COMPLETE HEART BLOCK
CLINICAL MANIFESTATIONS AND MODES OF CARDIAC PACING

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خلق الإنسان (3) علمه البيان (4)

صدق الله العظيم

سورة ال Rahman آية رقم (1-4)
Dedication

To

My Parents Who Gave Me

The Taste Of Life …

To

My Husband & Kids Hoping Them Happy

Successful Future …

Wisal
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Special thanks and gratitude to the medical directors, staff in the catheter laboratory and statistical employee in SHC for their great help.

Many thanks to all people who willingly participated in the study.
ABSTRACT

CHB is a common disease especially among elderly people.

Objectives: The aim of the study is to know the clinical manifestations of CHB and to assess the different modes of cardiac pacing and their short-term outcome.

Methods: Descriptive analytical cross-sectional study that was performed in SHC, 70 patients who presented with CHB and underwent cardiac pacing were studied.

Data analyzed and tabulated including personal data, presenting symptoms and signs, investigations, modes of cardiac pacing and their outcome.

Results: Male to female ratio 1.3 : 1.

The main presenting symptoms were S.O.B 52.3% syncope 45.7% and dizziness 40%.

The main signs were bradycardia 88.4%, systolic hypertension 45.7% and signs of congestive heart failure 25.7%.

ECG is the diagnostic investigation.

Different modes of cardiac pacing were implanted including VDD 54.3%, VVI 25.7% and DDD 12.9%.

Short-term outcome of cardiac pacing is good, 91.4% of the patients improved.

Conclusion: CHB is a common disease in the elderly people and cardiac pacing is a life saving procedure.
ملخص الأطروحة

هدف الدراسة:
دراسة الأعراض والعلامات الناتجة من مرض إحصار القلب الكامل والتعرف على الأنواع المختلفة من أجهزة تنظيم ضربات القلب المستخدمة ونتائجها.

الطريقة:
هذه دراسة وصفية تحليلية أجريت على 70 مريض يعانون من إحصار القلب وقد أجريت لهم عملية زراعة جهاز تنظيم ضربات القلب بمركز السودان للقلب.
تم جمع المعلومات باستخدام استبيان عن المرض واعراضه وعلاماته، وسائل تشخيصه، الأنواع المختلفة من أجهزة تنظيم ضربات القلب التي تم استخدامها والنتائج المرتبة على ذلك.

النتائج:
- نسبة الذكور للإناث 1.3 : 1.
- أهم الأعراض ضيق في التنفس 52.3% ، إغماء 45.7% ، دوخان 40%.
- أهم العلامات بطء شديد في معدل ضربات القلب 88.4% ، ارتفاع ضغط الدم 45.7% ، هبوط في القلب 25.7%.
- 91.4% من المرضى تحسنت حالتهم بعد عملية زراعة جهاز تنظيم ضربات القلب.

الخلاصة:
إحصار القلب الكامل مرض منتشر عند المسنين وعملية زراعة جهاز تنظيم ضربات القلب تنقذ حياتهم.
<table>
<thead>
<tr>
<th>Abbreviation</th>
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<tr>
<td>AV</td>
<td>Atrioventricular</td>
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<tr>
<td>AVR</td>
<td>Aortic Valve Replacement</td>
</tr>
<tr>
<td>BP</td>
<td>Blood Pressure</td>
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<tr>
<td>BPEG</td>
<td>British Pacing &amp; Electrophysiology Group</td>
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<tr>
<td>CHB</td>
<td>Complete Heart Block</td>
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<td>DCM</td>
<td>Dilated Cardiomyopathy</td>
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<td>DM</td>
<td>Diabetes Mellitus</td>
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<td>ECG</td>
<td>Electrocardiograph</td>
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<td>ECHO</td>
<td>Echocardiography</td>
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<tr>
<td>HTN</td>
<td>Hypertension</td>
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<tr>
<td>IHD</td>
<td>Ischaemic Heart Disease</td>
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<td>IVCD</td>
<td>Intraventricular Conduction Disturbances</td>
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<tr>
<td>JVP</td>
<td>Jugular Venous Pressure</td>
</tr>
<tr>
<td>LVH</td>
<td>Left Ventricular Hypertrophy</td>
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<tr>
<td>MI</td>
<td>Myocardial Infarction</td>
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<tr>
<td>MRI</td>
<td>Magnetic Resonance Imaging</td>
</tr>
<tr>
<td>NASPE</td>
<td>North American Society of Pacing and Electrophysiology</td>
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<tr>
<td>NBG</td>
<td>North American &amp; British Pacing &amp; Electrophysiology Group</td>
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<tr>
<td>SA</td>
<td>Sinoatrial</td>
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<tr>
<td>SDH</td>
<td>Subdural Haemorrhage</td>
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<td>SPSS</td>
<td>Statistical Package for Social Sciences</td>
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The Conduction System Of The Heart

Each natural heart beat begins in the heart's pacemaker-the sinoatrial (SA) node. This is a crescent-shaped structure that is located around the medial and anterior aspect of the junction between the superior vena cava and the right atrium. Progressive loss of the diastolic resting membrane potential is followed, when the threshold potential has been reached, by a more rapid depolarization of the sinus node tissue, and this triggers depolarization of the atrial myocardium. The atrial tissue is activated like a forest fire, but activation goes out when the insulting layer between the atrium and ventricle—the annulus fibrosus—is reached.

The conductive system of the heart is organized so that the cardiac impulse will travel from the atria to the ventricles slowly. This delay allows time for the atria to empty their contents into the ventricles before ventricular contraction begins. It is primarily the atrioventricular (AV) node and its adjacent conductive fibres that delay the transmission of the cardiac impulse partly because of their considerably smaller size than that of the normal atrial muscle fibres. However, most of the slow conduction is probably caused by two other different factors. First, all
these fibres have resting membrane potentials that are much less negative than the normal resting potential of other cardiac muscle. Second, few gap junctions connect the successive muscle cells in the pathway, so there is great resistance to the conduction of excitatory ions from one cell to the next.

The atrioventricular (AV) node is a small bean-shaped structure that lies beneath the right atrial endocardium within the lower interatrial septum. It continues as the His bundle, which penetrates the annulus fibrosus and conducts the cardiac impulse rapidly towards the ventricles. The His bundle divides into the right and main left bundle branches. The right bundle branch continues down the right side of the interventricular septum to the apex, from where it radiates to form the Purkinje network, which spreads throughout the subendocardial surface of the right ventricle. The main left bundle branch is a short structure, which fans out into many strands on the left side of the interventricular septum, and these strands can be grouped into the anterior hemibundle which supplies the subendocardial Purkinje network of the anterior and superior surfaces of the left ventricle, and the posterior hemibundle that supplies the inferior and posterior surfaces. The impulse conduction through the AV node depends on the action potentials largely produced by slow transmembrane calcium influx, while that in the atria, ventricles and His-Purkinje system is due to action potentials generated by rapid transmembrane sodium diffusions.\(^{(1,2)}\)
Definition of Atrioventricular Block:

A trioventricular (AV) block can be defined as a delay or interruption in the transmission of a cardiac impulse from the atria to the ventricles due to an anatomical or functional impairment in the conduction system. This can be transient or permanent. The conduction can be delayed, intermittent, or absent resulting in first degree (slowed conduction), second degree (missed beats, often in a regular pattern), and third degree or complete AV block respectively. (show ECG).

First Degree AV Block:

During first degree AV block, every atrial impulse conducts to the ventricles, producing a regular ventricular rate, but the P-R interval exceeds 0-2 seconds in the adult. Sometimes the P-R interval is very prolonged exceeding the P-P interval, a phenomenon known as “Skipped” P waves. Clinically important P-R interval prolongation can result from conduction delay in the AV node (A-H interval), in the His-Purkinje system (H-V interval), or at both sites.

Acceleration of the atrial rate or enhancement of vagal tone by carotid massage can cause first degree AV nodal block to progress to type I second degree AV block. Conversely, type I second degree AV nodal block can revert to first degree block with deceleration of the sinus rate.
Second Degree AV Block:

The block of some atrial impulses conducted to the ventricle at a time when physiological interference is not involved constitutes second degree AV block.

Wenckebach and Hay, by analyzing the $a-c$ and $V$ waves in the JVP, described two types of second degree AV block. After the introduction of the electrocardiograph, Mobitz classified them as type I and type II. Electrocardiographically, typical type I second degree AV block (Wenckebach or Mobitz type I) is characterized by progressive P-R prolongation culminating in a nonconducted P wave, while in type II second degree AV block (Mobitz type II), the P-R interval remains constant prior to the blocked P wave. In most instances, the differentiation between type I and type II second degree AV blocks can be made easily and reliably from the surface ECG. Often type II AV block can progress to complete AV block with development of Stokes-Adams syncope, while type I AV block is generally more benign and does not progress to more advanced forms of AV conduction disturbances.

In a patient with an acute myocardial infarction (MI), type I AV block usually accompanies inferior MI, is transient, and does not require temporary pacing, whereas type II AV block occur in the setting of anterior MI, can require temporary or permanent pacing, and is associated with high rate of mortality, generally due to pump failure$^3$. 
Third Degree (Complete) AV Block:

Third degree is a disorder of the cardiac conduction system with complete absence of AV conduction. No P waves conduct to the ventricles, and AV dissociation is complete.
Pathophysiology of Complete AV block:

In complete (AV) block no atrial impulses reach the ventricles. The block can exist in the AV node or in the infranodal specialized conduction system \(^\text{4-6}\). A His-bundle electrocardiographic study can determine the site of the block quite accurately, but the escape rhythm can provide important clues.

The escape rhythms occur when a pacemaker other than the SA node has sufficient time to depolarize, attain threshold, and produce a depolarization. In CHB, the escape rhythm that controls the ventricles can occur at any level below that of the conduction block and the morphology of the QRS complexes can help to determine the location at which the escape rhythm pacemaker is occurring:

- If the block occurs at the AV node, about two-thirds of the escape rhythms have a narrow QRS complexes, i.e., a junctional or AV nodal rhythm.\(^\text{7-9}\)

- Block at the level of the bundle of His is also typically with a narrow QRS complex.

- Patients with trifascicular block have a subjunctional escape rhythm with a wide QRS complex.

- If the escape rhythm has a normal QRS complex, the block occurs with almost equal frequency in the AV node and the bundle of His.\(^\text{6}\)

As a general rule, the more distal the block, the slower will be the escape pacemakers, and those patients are haemodynamically unstable.
The effects of autonomic tone, exercise, and atropine may all be helpful in determining the site of the block.\textsuperscript{(8,10-15)} High junctional pacemakers may respond with an increase in the heart rate to catecholamines, exercise, or atropine, and a decrease in heart rate after vagal maneuvers. In comparison, lower pacemakers are less responsive to autonomic manipulations.

**Etiology of CHB:**

Many processes can affect the atrioventricular (AV) conduction system. However, the most common are fibrosis and sclerosis (sclerodegenerative changes) of the conduction system and ischemic heart disease.

**Fibrosis and scleroses** – Fibrosis and sclerosis of the conduction system accounts for about one-half of cases of AV block and may be induced by several different conditions which often cannot be distinguished clinically\textsuperscript{(16)}.

Lenègre's disease, for example, is a sclerodegenerative affliction of the conduction system with some predilection for the right bundle branch and the left anterior fascicle in individuals over 50 years of age\textsuperscript{(17)}. It is frequently associated with slow progression to complete heart block.

Lev's disease \textsuperscript{(18,19)} is caused by fibrosis or calcification extending from any of the fibrous structures adjacent to the conduction system into the conduction system. Fibrosis of the top of the muscular septum is a common cause of right bundle branch block with left anterior fascicular block in the elderly patient. Calcification or fibrosis can also extend from the mitral or aortic valve rings into the conduction...
system. Involvement of the mitral ring or the central fibrous body, for example, may be the most common cause of complete heart block with a narrow QRS complex in the elderly. Aortic valve calcification, on the other hand, can invade the bundle of His, the right and/or left bundle branch as well as the left anterior fascicle. Thus, the QRS complex may be prolonged.

**Ischemic heart disease** – Ischemic disease accounts for about 40 percent of cases of AV block (16). Conduction can be disturbed with either chronic ischemic disease or during an acute myocardial infarction (20-24). It is estimated that approximately 20 percent of patients with an acute myocardial infarction develop AV block: 8 percent with first degree; 5 percent with second degree; and 6 percent with third degree (22-24). Intraventricular conduction disturbances (IVCDs), including bundle and fascicular blocks, also occur in 10 to 20 percent of cases of acute myocardial infarction (25-31). Left bundle branch block and right bundle branch block with left anterior fascicle block are most common, each occurring in about one-third of patients with an IVCD (29). RBBB with or without left posterior fascicular block and alternating bundle branch block are less frequently seen while isolated left anterior or posterior fascicle block is distinctly unusual

**Drugs** – A variety of drugs can impair conduction and cause AV block. Examples include digitalis, calcium channel blockers (especially verapamil and to a lesser extent diltiazem), amiodarone and β-blockers (32,33). In comparison, antiarrhythmic drugs that modulate the sodium channel, such as quinidine, procainamide, and
disopyramide, can produce block in the more distal His-Purkinje system.

**Increased vagal tone** – Enhanced vagal tone due to pain, carotid sinus massage, or hypersensitive carotid sinus syndrome can result in slowing of the sinus rate and/or the development of AV block.

**Valvular disease** – As noted above, calcification and fibrosis of the aortic or mitral valve rings can extend into the conducting system. In addition, AV block is not uncommonly associated with replacement of a calcified aortic or mitral valve \(^{(34)}\), closure of a ventricular septal defect, or other surgical procedures \(^{(19,35-37)}\).

**Congenital heart disease** – Congenital complete heart block may be an isolated lesion or may be associated with other types of congenital heart disease.

**Others** – AV block can also occur in a variety of other disorders:

- Cardiomyopathies, including hypertrophic obstructive cardiomyopathy and infiltrative processes such as amyloidosis and sarcoidosis \(^{(18,19,38)}\).
- Myocarditis due to rheumatic fever, diphtheria, viruses, systemic lupus erythematosus, toxoplasmosis, bacterial endocarditis, syphilis, and other causes \(^{(39-49)}\). The development of AV block in this setting is often a poor prognostic sign.
- Hyperkalemia, usually when the plasma potassium concentration is above 6.3 meq/L \(^{(50-52)}\).
- Infiltrative malignancies, such as Hodgkin's disease and other lymphomas, and multiple myeloma \(^{(18,19)}\).
• Neuromuscular heredodegenerative disease\textsuperscript{(17-19,53-55)}, dermatomyositis, rheumatoid disease, Paget's disease, hyperthyroidism, and myxedema \textsuperscript{(18,19)}.
• Cardiac tumors, cysts \textsuperscript{(17-19,56)}, myocardial bridging \textsuperscript{(57)}, and trauma\textsuperscript{(18,19,58)}.

**Clinical Manifestations:**

**History:**

Patients may be asymptomatic, or may have minimal symptoms, usually those with narrow complex escape rhythm, related to hypoperfusion including fatigue, dizziness, and impaired exercise tolerance\textsuperscript{(59)}.

More commonly, patients are profoundly symptomatic mostly due to bradycardia. They present frequently with dizziness or syncope. These are disturbances of consciousness due to an associated profound fall in blood pressure, which can also be the result of tachycardia. Cardiac syncope occurs typically without warning and often with resultant self-injury. These attacks, known as Stokes-Adams syncope, are usually of short duration and self-limiting but they have mortal potential. During the attack the unconscious patient is pale but if the episode is prolonged cyanosis ensues with epileptiform seizures. On recovery the patient flushes and full consciousness is restored rapidly, usually without amnesia or neurological sequelae, but on occasion neurological damage may result from systemic embolism, particularly in
SA node disease, or may be the consequence of co-existing local cerebral arterial disease.

Another presentation is with congestive cardiac failure. In this context presentation is more often of a bradycardia than sustained tachycardia. Severe bradycardia, particularly in the elderly may result in mental confusion, dementia or renal dysfunction due to low cardiac output.

It is important to recognize that recurrent falls may be due to transient disturbances of consciousness when cases may be referred to orthopaedic, geriatric or neurological departments.

There is no difference in incidence of bradycardia due to SA node disease or AV block between the sexes and the average age of presentation is 70 years\(^{(60)}\).

Patients also may have associated symptoms of acute (MI)\(^{(59)}\).

**Physical Examination:**

Physical examination will be notable for bradycardia which may be profound.

The physical findings include a variable intensity of the first heart sound as the P-R interval changes, atrial sounds, and \(a\) waves in the JVP lacking a consistent relationship to ventricular contraction. Intermittent large (Cannon) \(a\) waves may be seen in the JVP when atrial and ventricular contraction occur simultaneously. The second heart sound can split normally or paradoxically, depending on the manner of
ventricular activation. A premature beat representing a ventricular capture can interrupt a regular heart rhythm.

When the ventricular rate exceeds the atrial rate, a cardiac increase in intensity of the first heart sound is produced as the P-R internal shortens followed by a very loud sound (bruit de cannon). This is followed by a sudden reduction in intensity of the first heart sound and the appearance of giant $a$ waves$^{(3)}$.

Symptomatic patients may have signs of hypoperfusion, including:

- Hypotension.
- Altered mental status.
- Lethargy.

Signs of congestive heart failure may be present.

In patients with concomitant MI, signs of acute MI may be evident.

Regularized atrial fibrillation is the classic sign of CHB due to digitalis toxicity$^{(59)}$.

**Treatment:**

Therapy begins by looking for and correcting reversible causes of slowed conduction such as myocardial ischemia, increased vagal tone, and drugs that depress conduction.

The recommendations contained in the Report of the American College of Cardiology/American Heart Association Task Force on
Practice Guidelines (Committee on Pacemaker Implantation) for insertion of a pacemaker for third degree AV block are as follows\(^{(61)}\).

**GROUP 1: DEFINITE INDICATIONS FOR PACING** – Group 1 includes conditions in which implantation of a cardiac pacemaker is considered acceptable and necessary, provided that the condition is chronic or recurrent and is not due to a transient cause, such as acute myocardial infarction, drug interaction, and electrolyte imbalance. If the rhythm disturbance is chronic or recurrent, a single episode of a symptom such as syncope, presyncope, or seizure is sufficient to establish medical necessity.

Group 1 indications include:

- Acquired complete atrioventricular (AV) block. Some controversy exists concerning pacing in asymptomatic patients. The current ACC/AHA Guidelines continue to classify asymptomatic third degree AV block, at any anatomic site, with average awake ventricular rates of 40 bpm or faster as a Class IIA indication\(^{(61)}\). Nevertheless, others consider this condition a group 1 indication\(^{(62)}\). There are many disorders that may be responsible for acquired AV block.

- Following an acute myocardial infarction when there is persistent second degree AV block in the His-Purkinje system with bilateral bundle branch block or third degree AV block within or below the His-Purkinje system, transient advanced infranodal AV block and associated bundle branch block, and persistent and symptomatic second or third degree AV block

- Congenital complete heart block with severe bradycardia, significant symptoms, wide QRS escape rhythm, ventricular
dysfunction, or, if asymptomatic, ventricular rates <50 to 55 bpm in an infant and <70 bpm when associated with congenital heart disease

• Symptomatic Mobitz II AV block.

Asymptomatic Mobitz II second degree AV block is considered a class IIA indication for pacing in the most recent ACC/AHA guidelines under the category of "acquired AV block", but it is a class I indication under the category of "chronic bifascicular and trifascicular block". Many would agree that this should routinely be considered a class I indication for pacing (63).

• Symptomatic Mobitz I AV block (64).

• Symptomatic sinus bradycardia in which the symptoms are clearly related to the bradycardia. The most significant symptoms include syncope, seizures, congestive heart failure, dizziness, or confusion in patients with a heart rate below 40 beats/min. Symptomatic chronotropic incompetence is also a Class I indication for pacing.

• Neurocardiogenic syndromes with significant symptoms including but not limited to syncope and near-syncope.

Other indications for pacing – Other indications for pacing include:

• A less distinct group of patients with sinus bradycardia of lesser severity (heart rate >40 beats/min) who complain of dizziness or confusion that correlates with the slower rates

• Bradycardia that is the consequence of long-term drug therapy for which there is no acceptable alternative

• Bradycardia-induced ventricular arrhythmia or tachycardia which is potentially life-threatening.
• Exercise-induced AV block, even if asymptomatic, that is not demonstrated to be secondary to myocardial ischemia. In the absence of ischemia, almost all cases of exercise-induced AV block are due to disease of the His-Purkinje system \(^{63}\).

**GROUP 2: POSSIBLE INDICATIONS FOR PACING** – Group 2 includes conditions in which use of a cardiac pacemaker may be acceptable or necessary, provided that the medical history and prognosis of the patient can be documented and that there is evidence that pacemaker implantation will assist in overall management.

• Asymptomatic type 1 second degree AV block at intra- or infra-His levels when found incidentally at the time of electrophysiologic study performed for other reasons.

• First degree AV block when there is hemodynamic compromise because of effective AV dissociation secondary to a very long PR interval.

• Congenital complete heart block, beyond the first year of life, with an average heart rate <50 bpm, or with abrupt pauses in the ventricular rate which are double or triple the basic cycle length.

• Bifascicular or trifascicular block accompanied by syncope that is attributed to transient complete heart block after other plausible causes of syncope have been reasonably excluded.

• Markedly prolonged HV interval (>100 ms) in an asymptomatic patient or pacing-induced infra-His block that is not physiologic and which is found incidentally at the time of electrophysiologic study performed for other reasons.
• Persistent second or third degree AV block at the AV node level following an acute myocardial infarction.
• Asymptomatic Mobitz II second-degree AV block.
• Symptomatic sinus bradycardia (heart rate <40 beats/min) in which there is not a clear association between the bradycardia and significant symptoms. Chronic heart rates of <30 bpm in the awake and minimally symptomatic patient is considered a class IIB indication.
• In the patient with neurocardiogenic syncope, recurrent syncope without clear provocative events and with an abnormal response to carotid sinus massage is considered a class IIA indication. Neurally mediated syncope with significant bradycardia that can be reproduced by a tilt study or other provocative maneuvers is considered a Class IIB indication.
• Pacing in the patient with hypertrophic cardiomyopathy when there is medically refractory, symptomatic patients with significant resting or provoked LV outflow obstruction is now considered a Class IIB indication (6). 
• Symptomatic, drug refractory dilated cardiomyopathy with first degree AV block when acute hemodynamic studies have demonstrated hemodynamic benefit from pacing is considered a class IIB indication.
• Overdrive pacing in patients with recurrent and refractory ventricular tachycardia to prevent ventricular tachycardia.

**GROUP 3: PACING NOT INDICATED** – The following conditions are considered to lack adequate evidence of benefit from permanent
pacing; as a result, they should not be considered appropriate indications for permanent pacing.

- Syncope of undetermined etiology. This may require extensive investigation, including ambulatory monitoring, neurological evaluation, and electrophysiologic testing (including tilt-table testing). Cardiac pacing may be considered if the history strongly suggests that the syncope is of cardiogenic origin. In such cases, the patient must understand that permanent pacing may not alleviate the symptoms, since no correlation between symptoms and rhythm has been documented.
  - Sinus bradycardia without significant symptoms.
  - Sinoatrial block or sinus arrest without significant symptoms.
  - Asymptomatic prolonged RR intervals with atrial fibrillation or other causes of transient ventricular pause.
  - Asymptomatic bradycardia during sleep.
  - Asymptomatic second-degree Mobitz I (Wenckebach) AV block.
  - Right bundle branch block with left axis deviation without syncope or other symptoms of intermittent AV block.
  - Reversible AV block, such as those associated with electrolyte abnormalities, Lyme disease, sleep apnea, enhanced vagal tone, and some cases that occur postoperatively (63).
NOMENCLATURE - A three-letter code describing the basic function of the various pacing systems was first proposed in 1974 by a combined task force from the American Heart Association and the American College of Cardiology. Since that time, responsibility for periodically updating the code has been assumed by a committee consisting of members of the North American Society of Pacing and Electrophysiology (NASPE) and the British Pacing and Electrophysiology Group (BPEG). The code is designated the NBG code for pacing nomenclature; it has five positions, with the fifth position being rarely used (show table)(65).

The code is generic and does not describe specific or unique functional characteristics for each device. The first three positions are restricted to bradycardia support pacing.

**Position I** – The first position reflects the chamber or chambers in which stimulation and pacing occur. A refers to the atrium; V indicates the ventricle; and D means dual chamber, i.e., both atrium and ventricle.

**Position II** – The second position refers to the chamber or chambers in which sensing occurs. The letters are the same as those for the first position (i.e., A refers to atrium, V for ventricle, D means dual chamber (atrium and ventricle), and O for absence of sensing).

Manufacturers also use S in both the first and second positions to indicate that the device is capable of pacing only a single cardiac chamber. Once the device is implanted and connected to a lead in either the atrium or the ventricle, S should be changed to either A or V.
in the clinical record to reflect the chamber in which pacing and sensing are occurring.

**Position III** – The third position refers to the mode of sensing, or how the pacemaker responds to a sensed event.

- **I** indicates that a sensed event inhibits the output pulse and causes the pacemaker to recycle for one or more timing cycles.
- **T** means that an output pulse is triggered in response to a sensed event.
- **D** means that there are dual modes of response. This designation is restricted to dual chamber systems. An event sensed in the atrium inhibits the atrial output but triggers a ventricular output. Unlike the single chamber triggered mode, in which an output pulse is triggered immediately on sensing, there is a delay in the dual chamber mode between the sensed atrial event and the triggered ventricular output to mimic the normal PR interval. If a native ventricular signal or R wave is sensed, it will inhibit the ventricular output and possibly even the atrial output, depending upon where sensing occurs.

**Position IV** – The fourth position of the code reflects both programmability and rate modulation.

- **R** in the fourth position indicates that the pacemaker incorporates a sensor to control the rate independently of intrinsic electrical activity of the heart (i.e., rate responsive or rate adaptive pacing). Such pacemakers have the capability of increasing their pacing rate during exercise in response to vibration, minute ventilation, temperature, oxygen saturation, or other stimuli. Virtually all pacemakers with a sensor also have extensive telemetric and programmable
capabilities. There is insufficient space within the code to indicate which sensor is being used. From a practical standpoint, R is the only indicator commonly used in the fourth position.

- O indicates that none of the settings of the pacing system can be noninvasively altered.
- P is simple programmability; one or two variables can be changed, but this code does not specify which ones.
- M is multiparameter programmability, which means that three or more parameters can be changed.
- C reflects the ability of the pacemaker to communicate with the programmer; namely, it has telemetry. By convention, it also means that the pacemaker has multiparameter programmability.

Position V – The fifth position is restricted to antitachycardia functions and is rarely used

TECHNOLOGY OF PACEMAKERS:

Pacemakers are manufactured under rigorous conditions of cleanliness and quality assurance. The unit, weighing 25 to 40 g, contains a battery, which is commonly lithium-iodine cell developed especially for pacemakers. This cell has the advantage of high power to weight and size ratios, predictable discharge behaviour, and long life up to 10 years with low current drain from circuit and electrode. The other major component is the pulse forming circuitry, which of hybrid type using a silicone chip with the addition of a few discrete components. All
pacemakers have a, sensing, capability where an amplifier picks up spontaneous cardiac activity and uses this to recycle the pacemaker’s output. Thus, if the patient returns to normal sinus rhythm, the pacemaker output is inhibited; as soon as the rate of the spontaneous rhythm falls below the set rate of the pacemaker, stimulation of the heart commences. This is known as demand function. Pacemakers now are programmable and telemetric by means of an external programmer which can communicate bidirectionally by radiofrequency. It can receive the present setting of rate, output, sensitivity and refractory periods as well as information on lead and battery impedance and current drains allowing assessment of the remaining battery life and settings to adjust the pacemaker to suit the patient’s need or prolong pacemaker life^{(60)}.

**TECHNIQUES OF PACEMAKER IMPLANTATION:**

The implantation of a pacemaker is a surgical procedure requiring full sterile precautions performed by surgeons or cardiologists usually under local anaesthesia. The central cephalic approach by incision or the subclavian percutaneously are veins the veins of choice for the lead, which consist of a multistrand conductor wire insulated by silicone rubber or polyurethane with an electrode tip usually of platinum alloy and a connector plug at the distal end. The lead is passed with the aid of
fluoroscopy and stiffening stylet in the core of the lead to the right atrium through the tricuspid valve, and positioned in the apex of the right ventricle. Lateral fluoroscopy is useful to confirm the position. Close to the electrode an anchoring device is fitted to the lead, which engages the right ventricular trabeculae. The most effective of these is a group of tines, which project outwards and backwards from the tips at an angle of 60 degrees. If an atrial lead is to be used it is guided through the same vein to the right atrium. Once the lead is in position tests are made of the signal that the heart will convey to the pacemaker via the lead (electrograms) and the ability of an external pacemaker attached to the lead to pace the heart (threshold testing). If the lead is found satisfactory, it is sutured in two layers, in the veins and the subcutaneous tissues and a subcutaneous pocket over pectoralis major is constructed for the pacemaker, which is attached to the lead and implanted. Prophylactic antibiotics are recommended for a few days after surgery. The patient is mobilized on the same day and discharged the next (60).
Complications of pacemaker insertion:

Complications are few but include the following:\(^{(2)}\)

- Infection; when a pacemaker system is infected, antibiotic treatment is not sufficient and the pacemaker must usually be removed before antibiotics will subdue the infection. Another pacemaker is fitted later.

- Erosion; the pacemaker may erode through the skin. This is usually due to low-grade infection. Mechanical factors may also be responsible.

- Pocket haematoma; bleeding into the pacemaker pocket may result in significant pain, swelling, and on occasions wound breakdown and extrusion of the pulse generator. To avoid this complication warfarin is routinely stopped prior to pacemaker implantation and surgery is deferred in patients with an INR > 1.5.

- Lead displacement; in most cases the pacing lead is securely wedged into the trabeculae of the right ventricle. It takes approximately 6 weeks for the pacing lead to become firmly adherent to the myocardium. Most lead displacements occur early, typically within the first 24 hours. Lead displacement rarely occurs after 6 weeks but when it does it may lead to sudden loss of pacing and a recurrence of pre-pacing symptoms.

- Electromagnetic interference; this is not common with modern pacemakers. High-tension cables, high-energy radars, arc-welding
equipment and some medical equipment, such as MRI machines and lithotripters, may transiently inhibit the output of pacemaker or convert it to interference mode (continuous pacing despite an adequate underlying rhythm). Digital cellular telephones may cause similar problems, but only when the telephone is held in very close proximity to the pulse generator.

**Advantages And Disadvantages Of Pacing Modes** – In selecting the ideal pacing mode, the patient's overall physical condition, associated medical problems, exercise capacity and chronotropic response to exercise must be considered along with the underlying rhythm disturbance \(^{(61)}\). Some of the various ventricular and atrial pacing systems available and their NBG codes are described in the tables.
**Ventricular inhibited pacing** – VVI or ventricular demand pacing (ventricle paced, ventricle sensed, and pacemaker inhibited in response to sensed beat) remains the most commonly used pacing mode. Although VVI pacing will protect the patient from lethal bradycardias, it does not restore or maintain atrioventricular synchrony, nor does it provide rate responsiveness in the chronotropically incompetent patient (i.e., the patient who cannot achieve an appropriate heart rate for a given physiologic activity).

The earliest indication for cardiac pacing was high-grade or complete heart block with recurrent Stokes-Adams attacks. In patients with this disorder, establishing a stable ventricular rhythm was lifesaving and prevented catastrophic asystole. This fact alone overshadowed the observation that, although ventricular pacing improved cardiac output and/or patients' symptoms, it did not reestablish normal cardiac function. In addition, some patients with intermittent heart block experienced symptomatic hemodynamic deterioration with ventricular pacing \(^{(66, 67)}\).

**Pacemaker syndrome** – Adverse hemodynamics associated with a normally functioning pacing system, resulting in overt symptoms or limitation of the patient's ability to achieve optimal functional status, is referred to as pacemaker syndrome \(^{(66, 67)}\). The syndrome is characterized by the absence of AV synchrony, resulting in a decrease in stroke volume, and probable atrial contraction against a closed AV valve, similar to what is seen during complete heart block. Symptoms include presyncope or syncope, easy fatiguability, cough, dizziness, dyspnea, orthopnea, paroxysmal nocturnal dyspnea, and a sensation of
throat fullness. Physical examination may reveal hypotension, rales, increased jugular venous pressure with cannon A waves, peripheral edema, and murmurs of tricuspid and/or mitral regurgitation (66).

These clinical manifestations are associated with a constellation of findings on transesophageal echocardiography. These include higher reverse flow velocity in the pulmonary veins compared to patients with VVI pacing but no pacemaker syndrome, spontaneous echo contrast in the descending aorta, and significant mitral regurgitation (69).

Pacemaker syndrome was initially recognized in 5 to 15 percent of patients with ventricular (VVI) pacing. However, it can occur anytime there is AV dissociation (independent atrial and ventricular contraction), and it is now clear that it is a common occurrence. This was illustrated in a study in which 40 unselected patients with dual chamber pacemakers were entered into a blinded, randomized trial comparing the symptoms associated with VVI pacing to those associated with dual chamber pacing (67). Pacemaker syndrome was clinically recognized in 83 percent of patients paced in the VVI mode; 65 percent experienced moderate to severe symptoms.

The development of the pacemaker syndrome leads some patients to switch from VVI pacing. In one study in which 407 elderly patients with sick sinus syndrome or complete atrioventricular block were randomly assigned to either VVI or DDD pacing, 26 percent of patients assigned to VVI pacing crossed over to DDD pacing because of symptoms of pacemaker syndrome (69).
**Atrial inhibited pacing** – AAI pacing (atrium paced, atrium sensed, and pacemaker inhibited in response to sensed beat) is appropriate for patients with sinus node dysfunction who have intact AV nodal function. It is infrequently used in the United States. The obvious disadvantage of atrial pacing is lack of ventricular support should atrioventricular (AV) block occur. However, if the patient with sinus node dysfunction is assessed carefully for the presence of AV node disease at the time of pacemaker implant, the occurrence of clinically significant AV nodal disease is very low (less than 2 percent per year) (70).

Assessment prior to use of an AAI system should include incremental atrial pacing at the time of pacemaker implant. Although criteria vary among institutions and implanting physicians, the adult patient should be capable of 1:1 AV nodal conduction to rates of 120 to 140 beats/min.

**AV sequential pacing** – DVI (atrium and ventricle paced, ventricular sensing only, and inhibition of pacemaker in response to sensed ventricular beat) pacemakers were at one time implanted in large numbers of patients, often with a very satisfactory outcome. However, DVI pacing is limited by the absence of atrial sensing, which prevents the restoration of rate-responsiveness in the chronotropically competent patient. In addition, lack of atrial sensing may lead to competitive atrial pacing and initiation of atrial rhythm disturbances. For these reasons, DVI pacing is rarely the mode of choice at the time of implant but remains a programmable option in many dual-chamber pacemakers.
**DDI pacing** – In the DDI, dual chamber pacing mode, there is atrial sensing and pacing, and ventricular sensing and pacing; however, the pacemaker is incapable of tracking intrinsic atrial activity. As a result, there is only a single rate programmed and the paced ventricular rate will never exceed the programmed rate.

**Atrial synchronous ventricular inhibited (VDD) pacing** – VDD pacing (ventricle paced, atrium and ventricle sensed, and either inhibition or tracking of the pacemaker in response to a sensed beat) may be appropriate for the patient with normal sinus node function and conduction disease of the AV node. Dual chamber (two lead) VDD pacing systems have largely been supplanted by DDD pacemakers. However, VDD as the preimplant pacing mode of choice is gaining new acceptance with the introduction of single-lead VDD pacing systems (71,72). In this system, atrial sensing is accomplished from "floating" sensing electrodes on the atrial portion of the ventricular pacing lead.

**Dual chamber pacing** – The dual chamber (DDD) pacing system combines the functions of ventricular, atrial, AV sequential, and atrial synchronous pacing, providing physiologic pacing. Pacing and sensing occur in both atrium and ventricle.

- The pacemaker will be totally inhibited if sinus rhythm exists
- The atrium is stimulated if sinus bradycardia exists
- Both the atrium and ventricle are stimulated if bradycardia exists independently in both chambers
- The ventricle will be paced in synchrony with the atrium if heart block exists with normal sinus function
Thus, there are four different rhythms that can be seen with normal pacemaker function:

- Normal sinus rhythm
- Atrial pacing
- AV sequential pacing
- P-synchronous pacing (ie, atrial sensing and ventricular pacing or P wave ventricular pacing)

The DDD pacing mode is most appropriate for patients with normal sinus node function and AV block. DDD pacing is also considered by some to be the mode of choice in carotid sinus hypersensitivity with symptomatic cardioinhibition.

**Physiologic versus VVI pacing** – Dual chamber pacemakers have the following potential hemodynamic advantages over fixed rate VVI pacing:

- Higher cardiac output and arterial pressure $^{(73-75)}$
- Preservation of coronary blood flow $^{(76)}$
- Fewer or absent symptoms of "pacemaker syndrome" $^{(66,67,69)}$
- Lower incidence of heart failure and less left atrial dilation $^{(77)}$
- Possible reductions in recurrent atrial fibrillation, mortality, and thromboembolic events $^{(78-80)}$; the last benefit may be due in part to better left atrial appendage function with higher emptying and filling flow velocity, as well as less platelet activation $^{(76-81)}$.

The factors responsible for the reduction in atrial fibrillation are uncertain, although maintenance of AV synchrony may be important. In one report that compared VVI with DDD pacing in 30 patients, the loss of AV synchrony with VVI pacing was associated with atrial
electrical remodeling, which may promote atrial fibrillation; the remodeling was reversible after the restoration of AV synchrony with DDD pacing\(^{(82)}\).

**Non-atrial mediated rate-adaptive pacemakers** – These systems use a sensor (motion, vibration, minute ventilation, oxygen saturation, temperature) in a standard pacing mode, not based upon the sinus node rate, which allows for a rate response to stress. Following detection of movement or exercise, there is an increase in pacing rate. Such rate responsive pacemakers are indicated for patients who require an increase in heart rate in response to activity but who are chronotropically incompetent, i.e., they are unable to increase the heart rate in response to activity.

**VVIR pacing** – VVIR pacing, like VVI pacing, is contraindicated if ventricular pacing results in retrograde (VA) conduction and/or a fall in blood pressure. VVIR pacing should not be used as an excuse to forego attempts at placing an atrial lead in a patient who is undergoing pacemaker implantation, has normal sinus node function, and would benefit from rate-adaptive pacing. If the sinus node is intact, P-synchronous pacing should still be considered the optimal rate-adaptive parameter and used whenever possible.

There have been many studies comparing DDD and VVIR pacing. As an example, a randomized crossover design was used to evaluated 18 patients with complete heart block: cardiopulmonary performance and quality of life measures were similar with the two modes, although patients had a greater sense of well-being with DDD pacing\(^{(83)}\).
VVIR pacing is indicated in patients with chronic atrial fibrillation with a slow ventricular response.

**AAIR pacing** – AAIR pacing can be considered in the patient with sinus node dysfunction and normal AV node function, since this mode will restore rate responsiveness and maintain AV synchrony. If AAIR (or AAI) pacing is contemplated, normal AV node conduction must first be determined. As noted above, this assessment should include incremental atrial pacing at the time of pacemaker implant. Although criteria vary among institutions and implanting physicians, the adult patient should be capable of 1:1 AV nodal conduction to rates of 120 to 140 beats/min.

**DDDR pacing** – The ideal patient for DDDR pacing is one with combined sinus nodal and AV nodal dysfunction in whom DDDR pacing would restore rate responsiveness and AV synchrony. In a patient with paroxysmal atrial rhythm disturbances, for example, programming the pacemaker to the DDIR mode will allow rate modulation and AV synchrony but not allow tracking of any rapid pathological atrial rhythms.

**Mode switching pacing** – Pacemakers are now available that can mode switch in response to atrial arrhythmias. Such pacemakers will automatically switch from an atrial tracking mode to a mode not capable of atrial tracking, e.g., DDD to VVI mode, in response to rapid atrial rates as occur with atrial flutter and atrial fibrillation. These pacemakers avoid the potential for rapid paced ventricular rates if such arrhythmias occur in patients who have pacemakers capable of
atrial tracking. The following potential mode-switch combination are available in current generators:

- DDD to DDIR
- DDD to VVIR
- DDD to VVI
- VDDR to VVIR
- VDD to VVIR
- VDD to VVI

One report evaluated 48 patients with a history of paroxysmal atrial tachyarrhythmias and complete heart block who had a mode switching pacemaker; the patients preferred this pacing mode over conventional DDDR or VVIR pacing \(^{(84)}\). Inappropriate mode switching and tracking of atrial tachyarrhythmias was very uncommon.

**Algorithms** – A number of algorithms are available for determining the appropriate pacing mode for patients with sinus node disease and AV node disease. The first, more complex algorithms consider most of the available pacing modes. The second algorithm is simpler and assumes that a pacemaker capable of rate-adaptation will be used (see figure).
Results of cardiac pacing:

Permanent pacing results in abolition of dizziness and syncope and marked prolongation in life expectancy in AV block. It is now emerging that dual chamber pacing gives improve life expectancy over ventricular pacing when heart failure is the presenting feature and atrial or dual chamber pacing also offers benefits in life expectancy in sick sinus syndrome.

With modern pacing systems re-operation is rarely necessary until battery exhaustion is imminent\(^{(60)}\).

The history of cardiac pacing in Sudan:

The first temporary pacing in Sudan started in 1989 in Elshaab teaching hospital where it was performed by Mr. M. Saeed Elfeil.

The second temporary pacing was performed by Dr. Ahmed Babieker in June 1996 in Elshaab teaching hospital.

In August 1996, and as a revolution, the first permanent pacemaker implantation was performed in Ibn Sina hospital by Dr. Ahmed Babieker and Mr. Ibn Aouf. Two VVI pacemakers were inserted to two patients with CHB. The operation passed peacefully and successfully. The first patient died of a different disease and the second one underwent pulse generation replacement ten years later, he is still alive and well.
OBJECTIVES

1. To study the clinical characteristics and presentations of Sudanese patients with complete heart block.

2. To study the different modes of cardiac pacing used in Sudan.

3. To study the short-term outcome of cardiac pacing.
PATIENTS AND METHODS

This is a descriptive, analytical, partially retrospective and partially prospective study.

Study Area:

Sudan Heart Center. It is the third cardiac center in Sudan. It has been constricted in 2001. It is an advanced center where most of the cardiac investigations and operations are performed.

Study Population:

Patients with complete heart block who presented to or referred to SHC in the period from January to December 2004.

Inclusion Criteria:

Patients who underwent cardiac pacing in SHC in the appropriate period.

Exclusion Criteria:

Patients with CHB but refused or could not afford the cost of cardiac pacing.

Data Collection:

- Simple consecutive sampling.
- Data was collected by self-administered questionnaire that was constructed in sections to address the different aspects of the study including:

  a. Personal data
  b. Presenting symptoms
  c. Physical signs
  d. Important investigations
  e. Modes of cardiac pacing
  f. Short term outcome of cardiac pacing

**Data Analysis:**

The collected data was entered in a master sheet and analyzed in a statistical manner using SPSS (Statistical package for Social Sciences), the results were expressed in frequencies and percentages.
RESULTS

In the period from January to December 2004, a total of 70 patients with CHB who underwent cardiac pacing in SHC were studied. Those patients were followed up for 12 weeks to assess the short term outcome of the cardiac pacing on clinical basis.

The results of the study were as follows:

**Sex Distribution:** 55.6% of the patients were males and 44.3% were females with a male to female ratio 1.3:1. (Figure-1)

**Age Distribution:** The ages of the patients on presentation ranged from 35 to 85 years, with a mean age 64.6 years and the mode was 70 years. (Table-1)

**Associated Diseases:** 22.9% were hypertensive, 7.1% were diabetic, 15.7% had IHD, 21.4% had HTN, DM, IHD diseases and 32.9% had no associated diseases. (Table-2)

**Presenting symptoms:** The presenting symptoms were dyspnoea (52.3%), syncope (45.7%), dizziness (40%), chest pain (21.4%), cough (24.3%), palpitations (21.4%), L.L oedema (24.9%), fatigability (15.7%), convulsions (4.3%), confusion (4.3%), and vertigo (1.4%). (Figure-2)
Drugs: 12.9% of the patients were in Calcium Channel Blockers, 5.7% in Beta Blockers and 48.6% were using other drugs (Diuretics, Vasodilators, ACE inhibitors, Oral Hypoglycaemic Agents and Insulin). No patient was using Digitalis therapy. (Table-3)

Presenting signs: The main signs on examining the patients were bradycardia (88.4%) (Table-4), tachypnoea (31.4), and signs of heart failure (25.7%). (Figure-3)

The JVP was raised in 34.3%, (25.7% in heart failure and 8.6% had giant cannon waves but not in failure).

Blood pressure levels: On measuring the BP of those patients, 11.4% were hypotensive, 40.7% were normotensive, 45.7% had systolic hypertension and only 2.9% had systolic and diastolic hypertension. (Table-5)

ECG findings: ECG showed CHB alone in 74.3%, CHB and RBBB in 7.1%, CHB and ST segment and T wave changes in 15.7% and CHB with LBBB in 2.9%. (Figure-4)

ECHO findings: ECHO studies were normal in 31.4% of the patients while 25.7% had LVH with good systolic function, 15.7% had left ventricular dilatation with poor systolic function, 14.3% had ischaemic changes, 7.1% had ischaemic changes and dilated
cardiomyopathy, 2.9% had aortic valve sclerosis and 1.4% had mitral valve sclerosis. (Table-6)

Renal Function: 92.9% of the patients had normal renal function and 7.1% had renal impairment. Only 1.4% had hyperkalaemia. (Table-7)

Different modes of permanent pacemakers were implanted; VDD in 54/3%, VVI in 25.7%, DDD in 12.9%, DDDR in 4.3% and VVIR in 2.9%. (Figure-5)

**Complications of Pacemaker Implantation**: (85.7%) of the patients developed no complications peri or postoperatively, while only (14.3%) develop complications including bleeding (2.9%), infection (2.9%), lead displacement (4.3%), erosion through skin (2.9%) and pneumothorax (1.4%). (Table-8)

**The short-term outcome**: 91.4% of the patients improved after pacing mainly those with VDD pacing, (94.7% out of 83 patients improved) and in those with VVI pacing, (83.3% out of 80 patients improved). No significant difference regarding the outcome comparing VDD pacing versus VVI pacing. (Figure-6)

The improvement was shown by resolution of symptoms and better life performance.
Table (1): Age group distribution in the study population:

<table>
<thead>
<tr>
<th>Age group in years</th>
<th>Frequency</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>35 – 45</td>
<td>5</td>
<td>7.1</td>
</tr>
<tr>
<td>64 – 55</td>
<td>12</td>
<td>17.1</td>
</tr>
<tr>
<td>56- 65</td>
<td>17</td>
<td>24.3</td>
</tr>
<tr>
<td>66-75</td>
<td>27</td>
<td>38.6</td>
</tr>
<tr>
<td>76 -85</td>
<td>9</td>
<td>12.9</td>
</tr>
<tr>
<td>Total</td>
<td>70</td>
<td>100.0</td>
</tr>
</tbody>
</table>
Table (2): Incidence of associated diseases (DM, HTN, IHD) in study population:

<table>
<thead>
<tr>
<th>Diseases (s)</th>
<th>No. of patients</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>DM</td>
<td>5</td>
<td>7.1</td>
</tr>
<tr>
<td>HTN</td>
<td>16</td>
<td>22.9</td>
</tr>
<tr>
<td>IHD</td>
<td>11</td>
<td>15.7</td>
</tr>
<tr>
<td>DM + HTN</td>
<td>3</td>
<td>4.3</td>
</tr>
<tr>
<td>DM + IHD</td>
<td>2</td>
<td>2.9</td>
</tr>
<tr>
<td>HTN + IHD</td>
<td>5</td>
<td>7.1</td>
</tr>
<tr>
<td>DM + HTN + IHD</td>
<td>5</td>
<td>7.1</td>
</tr>
<tr>
<td>NO</td>
<td>23</td>
<td>32.9</td>
</tr>
<tr>
<td>Total</td>
<td>70</td>
<td>100.0</td>
</tr>
</tbody>
</table>
Table (3): Drugs used by the study population:

<table>
<thead>
<tr>
<th>Drug</th>
<th>Frequency</th>
<th>Percentage</th>
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</thead>
<tbody>
<tr>
<td>Beta blockers</td>
<td>4</td>
<td>5.7</td>
</tr>
<tr>
<td>Calcium channel blockers</td>
<td>9</td>
<td>12.9</td>
</tr>
<tr>
<td>Digoxin</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Other drugs</td>
<td>34</td>
<td>48.6</td>
</tr>
<tr>
<td>No drugs</td>
<td>23</td>
<td>32.9</td>
</tr>
<tr>
<td>Total</td>
<td>70</td>
<td>100.0</td>
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</table>
Table (4): Pulse rate in the study population:

<table>
<thead>
<tr>
<th>Pulse rate (bpm)</th>
<th>Frequency</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>25 – 35</td>
<td>12</td>
<td>17.2</td>
</tr>
<tr>
<td>36 – 45</td>
<td>37</td>
<td>52.8</td>
</tr>
<tr>
<td>46 – 55</td>
<td>13</td>
<td>18.5</td>
</tr>
<tr>
<td>56 – 65</td>
<td>8</td>
<td>11.6</td>
</tr>
<tr>
<td>Total</td>
<td>70</td>
<td>100</td>
</tr>
</tbody>
</table>
Table (5): Distribution of BP in the study population:

<table>
<thead>
<tr>
<th>BP</th>
<th>Frequency</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>8</td>
<td>11.4</td>
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<tr>
<td>Normal</td>
<td>28</td>
<td>40.0</td>
</tr>
<tr>
<td>Systolic hypertension</td>
<td>32</td>
<td>45.7</td>
</tr>
<tr>
<td>Hypertension</td>
<td>2</td>
<td>2.9</td>
</tr>
<tr>
<td>Total</td>
<td>70</td>
<td>100</td>
</tr>
</tbody>
</table>

N.B.:

Low < 100/60

High > 140/90
Table (6): ECHO findings in the study population:

<table>
<thead>
<tr>
<th>ECHO finding</th>
<th>No. of patients</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>22</td>
<td>31.4</td>
</tr>
<tr>
<td>LVH with good systolic function</td>
<td>18</td>
<td>25.7</td>
</tr>
<tr>
<td>Left ventricular dilatation with poor systolic function</td>
<td>11</td>
<td>15.7</td>
</tr>
<tr>
<td>IHD</td>
<td>10</td>
<td>14.3</td>
</tr>
<tr>
<td>IHD + DCM</td>
<td>5</td>
<td>7.1</td>
</tr>
<tr>
<td>Aortic valve sclerosis</td>
<td>2</td>
<td>2.9</td>
</tr>
<tr>
<td>Mitral valve sclerosis</td>
<td>1</td>
<td>1.4</td>
</tr>
<tr>
<td>AVR + healed vegetation</td>
<td>1</td>
<td>1.4</td>
</tr>
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</table>
Table (7): S. creatinine levels in the study population:

<table>
<thead>
<tr>
<th>S. creatinine levels</th>
<th>Frequency</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>65</td>
<td>92.9</td>
</tr>
<tr>
<td>High</td>
<td>5</td>
<td>7.1</td>
</tr>
<tr>
<td>Total</td>
<td>70</td>
<td>100.0</td>
</tr>
</tbody>
</table>

N.B.: Normal < 1.5mg/dl
Table (8): Complications of pacemaker insertion:

<table>
<thead>
<tr>
<th>Complication</th>
<th>No. of patients</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
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<tr>
<td>Infection</td>
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<td>Infection with erosion through skin</td>
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<td>Pneumothorax</td>
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Figure (1) : Sex distribution in the study population
Figure (2): Presenting Symptoms

- Dizziness: 22.9%
- Syncope: 45.7%
- Dyspnoea: 52.7%
- Chest pain: 15.7%
- Cough: 21.4%
- Lower limb swelling: 1.4%
- Palpitations: 21.4%
- Fatiguibility: 4.3%
- Convulsions: 4.3%
- Confusion: 1.4%
- Vertigo: 1.4%
- Asymptomatic: 0%
Figure (3): Presenting Signs

Bar graph showing the percentage of patients with various symptoms:
- Bradycardia: 88.4%
- Tachypnoea: 31.4%
- Raised JVP: 34.3%
- Murmurs: 8.6%
- Basal crackles: 25.7%
- Hepatomegaly: 20%
- L.L. Oedema: 25.7%
Figure (4): ECG finding in the study population
Figure (5): Modes of cardiac pacing in the study population
Figure (6): Short term outcome of cardiac pacing improved patients
DISCUSSION

In this study 70 patients underwent cardiac pacing, 65 primary pacemaker implantation and 5 pulse generator replacement. The site of the conduction delay and the underlying etiology were not satisfactorily determined. This is usual from a practical point, most centers do not further investigate patients with electrophysiological study (EPS). This is compared to the most recent data concerning the incidence of cardiac pacing for the United States 1993 world survey on cardiac pacing (93). In 1993, there were 426 pacemakers per million population with an estimated 112,064 primary pacemaker implantation and 20,965 pulse generator replacements. Sinus node dysfunction was the most frequent indication for pacing followed by AV node dysfunction. Pacing for tachyarrhythmias and miscellaneous indications accounted for only a small percentage.

In our study, males are more affected than the females. Most of the patients were elderly with the average age being 64.6 years and the commonest age on presentation was 70 years; but still 3 patients were young (<40 yrs) and 19 patients were in the middle age group (40-60 yrs). The patients presented with different clinical manifestations
mainly dizziness, syncope and dyspnoea, 2 patients presented with convulsions and one patient was asymptomatic and discovered during routine examination. On examining the patients, 18 patients were in heart failure, 24 patients had raised JVP, and 32 patients had systolic hypertension. Unfortunately no reference studies were found with regards to the different clinical manifestations of CHB in other populations.

In our study, different modes of cardiac pacing were used including VDD in 54.3%, DDD in 12.9%, VVI in 25.7%, DDDR in 4.3% and VVIR in 2.9%. Pacemaker implantation is generally safe and only 10 patients developed complications including; bleeding with pocket haematoma (2.9%), infection (2.9%), lead displacement (4.3%), erosion through skin (2.9%) and pneumothorax (1.4%). The patients were followed up for 12 weeks and the short term outcome was good. 91.4% of the patients improved and only 8.6% deteriorated, mostly due to lead displacement and pacemaker infection. Comparing DDD to VVI pacing; 88.9% of the patients with DDD pacing improved compared to 83.3% with VVI pacing. Moreover, 11.1% of the patients with DDD pacing deteriorated compared to 16.7% with VVI pacing. The outcome of cardiac pacing in the patients who presented in heart failure was good;
17 patients improved and only one patient, with VVI pacemaker, had his symptoms deteriorated and 6 weeks later he was crossed over to DDD pacemaker with evident improvement.

This study is compared to many randomized studies that have produced somewhat conflicting results concerning the long-term benefits of DDD compared to VDD or VVI pacing:

- Wiegand and his friends, in 1999 studied the implications for single lead VDD pacing. 441 patients with complete AV block treated with either VDD or DDD pacemakers They found that the cumulative incidence of sinus node disease was 0.65% per year and only 0.2% of the patients were symptomatic. The cumulative incidence of atrial fibrillation was 2% per year regardless of the mode of pacing (86).

- Lamas and his friends, the PASE trial, in 1998 randomly assigned 407 elderly patients (average age 76) with sick sinus syndrome or CHB to rate responsive VVI or DDD pacing. At 30 months, no difference was observed in total mortality, atrial fibrillation, or the combined end point of stroke, hospitalization for heart failure, or death. However, in the subset of patients with a sick sinus syndrome, DDD pacing was associated with reductions of borderline statistical significance in mortality (12 versus 20 percent), atrial
fibrillation (19 versus 28 percent), and the combined end point of stroke, hospitalization for heart failure, or death (20 versus 31 percent), and the modest increases in quality of life and functional status. During follow-up, 26 percent of the patients crossed over from VVI to DDD pacing because of symptoms related to pacemaker syndrome. \(^{(69,79)}\)

- Connolly and his friends, the much larger Canadian Trial of Physiologic Pacing (CTOPP), in 2000 randomly assigned 2568 patients to VVI or physiologic (AAI or DDD) also found no difference between the groups in the primary end point or cardiovascular death or stroke (5.5 versus 4.9 events per year, the annual rate of all-cause mortality (6.6 versus 6.3 percent), and functional capacity as assessed with a six minute walk. The only benefit was not apparent until two years after the implantation. There were significantly more perioperative complications with physiologic pacing (9 versus 3.8 percent). \(^{(88)}\)

- Jahangir and his friends, in 1999 studied the value of ventricular or dual chamber pacing in the very elderly retrospectively, in a report of 432 patients (average age 85). After a 3.5 year follow-up, there was no difference in survival between patients with a sick sinus
syndrome receiving either type of pacemaker and that expected in an age-matched population; in comparison, survival after pacemaker insertion in patients with high grade AV block was significantly lower compared to expected survival of age and gender-matched population. Ventricular pacing was associated with a poorer survival compared with dual chamber pacing in this group.\(^{(89)}\)

- Deharo and his friends, in 1996 evaluated 18 patients with CHB in a randomized crossover design comparing DDD and VVIR pacing: cardiopulmonary performance and quality of life were similar with the two modes, although patients had a greater sense of well-being with DDD pacing. \(^{(83)}\)
CONCLUSION

- CHB is mainly a disease that increases with advancing age in Sudan but occasionally occurs in young people.
- Cardiac pacing is relatively simple procedure with few complications.
- Cardiac pacing improved the clinical status of patients with CHB especially those in heart failure improved significantly after pacing.
- VVI pacing is still used in Sudan only due to financial restraints with good outcome.
RECOMMENDATIONS

- Elderly people who presented with convulsions, recurrent falls or their complications (fractures, SDH) should be investigated for AV block.
- Early detection and treatment of the causes of AV block are needed to decrease the incidence of CHB and the need for cardiac pacing.
- Cardiac pacing is a life saving procedure and should be available in every cardiac center.
- More research is needed in the field in Sudan.
REFERENCES


64. Campbell RW. Chronic Mobitz Type I Second Degree AV Block. Has its importance been underestimated? Br Heart J 1985; 53: 585.


77. Nielsen JC, Andersen HR, Thomsen PE. Heart failure and echocardiographic changes during long-term follow-up of patients with sick sinus syndrome randomized to single-chamber atrial or ventricular pacing. Circulation 1998; 97: 987.


81. Simantirakis EN, Parthenakis FI, Chrysostomakis SI. Left atrial appendage function during DDD and WI pacing. Heart 1997; 77: 428.

82. Sparks PB, Mond HG, Vohra JK. Electrical remodeling of the atria following loss of atrioventricular synchrony: A long-term study in humans [hi Process Citation]. Circulation 1999; 100: 1894.


87. Ellenbogen KA, Stambler BS, Orav EJ. Clinical characteristics of patients intolerant to WIR pacing. Am J Cardiol 2000; 86: 59.

**Questionnaire**

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If Yes specify .................................................................

**Drugs:**

1) Diyoxin

2) Beta bloker

3) Calcium channel blocker

4) Others, specify .................................................................

**Physical examination:**

1) Confused Yes | No |

2) Tachyapnocc Yes | No |

3) Pulse rate |

4) BP |

5) Raised JVP Yes | No |

6) Murmur or added sounds Yes | No |

7) Basal crepitations Yes | No |
8) Hepatomegaly Yes ☐ No ☐
9) L.L. Oedema Yes ☐ No ☐

Investigations:
1) ECG: .................................................................

.................................................................
2) ECHO: .............................................................

.............................................................
3) S.K^+
4) B. Urea
5) S. Creatinine

Mode of cardiac pacing:
1) VVI
2) DDD
3) Others, specify..............................................

Complications
1) Bleeding Yes ☐ No ☐
2) Failure of route Yes ☐ No ☐
If Yes Specify:

Outcome in three months:
1) Improved
2) Static
3) Deteriorated
4) Died