apex beat*—The impulse to be felt with the hand over the chest wall in the region of the apex is a very characteristic one. The hand feels the apex strike abruptly and forcibly against the chest wall and then at once drop away again. This type of apical impulse may sometimes be felt before the presystolic murmur or thrill have appeared or after they have disappeared with the onset of auricular fibrillation.

Auscultation

1. *Presystolic murmur*—If there is one single sign on which, today, a diagnosis of mitral stenosis is based it is a well defined presystolic murmur and yet the profession was slow in recognizing and accepting it. Laennec did not recognize it, yet here is his description of mitral stenosis in a boy of sixteen. "The hand placed over the region of the heart felt the pulsation strongly and accompanied by the purring vibration; the stethoscope applied between the cartilages of the fifth and seventh ribs gave the following results: contraction of the auricles extremely prolonged, accompanied by a dull but loud sound exactly like that produced by a file on wood. This sound was attended by a vibration sensible to the ear, and which is evidently the same as that felt by the hand. Succeeding this, a louder sound and a shock synchronous with the pulse announced the contraction of the ventricle, which occupied only one fourth part of the time. From these signs I gave the follow-

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MONTREAL

ing diagnosis: ossification of the mitral valves, slight hypertrophy of the left ventricle."

Twenty years later this curious paragraph occurred in Latham's book published in 1836. "But it would almost seem that the mitral orifice could be the seat of only one murmur and that murmur the systolic. The cases are so rare in which either the diastolic murmur alone or the systolic and the diastolic murmurs together, can be fairly imputed to the mitral valve, that they are a sort of medical curiosity. Yet the condition of disease in the mitral valve is often found to be such as must have raised certain impediment to the passage of blood from the auricle into the ventricle. Why, then, is the murmur which would indicate such impediment, and which would be coincident with the diastole of the heart, a thing not found in practice, when the mitral valve is alone diseased?"—and he goes on to explain the discrepancy as due to there being no noise produced by the blood being forced through by the thin-walled auricle while regurgitation was accompanied by a murmur because the rush of blood was occasioned by the thick-walled ventricle.

The first clear description of the presystolic murmur was given by the Frenchman Fauvel in an article published in the "Archives Generales" in 1843, but even as late as 1870, Austin Flint deemed it necessary in his chapter on mitral stenosis to occupy some space with arguments to controvert the school that would not accept the presystolic.

This murmur is caused by the stream of blood passing through the stiffened mitral valve into the ventricle. It begins softly with the commencement of the auricular contraction, increases in intensity as the force of that contraction increases and ends in the hard first soud that signalizes the onset of the ventricular cycle. It *is* absent in the early stages of sclerosis of the mitral leaflets and when auricular fibrillation is present. It *may* be absent when the left auricle is greatly enlarged and flabby and when there is considerable dilation of the left ventricle with an accompanying dilation of the mitral ring. When there is doubt as to its presence it is best sought by listening over the apex with the patient lying down on his left side after being exercised or after he has inhaled some amylnitrite. When there is no doubt as to its presence there can be no doubt about the existence of mitral stenosis in the vast majority of cases. Rarely, in some cases of aortic regurgitation there occurs a soft presystolic murmur in the absence of a mitral sclerosis. This so-called Austin Flint murmur is caused by the stream of regurgitating blood from the aorta inpinging on the nearest mitral leaflet, pressing it into the mitral lumen and, producing thereby a slight relative stenosis.

Other Murmurs

Systolic—due to regurgitation is present when the stiffened mitral valve fails to close or when it cannot close because of dilatation of the mitral ring.

Diastolic—This rumbling murmur, recognized long before the presystolic, is caused by blood running through the patent mitral open-

ing from the engorged left auricle into the ventricle during ventricular diastole. In contra distinction to the presystolic it starts loudly and the volume of sound gradually diminishes until it ceases altogether when the pressures in the auricle and ventricle are equal. It is to be distinguished from the diastolic of aortic regurgitation which commences immediately after the second heart sound; there is a distinct pause after the secon dsound before the diastolic of mitral stenosis commences. There is no collapsing pulse and no evidence of left ventricle hypertrophy. The onset of auricular fibrillation does not mean the disappearance of either the systolic or the diastolic murmurs.

It can be seen that with the presence of all these murmurs at the apex of the same heart the whole cardiac cycle will be fairly well filled with murmur. The presystolic leads off and travels up to the first heart sound where the systolic takes up the refrain and persists almost to the second heart sound. Follows a very slight interval of silence and then enters the rumbling diastolic that fades gradually away into the commencement of the presystolic. The differentiation of these murmurs when all are present may be easy in a slow heart and impossible in a fast one.

There are still other murmurs sometimes present in mitral stenosis but their importance is secondary and their significance uncertain.

Enumerated briefly they are:

1. A loud blowing pulmonary systolic. This has been accounted for as follows—The increased presure in the pulmonary circulation causes a widening of the pulmonary artery and the enlarged left auricle not only strips and exposes the artery more but presses it up against the anterior chest wall producing a murmur, or the widened pulmonary artery causes a systolic murmur as does a widened aorta.

2. *A fine blowing diastolic murmur over the pulmonary area*—the Graham Steele murmur of high lesser circulation pressure. Here the murmur is that of a relative pulmonary insufficiency and it is not present early or late in the course of a mitral stenosis and it varies with position, deep breathing, etc. Very rarely this murmur is due to a widening of the pulmonary ring due to rheumatic infection in the region of the pulmonary valve. In this condition when the murmur is established it is constant unless very late in the picture. Its differentiation from the diastolic of aortic regurgitation is in the absence of a Corrigan pulse and in the shape of the heart. The so-called Graham Steele heart shows a big right heart and an enlarged pulmonary artery that on the screen is well seen projecting out and slightly up from the upper part of the "waistline' of the heart. In aortic regurgitation the left ventricular enlargement deepens and accentuates the waist-line.

Alteration of the Heart Sounds

1. All are familiar with the *hard ringing first sound* of mitral stenosis but its origins are clouded. Is it due to an increased amount of connective tissue in the valves? To some extent "yes." It is also due to incomplete filling of the left ventricle? Also to some extent "yes"

because it is noted that in badly shocked patients the same ringing first sound may be heard when most of the blood is in the peripheral circulation and the left ventricle is underfilled. This type of first sound is a good and early sign of sclerosis of the mitral valve and persists after the onset of auricular fibrillation.

2. *Accentuation of the pulmonary second*—does occur but it is not of much diagnostic importance. When present it is due to the enlarged left auricle exposing the widened pulmonary artery and pushing it closer to the anterior chest wall, or (and) an increased pressure in the pulmonary circulation.

3. *Reduplication of second sound at apex*—This has been considered by some not a reduplication of the second heart sound but in reality an accentuation of the early diastolic heart sound—the so-called third heart sound. Thayer considers that "it is probably due to sudden tension of the mitral and, perhaps, at times, tricuspid valves occurring at the end of the first and most rapid phase of diastole." In mitral stenosis with thickening of leaflets you may then get an accentuation of this third sound at the apex.

4. *Reduplication of the pulmonary second*—does also sometimes occur but why? Because of increased pressure in the pulmonary circulation the pulmonary valve close slightly before the aortic? But in hypertension where the aortic valve closes slightly before the pulmonary there is no reduplication. Just as good, if not better theory is that the heart pressing somewhat on the widened pulmonary artery compresses it slightly and causes the three leaves of the valve to close at different times.

5. *Feeble or absent second at apex* and a feeble aortic second.

INFARCTION in spleen, liver, kidneys and lungs is not an infrequent occurrence in mitral stenosis, much more frequent in decompensated cases but also occurring in a definite number of well compensated ones.

CYANOSIS

May or may not be present but is the usual rule. It is present where there is stagnation and increased pressure in the pulmonary circulation and in cases of long standing it is aggravated by sclerosis of the peripheral branches of the pulmonary artery. This type of cyanosis is also seen in some cases of emphysema with sclerosis of the peripheral lung arteries. Rapid marked cyanosis accompanied by acute dyspnoea is due usually to the sudden onset of aricular fibrillation or the occurrence of thrombosis in the left auricle.

JAUNDICE

Is sometimes present in a mild or slight degree due to liver congestion. It may, however, appear in quite a marked form due to haemorrhagic infarcts in the lung with resultant destruction of the extravasated blood and with many of these lung infarcts there is no accompanying haemoptysis.

Oedema

Is not a characteristic of mitral stenosis and even in late cases with lung congestion and blood stained sputum, failing right heart and a large tender liver there may be no peripheral oedema.

Auricular Fibrillation

The older clinicians had neither a conception of the cause and nature of auricular fibrillation, nor a a knowledge of its frequent occurrence in the course of a mitral stenosis. They recognized the character of the pulse which accompanies it, but thought that it was a definite indication of mitral regurgitation and spoke of that pulse as "the mitral pulse." Trousseau, in 1868, only affirmed what was then considered a well known fact when he said "in contraction of the mitral orifice the pulse is regular (but) in insufficiency of the mitral valve the pulse is always irregular and of an irregularity which is absolute." It was not until 1906 that Cushny and Edmunds published the first record of a case they had observed in 1901 and it was 1910 before Lewis, Rothberger, and Winterburg first definitely proved that what was taking place was fibrillation of the auricle.

The condition is not due to damage of the sino-auricular node but to irregular degeneration of the ordinary muscle fibres in the auricular wall. This uneven degeneration is responsible for irregularity in the rate of the transmission of stimuli in various directions from the sino-auricular node through the auricular wall and there is finally aroused the "circus movement" explained by Lewis and auricular fibrillation sets in.

In mitral stenosis auricular fibrillation occurs eventually in the majority of cases; it first appears, in four-fifths of all the cases, in patients over thirty years of age and so signalizes the final defeat of an auricle within the muscular element of whose walls a slow but steady degeneration has been taking place over a period of many years. Its approach is often deduced from the appearance of extra-systoles that gradually occur more and more frequently; or short recurring bouts of fibrillation itself may be noticed before the final permanent total arrhythmia sets in. Its arrival may or may not be noticed by the patient; there may be only some increase in dyspnoea and cyanosis or there may be such acute distress and cardiac embarrassment that the patient dies within a week. If conduction through the A-V bundle is excellent and the left ventricle weak death may occur before digitalization is accomplished; if, on the other hand, there is poor conduction through to the ventricle, the ventricle is healthy and not hypertrophied and, with digitalis, the pulse can be kept below 80, then the patient may live for another fifteen years. The average duration of life after the establishment of a permanent auricular fibrillation in a case of mitral stenosis is, however, about two years.

Wheer auricular fibrillation occurs in mitral stenosis the diagnosis of its presence offers no difficulties—the appearance of a totally irregular pulse, a pulse deficit and the disappearance, if present before,

of the presystolic murmur and the thrill makes the picture clear. If there is still any doubt the electrocardiogram with its absence of "p" waves is characteristic.

It would appear from the enumeration of the various signs and symptoms to be found in mitral stenosis, that this lesion should not be difficult of recognition. That is true in the majority of cases but there are such things as a "silent" mitral stenosis and if a search is not definitely and particularly made for it, it will be overlooked.

1. It is sometimes overlooked because we are satisfied with hearing a systolic at the apex, and the patient is not exercised or examined while lying on the left side; or, in the absence of murmurs, we neither search for a rheumatic history nor give enough weight to those important ancillary signs—the banging apical first sound, the feeble or absent apical second, the accentuated pulmonary second, the small pulse, comparatively low blood pressure, etc.

2. We are too often satisfied with saying "auricular fibrillation" which is not a diagnosis at all; when a further search would reveal the mitral stenosis of which the fibrillation is only an accompaniment. In patients below the age of forty-five, in the absence of syphilis and hypertension, a fibrillating left auricle is nearly always embarrassed by a stenotic mitral valve.

3. A specific search for this lesion should be made in patients with recurring bronchitis and occasional slight haemoptysis with no signs of pulmonary tuberculosis and where there occurs an embolus or infarct with no syphilis, vascular degeneration or hypertension.

4. All doubtful cases should be fluoroscoped for evidence of an enlarged left auricle.

Prevention

If it is true that the antecedent of mitral stenosis is rheumatic fever and that the beginning of the sclerosis occurs in early life and, if it is true, that the fully developed lesion is the result of repeated insults over a period of years, then it should be possible to do something in the way of prevention. Can the medical man by the early recognition of vague indefinite symptoms and the active persistent treatment of all the numerous rheumatic manifestations in children reduce the incidence of the "button-hole" mitral? I think so. In spite of the tonsils being considered a wide-open door for the entrance of the rheumatic virus it has not been proved that tonsillectomy will either prevent an initial attack or ward off a recurrence. At the rate at which children's tonsils are being removed nowadays the next decade should at least produce extensive statistics to conjure with.

Prognosis—depends on many factors.

Environment and Circumstances

If a child with a damaged valve lives in a home where prompt attention is given to every pain, ache and, indisposition and where it

In 1660, Francis Bacon wrote, for James I., a treatise on the "Advancement of Learning." It was a survey of the whole state and extent of the intellectual world at that time. Amongst other things this is what he said of the shortcomings of medicine. "The first is the dis-

is protected from the rough-and-tumble of ordinary school life it may attain adult life without bringing with it an extensively sclerosed valve. Latham's statement that "the man who, having an unsound heart, must traffic with his sinews for his daily bread has a poor chance of benefit from medicine' 'is a true one and in a young adult the chance of acquiring a decent span of life is much brighter where circumstances are such that a living can be earned by other means than by the sweat of his brow. Figures are not yet collected but my impression is that the war veterans who are now drawing pensions for mitral stenosis will certainly live longer than a similar group of their fellows who live a less sheltered life in the struggle for existence. The man who goes out into industry and works for years under the handicap of a damaged heart is, nevertheless, as things are, entitled to his job as long as he can keep his end up and is not a menace to his fellow-workmen. However, one day he suddenly quits and goes home with a cardiac compensation irretrievably lost.

That man is an example of the inadequacy of things as they are. If we can neither prevent his stenosis nor cure it one of several things should be done. Either he should be killed off as soon as a diagnosis is made, given a sheltered job, or, when it has arrived, his broken compensation should be considered an injury just as much as a broken back. Organized sheltered employment for cardiac cases is still in the future with us, complete prevention is as yet but a dream, but it is to be hoped that in the meantime the Act of this Province will be such that the Workmen's Compensation Board can look with friendly eyes on the man with mitral stenosis who spits blood as he waits in their ante-room.

The following concerning prognosis in mitral stenosis are from the statistics of Cabot:

1. The addition of another valve lesion always shortens life.

2. In mitral and aortic cases the disease apparently killed in one year in more than 50%, and the average duration of life was three years compared to fifteen years in mitral stenosis alone.

3. Out of thirty-six cases of mitral stenosis going to autopsy 75% lived ten or more years after onset of disease (and here it probably means onset of symptoms in some of the cases), and six cases lived from twenty-five to thirty-five years.

4. Cabot gives the average duration of life after onset of auricular fibrillation as about two years but some live for another fifteen and in not all of the cases by any means is the expectancy of life shortened by the arrival of fibrillation.

The question of treatment is not a small one and it cannot be compressed within the space of time allotted. In closing may I say just this. To not a few it may have appeared that at times this discussion smacked too much of over-precision and over-emphasis of possibly unessential details; but if we are familiar with those details there will come times in our work when a single one, properly evaluated, may clinch our diagnosis. Not only that but the simple observing and recording of them will assuredly give more confidence and enlarge our mitral stenosis horizon.

continuance of the ancient and serious diligence of Hippocrates, which used to set down a narrative of the special cases of his patients, and how they proceeded, and how they were judged by recovery or death. . . . . This continuance of Medicinal History I find deficient, which I understand neither to be so infinite as to extend to every common case, nor so reserved as to admit none but wonders; for many things are new in the manner, which are not new in the kind, and if men will intend to observe, they shall find much worthy to observe." The question as to whether Bacon's dictum applies equally well to medicine with us, in Vancouver, today I will leave with you.

* * *

INDEX

SONG WITHOUT A MORAL

"With business college she is through"
Says Pa: "What shall our daughter do.
Shall it be banking or insurance,
It worries me beyond endurance,
A wholesale warehouse or a broker
Into what business shall I poke her?"

Said Ma (who had a gentle mind)
"We want our girl to be refined,
Aloof from such vulgarity
The business world must daily see;
She must not even be aware
Of girls who paint and men who swear."

Eventually the day was won
The thought was thunk, the deed was done
In atmosphere both strict and sterile,
They had disposed their little gir-l;
They did decide to make of her
A Medical Stenographer.

Doctors, of course, are simply sweet,
They never curse and stamp their feet,
They never tear their hair and roar,
They never slam a single door,
Their morals must be all perfection
—This was the cause of their selection.

The years roll by, alas, alack,
If we could only turn them back.
Is this the sweet and simple maid
Whose course these parents calmly laid.
Can it be true—oh, say not so
That things so very wrong could go.

With steely eye and firm-set jaw
And sturdy wrist and calloused paw,
A calculating steno she
Can spell your whole anatomy
And with the surgeon stitch by stitch
She knows what part joins on to which.

She knows that in this world of strife,
Internes are lowest forms of life;
And though her sense of humor mocks her,

She sweetly smiles and says "Yes doctor."
Corrects his grammar and his spelling
And never even thinks of telling.

She hears the surgeon fume and roar
And stamp his feet upon the floor;
She hears him shout: "I'll have you fired,"
And inward thinks, "You make me tired."
She knows it is not really meant
Perhaps artistic temperament.

She knows that come what may or might
The doctor's always, *always* right,
Though proof and a conviction strong
Convince her that the doctor's wrong;
Assertive longings may laggress her,
And yet she humbly murmurs, "Yessir."

Up on the glistening O. R. floor
Of anaesthetic smells and gore,
The doctor in his O. R. rig
For nurse or steno cares no fig,
Enough one's modesty to spill
A doctor in his dishabille.

Before her desk himself he stations
And talks of gooey operations
—She very seldom hears him speak,
Perhaps she's thinking of her sheik;
While in some spring-time thought she loiters,
He raves of thyroid glands and goitres.

You think she may be hard and cold
Without the standard heart of gold,
Or too well versed in mental mysteries
from taking psychiatric histories,
But she 'tis only truth to tell
reacts to moonlight just as well.

You most assuredly may list her
As equal to her business sister,
Her notes are all that they should be
Her finished work a charm to see;
In starched white coat she's spick and span,
She never fails to "Get her man."

—*The Joint Effort of Two Lowly Stenos.*

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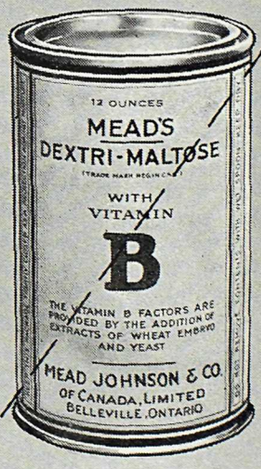

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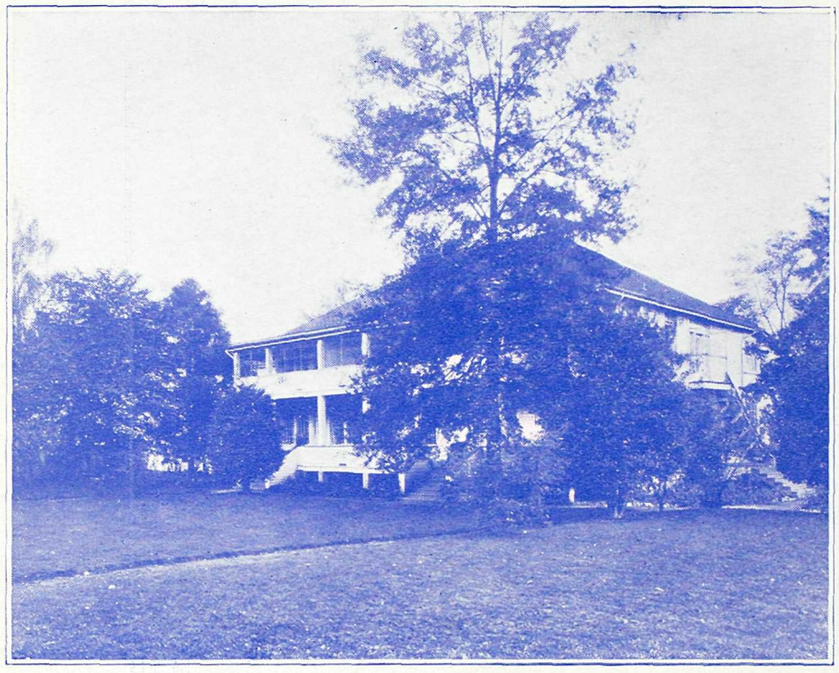

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