Archive

JIMA (Volume XX, No 3, December, 1950, Page 106-9)*

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Recently I came across a paper titled "The importance of valvular defects and arrhythmias of heart and blood pressure determinations in life insurance medicine", by Dr K. Sitapati Rau who was the Medical Referee of the Andhra Insurance Co. Ltd at Masulipatam. The article was presented at the Third International Congress of Life Insurance Medicine held in Rome in 1949. The paper was published in the Journal I.M.A. in December, 1950.

As the entity of "international congress of life insurance medicine" was new to me, I tried to find some information about this. By Google search, I could track down till 1979 when the 13th International Congress of Life Assurance Medicine took place in Madrid. It is to be noted that by then the term "insurance" has been changed to "assurance". Further research revealed that in 2001, in Sydney, the 20th International Congress of Insurance Medicine took place. I think that as a medical graduate very few of us are aware of this stream of insurance, or life insurance, or life assurance medicine. Dr S Rau himself has observed this and proposed that medical students should have an exposure to this stream of medical science. But even after 70 years of publication of this article, situation did not change.

Now, the relevant question is whether we need such an exposure. I see this issue from two separate angles.

Firstly, insurance medicine looks into normal healthy population and follows them to find out the longevity and the factors affecting the same. What appears to be a normal healthy condition or parameter by today's knowledge may prove to be a death determinant in future. The classical example is hypertension. Till the death of USA president F. D. Roosevelt in 1941 due to untreated high blood pressure, medical community did not consider high blood pressure as a treatable entity. Even after that, till late sixties, there was not enough evidence to suggest routine treatment of high blood pressure. However, even in 1950s, the USA insurance companies refused life coverage for people with high blood pressure. So, at least in this particular field, they outsmarted the medical community.

Secondly, the insurance medicine promotes thorough clinical assessment along with available investigative modalities to determine the life expectancy of a person. This is not an easy job. In how many clinical situations we, as medical practitioners, can accurately predict the longevity of our patients? Even when we predict, we may give a casual estimate, as there is no stake involved. But when it matters to the insurance company for the running of their business on the correct predictions of the doctors, the responsibility and pressure on the doctors can well be imagined. That is reflected in the way the clinical assessment is done. This article is a fantastic example of how, by systematic approach on history taking and bedside clinical examination, one can assess the remaining longevity of a person with various cardiac problems. If we remember that the first ever electrocardiography (ECG) machine was used in K.E.M. Hospital, Mumbai, in the late 40's, we can somewhat guess the excellence of this paper which was presented in 1949, talking on cardiac arrhythmias, and how to assess them at bedside, and how to predict outcome of a person with different arrhythmias of heart. This is mind boggling.

Even more astonishing fact is that the echocardiography was first introduced in India again in the K.E.M Hospital and Port Trust Hospital, Mumbai in 1974-75, and the descriptions of the valvular lesions given in this paper was in 1949! Even today, we cannot beat the elaborate way the valve diseases have been described. It is a big lesson to us that even without gadgets, just what a simple stethoscope can achieve. Obviously, the bias of their examination was to predict who could live for how long, but nevertheless, to achieve that, they needed to go through such amazingly thorough examination of a person.

Of course, with time, the perspectives have changed. Most valvular and arrhythmic diseases are now well treatable, and consequently the longevity with such conditions has improved considerably. But, the fundamental premise remains unchanged. And that is the fact that an astute clinician with his thorough bedside clinical approach, can diagnose and prognosticate most conditions even without modern gadgets. The skill of clinical examination, which is sadly receding in the modern era of medicine, can be reappraised by our medical students and teachers by having a read of this phenomenal paper. At the same time, we may get interested to acquire a new skill of predicting the longevity of our patients. I strongly recommend all readers to go through this article and enjoy its richness.

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PRACTITIONERS' CORNER

THE IMPORTANCE OF VALVULAR DEFECTS AND ARRHYTHMIAS OF HEART AND BLOOD PRESSURE DETERMINATIONS IN LIFE INSURANCE MEDICINE*

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Insurance medical examination shall be regarded as a speciality in that a medical examiner's experiences in hospital and private practice may not be sufficient for dealing with applicants for life assurance, as in the former he comes across with diseased persons only, while in the latter he is face to face with persons leading their day to day lives, working hard, playing strenuous games or indulging in pleasures; some of them are sedentary, some jovial and some eccentric. Sometimes he comes across emotional persons (with insurance-tachycardia) too. Moreover, the medical examiner has to use his shrewd commonsense and balanced judgment in assessing human lives, since the human frame is a rare and composite thing and some applicant despite debility, may outlive another with an apparently fine physique.

In view of the rapid advance of medical science and consequently of increased knowledge about the correlation of diseases, it is not justifiable when an applicant has perhaps a valvular heart disease, kidney affection, diabetes, syphilis, high blood pressure or after an operation for nephrectomy, gastro-enterostomy etc., to recommend him down with one stroke of pen for rejection. Such cases require a careful study of all the circumstances which make up the sum-total of his life *i.e.,* his response to the strain (whether he has a tendency to sedentary or active, or eating and drinking habits), his strength of will power, etc.

The medical examiner is expected to equip himself with all modern appliances to arrive at a correct diagnosis and to know that he and the field worker are the two pivots of a life office.

The writer has observed that many substandard lives are being rejected by life offices in view of the absence of proper statistics regarding the after longevity of the previously rejected cases.

Lastly, it is highly desirable that the subject of insurance medical examination may be included as one of the subjects in the post-graduate or specialisation institutions and life offices may send their medical referees periodically to such institutions for courses, refresher or research, in that particular branch of medical science.

Cardiopathies and Life Insurance:
(a) *Valvular defects,*
(b) *Arrhythmias.*

Valvular Defects

Physical diagnosis.—Accurate physical diagnosis is a product of modern era, and is made clinically by means of four traditional procedures: (1) inspection, (2) palpation, (3) percussion, (4) auscultation.† In a post-graduate journal, it is not necessary to go into details regarding the various points observed by these methods.

The real judgment of the doctor is taxed when an organic heart disease has been diagnosed and the author has found the following criteria quite good.

Organic Murmurs: (1) *Mitral regurgitation*—The first sound is audible as *floo;* it radiates towards axilla and back. The second sound is audible as *dub;* the murmur is systolic in time, sometimes, replaces the first sound at the apex (which condition is serious); in this condition, the pulmonary 2nd sound in the 2nd left interspace is accentuated. If the applicant is young under 25, with otherwise flawless personal and family history and if the reserve power of the heart is good with no dilatation, he may be accepted under Pure Endowment Policy of 15 years' term or 10 years' Endowment with a constant lien of 80 per cent on the 2nd half of the term. But if the applicant is obese, he is uninsurable. If the murmur is of rheumatic origin, one should recommend for complete rejection.

(2) *Mitral Stenosis*—The first sound is audible as *lub* sharp and loud or as *lub-dub;* the second sound is heard as *froo;* a presystolic murmur occurs without fibrillation and in aortic insufficiency. In the latter case it is known as Flint's or crescendo murmur. Hirsh Felder showed that a "relative" mitral stenosis, in aortic insufficiency may be caused by the interference of regurgitant blood with the complete opening of mitral orifice. This doubtless accounts for the fact that the quality of Flint's murmur may be identical with that of an organic mitral stenosis. It radiates towards axilla and back. Pulmonary accentuation of pulmonic 2nd sound occurs with pulmonary engorgement; right ventricle hypertrophies in an effort to propel the blood past the narowed mitral orifice; this murmur is heard best at apex within a circle of 2 inches; it is heard best in erect posture; it may disappear on assuming a recumbent posture and may be entirely absent on some days; to avoid missing it, make the proposer lie on his left side or hop or bend or walk upstairs to quicken the heart's action. There are cases of mitral stenosis without murmur which are diagnosable by the sharp accentuated 1st

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*This article was read at the Third International Congress of Life Insurance Medicine held in Rome in the 1st week of June, 1949.

†Extensive discussion in the original article regarding differential diagnosis has been omitted to conserve space—Ed. *J. Indian M. A.*

sound which becomes like the 2nd sound in character especially if there be also accentuation or reduplication of the 2nd sound at both aortic and pulmonary valve regions; if it is accompanied with a rapid pulse, high tensioned, or small or empty it indicates a serious condition. It is best heard at the end of the diastole rather than pre-systole. Compensation may not last very long unlike in regurgitation; and present with small, rapid, irregular alternating and dicrotic pulse and lastly cough, dyspnœa and cyanosis are present. Presystolic thrill is present along the left margin of the heart area and with low blood pressure. It is advisable to reject all cases of murmurs with thrills, and mitral stenosis comes under that category.

Aortic Regurgitation—Murmur begins early in the is more common after the age of 40, but often dates from an attack of "rheumatism" in childhood. It is usually accompanied by regurgitation (a rare isolated valve lesion); is usually loud, long and harsh. A rough thrill is present in erect position, but is heard in recumbent position over the aortic valve; may be transmitted to the apex area while the apex is displaced downward and to the left. One fact to be remembered is that an aortic murmur is loudest at the base and grows fainter towards the apex, while it is just the opposite in the case with mitral murmur; and an aortic murmur is sometimes heard in the mouth, while mitral is not. Applicants of this type of heart disease are uninsurable.

Aortic Regurgitation—Murmur begins early in the diastolic phase; heard best in the left 3rd interspace; also audible over the mitral area, as also in the aortic area and fades out to the median line; usullly replaces the 2nd sound, which over the aortic area is faint; usually loudest in the erect posture and is intensified by exertion; the 2nd sound prolonged, sometimes harsh, radiates upwards into the carotids and sometimes downwards to ensiform cartilage. Pulse-pressure is high on account of high systolic and low diastolic blood pressure. Pulse is that of Corrigan's—"water-hammer" soon full, soon empty. Capillary pulsation detected in mucous membranes of lips and under finger nails; pulsation in supra-sternal notch (due to aortic dilatation). Apex impulse displaced downward and outward and is diffuse and heaving in character, decided increase in dullness in transverse diameter due to the enlarged left ventricle. It is a very common complication of syphilitic aortitis but Wassermann and Kahn tests may often be negative. Rheumatism attacks the mitral valves twice as frequently as it implicates the aortic valve. It is said that aortic valve is most commonly involved in males whereas the mitral valve is usually favoured in females (in the so-called "pure" mitral cases only).

In the case of heart, beating forcibly, slowly and regularly and with enlargement down and out and an aortic murmur in the diastole, a functional causation is out of question; but if therewith, the diastolic pressure is low with high pulse-pressure, in four out of five cases there is grave aortic regurgitation.

In young persons below 25 years of age, such lesion coupled with a rheumatic history may be accepted on ten years Endowment with 80 per cent constant (latter) lien (in the 2nd half of the term); all older persons and all syphilitic subjects with the above lesion are decidedly uninsurable.

Tricuspid Stenosis—Resembles mitral stenosis in character and stenosis of this orifice never exists alone; there is always an associated tricuspid regurgitation.

The strain on the right auricle whose walls are thin, is so great that it cannot compensate with sufficient hypertrophy to overcome the obstruction at the tricuspid orifice; such subjects do not survive very long.

Tricuspid stenosis produces a mild diastolic murmur in the tricuspid area which is limited to the lower half of the sternum and just to the left of it, which is often described as a presystolic one, not accompanied by a thrill.

Tricuspid stenosis exists as a confirmatory evidence of rheumatism which has already attacked the mitral valve (mitral stenosis); there exists increased cardiac dullness to the right, with dilated cervical veins and cyanosis and large pulsating liver; it may also be present in tuberculous subjects.

Pulmonary Regurgitation—Organic lesions are extremely rare; relative insufficiency more common; a soft blowing murmur heard best in the 2nd and 3rd left interspace and close to the sternal margin, transmitted downward along the left sternal border for a short distance and curving slightly towards the apex and best heard during expiration.

Pulmonary Stenosis—It is the rarest of acquired organic valve lesions; a very common congenital anomaly; usually associated with pulmonary regurgitation; systolic thrill present over pulmonic valve area and hypertrophy and dilatation of left ventricle present also. The murmur is harsh, best heard over pulmonic valve area, often distributed diffusely over a wide area on the chest but rarely into the great vessels of the neck (as observed in aortic stenosis). Pulmonic 2nd sound absent or faint, clubbing of the fingers and polycythemia may occur. If pulmonary stenosis is coupled with partial transposition of aorta, it is called "Fallot's tetralogy" which exhibits visible precordial pulsation and bulging and cyanosis with dyspnœa from birth in addition to all the signs and symptoms of pulmonary stenosis. Applicants with this heart valve lesion should be given a short Endowment upto 40 years of age.

HAEMIC MURMURS: These are functional murmurs and are probably due to dilatation of the muscular valve ring or improper closure of healthy valve cusps or to shrinking of the chordæ tendinæ and there is, therefore, some slight regurgitation but it should be clearly understood that the cause is not disease of the valve, but simply imperfect apposition. They are characterised by (1) being usually systolic in time and usually not intensified by exercise. (Organic murmurs are either presystolic, systolic or diastolic). (2) They are usually soft and blowing in character and are never harsh. In febrile diseases, a systolic murmur is often heard in the mitral area and is soft and if harsh is not functional. (3) The pulmonic area is the usual location in the large majority. Since organic pulmonary valve lesions are extremely rare, regard any murmur heard in the pulmonic area as functional. (4) They are accompanied by a venous hum over the right jugular vein; to hear this hum apply the bell of the stethoscope over the sternal attachment of the right sterno-cleido-mastoid muscle, the subject sitting or standing (not recumbent). (5) They are not associated with cardiac hypertrophy or dilatation. (6) They are transitory; persist for sometime in chlorotic (anæmic) subjects or persons with flabby musculature and finally disappear on their being toned up by health or mature growth. (7) They do not replace but only accompany the first sound and (8) are found most in child hood and adolescence and in those adults who are: (a) anæmic or chlorotic subjects; (b) neurotic subjects or those with hearts, which though normal in condition, yet beat exceptionally vigorously or are tachycardiac; (c) subjects with flabby muscles; (d) sedentary persons and especially those with ptosed abdominal organs. (9) They are not transmitted and may disappear in various phases of respiration and hence are termed as cardio-respiratory. Cardio-respiratory murmur is a soft systolic blow at the apex or along the left border of the heart, it is not a murmur; it is the impact of the ventricular systole against the overlying lung, suddenly expelling the air; usually heard only during inspiration or at least, intensified during inspiration. Cases of applicants with such murmurs (functional) should be postponed until the murmurs disappear completely and at subsequent medical examination the term and plan should be decided as per the conditions of the then health of the applicant with or without extra (loading). As already stated presence of thrills indicate organic valvular disease with stenosed valves and hence such cases are uninsurable.

Till a few years ago all applicants with heart murmurs were taken as serious and alarming and hence declared unfit for life assurance.

All the organic valvular diseases of heart should not be thrown under the category of uninsured lives, but some may be considered as substandard lives. Professor Brock Bank, states "it is the condition of cardiac muscle, not of the valve, that determines prognosis; a loud murmur does not necessarily indicate serious disease of the cardiac valves, therefore, the outlook depends entirely on the condition of the cardiac muscle, the weakness or failure of which is the cause of most of the physical signs and symptoms of heart disease".

A mitral regurgitant murmur may be considered as "quiescent" provided there is good cardiac efficiency and with no hypertension; applicant with such lesion may be accepted on short Endowment with 40 per cent latter lien; and a moderate degree of hypertrophy with murmurs may be given double endowment with 6 or 7 years' loading.

A systolic murmur at the apex and at the base is a common feature of many normal hearts and if without cardiac enlargement and with good cardiac reserve, may be ignored.

A young applicant with a mitral systolic murmur and with rheumatic history may be accepted on double endowment short-term policy but one with acute articular rheumatism is not acceptable; applicants with aortic disease even though young are to be declined.

When assessing lives with cardiac valvular disease, one should not ignore the character of the heart sounds —normal, weak or accentuated (*a*) a markedly feeble 2nd sound indicates myocardial degeneration, dilatation and failing cardiac efficiency, which condition is not of ordinary but of very serious significance, (*b*) a ringing aortic sound sugggests aneurysm, arteriosclerosis or regurgitation—such states require rejection. A booming first sound may be accepted on a short endowment with lien or double or triple endowment, but a clear and sharp 2nd sound indicates dilatation which requires rejection. Female applicants with any organic valvular disease of heart are uninsurable in view of maternity risk; and after the onset of menopause they may be placed in the same category as males of similar age with similar lesions of heart.

CARDIAC ARRHYTHMIAS

There are three common irregularities (1) Sinus arrhythmia, (2) Premature beats or extra systolic arrhythmia, (3) Auricular fibrillation.

Sinus Arrhythmia—Irregularity in the cardiac rhythm due to irregular impulse formation in the sino-auricular node is intimately dependent upon variations in vagal tone. It is perhaps the common cause in normal adults in deep breathing and while the rate decreases; very common in childhood with quiet breathing; often the heart rate will be noticed to flow at the end of inspiration and again at the end of expiration; the important fact is that changes in rate and rhythm

can almost always be induced by the full inspiration or full expiration; nor is it proper to think of this irregular heart beat as a gradual waxing and waning in rate; the changes are often very abrupt. There is another type described by Lewis as "phasic" variation because the rhythm is characterised by alternation of rapid and slow phases; not related to respiration; and changes from the faster to slower rate not abrupt, but extends over a few beats. There is still a third type of disturbance of heart's rhythm which may be properly described as a sinus disturbance, though seldom designated as sinus arrhythmia, which consists of sudden fall of heart rate to very low levels (30 to 40 per minute) associated with a corresponding fall in blood pressure; such changes are brought about by strong vagal influences and often lead to fainting or to extreme giddinesss; they are chiefly responsible for the loss of consciousness frequently observed in people exposed to emotional influences such as the sight of an injured patient, the withdrawal of blood or similar events.

It is abolished by increasing the rate over 100, disappears after exertion and febrile state; also after administration of amyl nitrite or atropine (vagal stimulation), (digitalis often causes sinus arrhythmia); may sometimes he observed during convalescence. Differentiated from auricular fibrillation by increasing the rate of the heart, auricular fibrillation becomes intensified while sinus arrhythmia becomes abolished when the heart rate is raised.

Premature Beats or Extra-Systolic Arrhythmia—Premature beats (commonly but erroneously called extra systole) are cardiac contractions which disturb the regular rhythm arising in response to impulses generated in some portion of the heart other than the sino-auricular node; premature beats may occur at almost all ages from early infancy to ninth decade, but are relatively infrequent in infancy and childhood, but are more common in males than females. It is mainly due to causes outside the heart such as (*i*) a local chronic focal infection (such as tonsillitis or infected tooth and disappears permanently after their removal), (*ii*) gastro-intestinal disturbances more especially chronic disease of the gall bladder, (*iii*) over-digitalization, (*iv*) excessive indulgence in tea, coffee, tobacco or alcohol. The subject has a feeling of constriction in throat, as if the heart was "stopped" or "turning over"; transient weakness or giddiness; precordial distress, sometimes substernal pain (pain due to forcible contractions which follow the long diastolic pause); radial pulse—feeble and small; both heart sounds weak or 2nd sound absent; increased in frequency by deep inspiration or holding the breath and by fatigue; disappear if the heart rate is increased. The diagnosis of premature beats, in most cases, occasions little difficulty, although it is often difficult or impossible to distinguish between those of auricular or of ventricular origin. Recognition usually depends upon auscultation of the heart rather than upon palpation of the radial pulse; in ventricular premature beats, the auscultatory signs will vary, depending upon whether or not the contraction of the ventricle lifts or does not lift the aortic valves. If it does, the rhythmic sounds of the heart are suddenly interrupted by a similar 1st and 2nd sounds occurring much sooner than expected and followed by a fairly long pause. The regular rhythm is again resumed only to be similarly disturbed again after an interval. In this instance, the premature beat is felt at the wrist as a small wave occurring prematurely followed by a pause. If the abnormal ventricular beat occurs so prematurely that it fails to lift the aortic valves, auscultation will reveal merely a single sound interrupting the regular rhythm followed by a long pause and the radial pulse the entire omission of a beat. The signs may also vary with the frequency of premature beats; if they follow every normal beat and do not lift the aortic valves there will be heard a regular succession of three sounds or "pulsus trigeminus" followed by a long pause, the two normal heart sounds and the single sound due to the premature beats.

There is always a conspicuous pulsation of cervical veins especially on the right side, synchronous with the premature beats; this is due to the fact that auricular systole coincides with the contraction of the ventricle, pumps its blood into the cervical veins instead of into the ventricles. Auricular premature beats are less frequent and in general less important than those which arise in ventricle and are recognised clinically in the same way as the ventricular type but they differ in that the length of the pause following the premature contraction is usually less than compensatory. It is seldom important to distinguish between the two types which can be elicited by means of electrocardiograms only. Lastly, premature beats are usually ignored but in applicants over 35 years of age when the condition may point to myocarditis, they should indicate the applicant as uninsurable.

Auricular Fibrillation—It is the most important and one of the most frequent cardiac arrhythmias. The rate of auricles is usually much higher than in auricular flutter (400 to 600 per minute). The ventricular rate is from 90 to 160 per minute and fibrillation is about 35 times more common than flutter and is common in mitral stenosis, degenerative myocardial diseases (arteriosclerosis, etc.), and thyrotoxicosis; violent throbbing in precordia present and vertigo and weakness as well.

Conspicuous Pulse Deficit—Premature beats and auricular fibrillation are the two sole causes of pulse deficits. Posture, exertion, excitement, nitrites, or atropine, all fail to influence the auricular rate but they will change the ventricular rate and even more than normally. Fibrillation of auricles does add one element of danger; thrombus formation upon the walls of the dilated and quiescent auricles and fragments of this mural thrombus may at any time break off and become emboli in the pulmonary or systemic circulation; and death from pulmonary or cerebral infection is not uncommon. Any grossly and irregular pulse rate about 110 per minute is hightly suspicious of auri-