HIGH DEGREE ATRIOVENTRICULAR BLOCK IN ACUTE INFERIOR MYOCARDIAL INFARCTION

CORONARY CARE UNIT

GROOTE SCHUUR HOSPITAL

1994 – 1997

ES FREDERICKS

FOR THE DEGREE: MMED(MED)
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DECLARATION

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DATE: August 2001
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ABSTRACT

Acute inferior myocardial infarction is generally associated with a benign course and low in-hospital mortality. However, a group of patients with high degree atrioventricular block has significantly increased mortality. Although some of the prognostic factors associated with increased mortality have been identified, the explanations for these remain less than satisfactory.

To determine the experience at Groote Schuur Hospital, the data of all patients admitted with acute inferior myocardial infarction to the Coronary Care Unit from 1994 to 1997 were retrospectively analysed, especially with regard to high degree atrioventricular block and mortality. A total of 292 (two hundred and ninety two) were admitted with acute inferior myocardial infarction. High degree block was present in 55 of 292 patients (18.8%). Significantly more patients in the high degree block group had a low mean blood pressure on admission (p<0.0001), low pulse rate on admission (p=0.0001), cardiogenic shock (p<0.0001) and RV involvement (p=0.01). They also received more specific supportive interventions like inotropic support (p<0.0001), pacemaker placement (p<0.0001) and intra-aortic balloon pump insertion (p=0.05).
There were a total of 21 (twenty one) deaths (7.2%). The in-hospital mortality of those with high degree atrioventricular block was 16.4 % (9 of 55 patients). Cardiogenic shock was significantly more common (43.6%) in those with high grade AVB. However, despite temporary pacing, 37.5% of patients with high grade AVB died in cardiogenic shock. The patients that died were generally older (p<0.0001), had previous myocardial infarctions (p=0.007) and all had cardiogenic shock (p<0.0001). Multivariate analysis showed cardiogenic shock and previous myocardial infarction to be independent predictors of mortality.

Conclusion: High degree atrioventricular block is not independently associated with increased in-hospital mortality. The mortality was associated with additional factors as shown by multivariate analysis. These patients were sicker and required more interventions like pacemaker placement, inotropic support and intra-aortic balloon pump insertion.
LIST OF ABBREVIATIONS

AMI: Acute myocardial infarction
AV: Atrioventricular
AVB: Atrioventricular block
CABG: Coronary artery bypass graft
CAD: Coronary artery disease
CCF: Congestive cardiac failure
CCU: Coronary Care Unit
CHB: Complete heart block
CK: Creatinine kinase
DM: Diabetes Mellitus
ECG: Electrocardiogram
GISSI: Gruppo Italiano per lo Studio...ncll Infarto miocardico
GSH: Groote Schuur Hospital
IABP: Intra-aortic balloon pump
IHD: Ischaemic heart disease
ISSIS-2 Second International Study of Infarct Survival
IV: Intravenous
LAD: Left anterior descending coronary artery
LV: Left ventricle
LVEF: Left ventricular ejection fraction
MI: Myocardial infarction
NS: Not significant
PDA: Posterior descending coronary artery
PPM: Permanent pacemaker placement
PTCA: Percutaneous transluminal coronary angioplasty
RCA: Right coronary artery
RV: Right ventricle
RVI: Right ventricular infarct
SPRINT: Secondary Prevention Reinfarction Israeli Nifedipine Trial
TAMI: Thrombolysis and Angioplasty in Myocardial Infarction Study
TIMI: Thrombolysis in Myocardial Infarction Studies
TPA: Tissue plasminogen activator
TPM: Temporary pacemaker placement

Arrhythmia:

VT: Ventricular tachycardia
VF: Ventricular fibrillation
AF: Atrial fibrillation
AT: Atrial tachycardia
Flutt: Atrial flutter
INTRODUCTION (AND LITERATURE REVIEW)

Inferior myocardial infarction accounts for 40-50% of all acute myocardial infarctions (AMI). The prognosis for inferior wall myocardial infarction (IMI) is generally regarded as favourable compared to anterior wall myocardial infarction. In-hospital mortality rates for IMI are reported as 2-9% in the post-thrombolytic era. However, there is a subset of patients with IMI that have an increased mortality. Although some of the prognostic factors associated with increased mortality have been identified, the explanations for these remain less than satisfactory.

This review will focus on these high-risk subgroups in IMI. The effect of early reperfusion/revascularization in these subgroups will also be discussed.

HEART BLOCK

Patients with IMI and atrio-ventricular block (AVB) have a higher mortality rate than those without block. High degree AVB is defined as the presence of 2:1 or higher AVB or complete heart block. The in-hospital mortality of patients with high degree AVB ranges from 13-44%
and averages at 23%, while those without AVB have a mortality range between 4-16% and an average of 9%. Various studies in the thrombolytic era have found similar mortality rates. 6,7

The incidence of high degree AVB in IMI is estimated at 19% (13-34%) in the prethrombolytic era. 5 An incidence of 12-13% is reported in the thrombolytic trials. 8,9 This incidence increases sharply to 48% in the presence of right ventricular infarction (RVI). 10 The reported incidence of high degree AVB in RVI varies between 40-50%. 11,12

Timing of AVB:

Two distinct groups are recognised:

(a) Early group: These patients will develop AVB on admission or within the first 24 hours after admission. Up to two thirds of patients with high degree AVB and IMI will fall in this category. 5 Furthermore, approximately 65% of all patients in this category will have high degree AVB on admission, rather than developing it through the first 24 hours. 8

Patients in this category have following characteristics:

- AVB is of short duration
- Usually complete heart block without previous documentation of first and second degree AVB
- Poor response to Atropine (35%)
- Increased requirement of pacemaker placement 65%
- Increased morbidity and mortality (23%) with higher incidence of syncope, left heart failure and cardiogenic shock.\textsuperscript{13}

(b) Late group: These patients will develop AVB after 24 hours of admission. All of those that will develop AVB will do so within three days of admission.\textsuperscript{5}

They have the following characteristics:

- prolonged AVB
- usually of second degree with a documented previous first degree block
- good response to Atropine (77%)
- need for pacemaker placement low (42%)
- morbidity and mortality much lower.\textsuperscript{13}
- mortality of 7% which is equal to mortality in patients without AVB

Some investigators however, did not find a significant difference in mortality in early and late AVB in a subgroup of patients with and without RVI.\textsuperscript{14}
Pathophysiology of AVB:

The pathophysiology of advanced AVB during IMI is not completely understood.\textsuperscript{13} Two theories have been proposed to explain to some extent the aetiology of heart block in IMI:

(1) Interruption of the blood flow to the AV node: acute IMI usually results from occlusion of the right or left circumflex coronary arteries. The right coronary artery is the source of the AV nodal artery in 90\% of the population.\textsuperscript{15} Therefore it was originally believed that heart block was caused by necrosis of the AV node. However, it has been shown that even complete occlusion of the AV nodal artery rarely causes necrosis of the AV node.\textsuperscript{16} There are three explanations for this:

$\checkmark$ First, the conducting system is more resistant to hypoxia, because the rate of it's oxygen consumption is only one fifth of that of the contractile tissue.\textsuperscript{17}

$\checkmark$ Second, the conducting tissue has high glycogen concentration allowing less dependence on oxidative phosphorylation.\textsuperscript{17}

$\checkmark$ Finally, timeous spontaneous recanalization or sufficient collateral blood flow to the AV node may prevent necrosis. However, De Wood et al demonstrated that spontaneous recanalization only occur in about one third of patients studied angiographically in the first 24 hours after AMI.\textsuperscript{18}
On the subject of collateral blood supply to the AV node, post mortem studies on humans showed that the AV nodal artery supplied the proximal two-thirds of the AV node, but not the distal third or the His bundle. A further observation was that in 50% of cadaver hearts there were connections between the AV node and left anterior descending (LAD) coronary artery and collateral vessels. Kennel and Titus demonstrated that 80% of human hearts have anastomotic channels between the AV nodal artery and the first septal perforator branch of the LAD coronary artery. These findings have been corroborated by other investigators.

Despite the dual blood supply of the AV node, ischaemia without necrosis may still play a crucial role in the pathogenesis of AVB in IMI. The AV nodal artery is a relatively distal branch of the dominant artery, arising some distance from the ostium. Because most infarctions are the result of occlusion of the artery proximal to the origin of the AV nodal artery, nodal ischaemia would be expected in the majority of patients with IMI. The fact that only about 20% of infarctions result in high degree AVB suggests that factors other than ischaemia may also play a role.

The role of concomitant LAD coronary artery stenosis in IMI with AVB has been systematically investigated. This would predispose the AV node to greater degrees of ischaemia. Bassen et al in a study of 51
patients with IMI, 11 of whom developed heart block, found that the development of heart block had a predictive value of 91% for concomitant LAD coronary artery disease.\textsuperscript{22} The LAD coronary artery in this instance was 75% stenosed. However, two subsequent studies found opposite results. In both the Thrombolysis in Acute Myocardial infarction (TAMI) and Thrombolysis In Myocardial Infarction (TIMI) studies, no greater incidence of LAD stenosis was found in the groups with IMI and AVB.\textsuperscript{8,9} Heart block during IMI was not predictive of multivessel disease as was originally suggested by Bassen et al.

(2) High vagal tone resulting from the Bezold-Jarisch reaction: this hypothesis invokes increased vagal tone resulting from the stimulation of afferent nerves adjacent to the AV node by ischaemia.\textsuperscript{23} The resultant outpouring of parasympathetic stimulation via the vagus nerve produces sinus bradycardia, hypotension, and in some patients, heart block. This proposed mechanism fails to explain the increased infarct size seen in patients with heart block. Furthermore, it does not explain heart block that occurs in the absence of sinus slowing.

Several studies have however shown that reperfusion of the right coronary artery with thrombolytic agents is also a strong stimulus of the Bezold-Jarisch reflex.\textsuperscript{24} If this is true, one would expect to find an increased incidence of AVB in patients receiving thrombolytic
therapy. However, it is well established that the incidence rates of AVB are similar in the pre- and post thrombolytic era’s. Moreover, the TIMI investigators found that heart block was associated with occlusion rather than patency of the infarct-related artery on cardiac catheterization 18-48 hours post thrombolytic therapy. Reperfusion is therefore not associated with the development of AVB during IMI through either a direct mechanism or the stimulation of the Bezold-Jarisch reflex.

An additional proposed mechanism of heart block in patients with IMI involves the role of intracellular electrolytes and metabolites, particularly potassium and adenosine, that are released from ischaemic myocardial cells. A high level of potassium is well known to cause heart block. The potassium release is greatest 24 hours after the AMI and the necrotic area gradually loses potassium until the forth day.

Adenosine has been shown in animal models to produce AVB, and has been used in several human studies for this effect in the treatment of AV nodal re-entrant arrhythmia. The depressant effect of adenosine on AV nodal tissue appears to be through interaction with a specific extracellular receptor that is blocked by methylxanthines such as aminophylline, but not atropine. Aminophylline, which blocks adenosine receptors in the heart, has been shown to reverse AVB not
only in animals, but also in humans with late AVB which was resistant to atropine.\textsuperscript{28} If chemical mediators play an important role in the development of heart block, this mechanism might serve to explain the association with larger infarct size, as larger infarcts would release proportionately more of these mediators.

**Second degree- or complete AVB: Does it matter?**

Second degree AVB is divided into:

- Mobitz I block also known as Wenckebach phenomenon
- Mobitz II block and 2:1 or higher AVB.

High degree AVB is defined by the presence of 2:1 or higher AVB or complete heart block.\textsuperscript{23} As mentioned before high degree AVB is associated with a higher in-hospital mortality compared to patients with no AVB.\textsuperscript{9,13} Most studies however, have looked at the incidence and prognostic significance of high degree AVB only in IMI. Only two investigators had looked at the prognostic significance of second degree AVB in acute IMI.\textsuperscript{29,30}

In both these studies the second degree AVB was of the Wenckebach type or advanced AVB, no Mobitz type II block was observed. In the Secondary Prevention Reinfarction Israeli Nifedipine Trial (SPRINT) the
incidence of Wenckebach type block was 68%, compared to 32% for advanced AVB. The clinical as well as short- and long-term outcomes in these groups did not show statistical difference apart from higher pacemaker placement in the advanced AVB group. However, a trend for higher mortality was observed in the advanced AVB group, relative risk 1.46. The overall conclusion was that Wenckebach type second degree AVB is not associated with an unfavourable hospital or post-discharge prognosis in patients with acute IMI, compared with advanced AVB.²⁹

Sclarovsky et al have done a similar study, but they additionally subdivided the patients into early and late AVB groups.¹³ In the early group, 58% of patients presented with complete AVB and 42% with second degree AVB, of whom 31% had Wenckebach phenomenon and 69% advanced AVB. No patient had previously documented first degree block. In the late group only 4% had initial complete AVB, while of the remaining 96%, 81% had Wenckebach phenomenon compared to only 19% with advanced AVB. First degree AVB was previously reported in 72% of this group. This data is in accordance with previous results.⁸

In all studies it was documented that the clinical outcome in patients with first degree AVB was identical to patients with no AV block.
Is it possible to predict subgroups of patients with acute IMI that will develop AVB? Very few investigators looked into this, but there is one landmark study by Birnbaum et al that looked into the association between clinical and electrocardiographic admission characteristics and development of heart block. The main findings in this study were: female gender, Killip class >2 on admission, J point/R wave ratio >0.5 and absence of abnormal Q waves (in inferior leads) on the admission ECG were all associated with an increased risk of high degree AVB.

**Prognostic significance of high degree AVB:**

The prognostic implications of high degree AVB in acute IMI are not entirely clear. High mortality rates in this setting have been reported. However, the exact mechanism of this unfavourable prognosis of AVB is not completely explained.

Gupta et al have found that patients with AVB have a higher incidence of congestive cardiac failure and higher CK levels; they concluded that these patients have a greater amount of myocardial necrosis (= larger infarct size) with a higher incidence of complications. Rotman et al reported that high degree AVB in this setting has a higher mortality only in the presence of heart failure. They could otherwise not demonstrate a higher in hospital mortality or morbidity.
Nicod et al showed that larger infarct size and an increased incidence of left ventricular failure is associated with increased in-hospital mortality rates in patients with high degree AVB in acute IMI. They further showed that high degree AVB is an independent predictor of increased mortality.

Mavric et al showed further that high degree AVB in acute IMI, regardless of right ventricular involvement, was associated with higher maximal serum CK values, higher incidence of left ventricular failure and a higher mortality. In addition, they showed that the mortality was much higher in the presence of right ventricular infarction and high degree AVB, compared to patients without high degree AVB. They concluded that right ventricular infarction does not confer higher mortality independently, but only in association with high degree AVB. Zehender et al however, showed that right ventricular infarction was an independent predictor of prognosis in acute IMI.

Temporary transvenous ventricular pacing is the preferred method of treatment for high degree AVB in acute IMI. The use of this procedure is quoted as being between 29 and 36% in this setting. In the majority of patients this is an effective way of managing AVB, however there is a subset of patients with high-degree AVB and cardiogenic shock with a ventricular escape >50 in whom pacing does not influence mortality.
The role of early reperfusion in patients with high degree AVB and acute IMI is still unsettled. The relative risk of mortality associated with AVB after thrombolytic therapy was 3.8 in the TIMI II trial, compared with 2.6 in the pre-thrombolytic era. The benefit of thrombolytic therapy in this particular group of patients is therefore questionable. A more recent study by Kimura et al showed that early reperfusion was associated with abolition of high degree AVB and maintenance of normal sinus rhythm throughout the study period. Reperfusion was defined as establishment of TIMI grade III flow, thus avoiding the need for pacing. Although the short-term prognosis in these patients is poor, many studies have shown that the long-term outcome in patients with high degree AVB and acute IMI was favourable.

RIGHT VENTRICULAR INFARCTION

Previously, RV infarction was not believed to be a clinically significant entity. It was only in 1973 that the clinical syndrome produced by acute ischaemic RV dysfunction had been described. RV myocardial infarction is predominantly a complication of acute IMI. The incidence of RV infarction in the setting of acute IMI is estimated between 40-50%. In a half of these patients it is associated with significant haemodynamic compromise, and high degree AVB is present in as
high as 60% of these cases.\textsuperscript{40} In contrast, RV infarction in association with anterior AMI occurs in less than 10%.\textsuperscript{41}

The clinical consequences of RV infarction range from no haemodynamic compromise to severe hypotension and cardiogenic shock.\textsuperscript{42,43} In the absence of haemodynamic compromise, no specific treatment is required for RV infarction. Characteristic clinical signs may be absent on physical examination despite noninvasive evidence of RV involvement.\textsuperscript{44} There are various ways to make the diagnosis of RV infarction ante-mortem. The commonest ones are: echocardiography, first pass equilibrium radionuclide ventriculography or right precordial electrocardiography. The diagnostic accuracy of the electrocardiographic V\textsubscript{4R} lead is superior in all respects and has a sensitivity and specificity of >90%, with a positive predictive value of >80%.\textsuperscript{45} It's use is limited however, in co-existing cardiac disease with ST-segment elevation.\textsuperscript{46}

Initial treatment for haemodynamically significant RV infarction requires prompt fluid therapy to abort the vicious cycle set in motion by this event.\textsuperscript{47} In patients who do not respond to fluid challenge, dobutamine has been proven to be effective.\textsuperscript{48}

The short-term consequences of RV infarction are not limited only to the development of cardiogenic shock and right heart failure. Many
investigators have now confirmed that patients with IMI and RV infarction have a high incidence (up to 60%) of high degree AVB, compared to patients without RV infarction (13%). Others have found that ST-segment elevation in V4R during acute IMI is predictive of subsequent development of heart block in 48-75% of patients. Therefore, RV infarction is regarded as an independent risk-factor for inhospital mortality in patients with acute IMI. The long-term prognosis of RV infarction is not entirely clear.

What is the impact of thrombolytic therapy in acute IMI and RV infarction? Data from the TIMI II trial support the view that successful thrombolysis reduces the incidence of RV involvement during acute IMI. Further studies are needed to evaluate the prognostic effect of revascularization in this setting.
STUDY OBJECTIVES:

In the light of the above it is clear that many questions remain unanswered regarding high-risk subgroups in acute inferior myocardial infarction. The purpose of this study therefore, is to clarify the following issues in this group of patients:

♦ The incidence of heart block and right ventricular infarction in all patients admitted with inferior myocardial infarction during the study period.

♦ The contribution of both the above on the mortality in these patients.

♦ The overall mortality rate in the study population.

♦ To identify any other risk-factors that may be associated with increased mortality in the study population.

♦ To assess the effects of thrombolysis and/or percutaneous transluminal coronary angioplasty on survival.

♦ To clarify the role of temporary transvenous ventricular pacing in patients with acute inferior myocardial infarction and high degree atrioventricular block.

And finally..

♦ To compare our local results against international data.
METHODOLOGY

This is a retrospective analysis of all patients admitted to the Coronary Care Unit (CCU) with acute IMI between the periods January 1994 to December 1997.

A computer generated list of all CCU admissions during the above time period was used to access the medical records of all patients admitted with acute IMI. This information was cross-checked with medical information stored in the CCU.

During their CCU admission patients were monitored constantly for the development of arrhythmias. Those who developed AVB had a 12-lead ECG performed immediately. Daily ECG’s were recorded in the CCU.

All patients received standard medical care for acute myocardial infarction. Any decision regarding intervention during this admission was made by the cardiologist in charge of CCU.
Diagnosis of IMI:

1. History suggestive of acute myocardial infarction i.e. characteristic, prolonged precordial chest pain
2. ST-segment elevation of at least 1mm in the ‘inferior’ chest leads – leads II, III and aVF.
3. Elevation of serum creatinine kinase (CK). A significant rise in CK was considered to be twice the upper limit of normal. In doubtful cases the cardiac specific iso-enzyme of CK (MB-fraction) was used to confirm myocardial injury, and thus infarction.

Two of the above were required for the diagnosis of acute IMI.

Atrioventricular block:

AVB was diagnosed using existing criteria. Patients were subdivided according to the following ECG criteria:

1. No AVB
2. First degree AVB – PR interval pronged 200ms or greater.
3. Mobitz I block – also known as Wenckebach phenomenon. Here there is progressive prolongation of the PR interval until the P wave fails to conduct.
4. 2:1 or higher atrioventricular block – when every second or more P wave conducts to the ventricles.
5. Complete AV block – when there is complete dissociation between atrial and ventricular contraction. Here the ventricular escape rhythm is important.

High grade AVB is generally defined by the presence of either 2:1 or higher block or complete AVB.\textsuperscript{23}

\textbf{Right ventricular involvement:}

The diagnosis of right ventricular infarction was made on the following grounds:

1. ST-segment elevation of equal to or greater than 1 mm in the right precordial chest leads, especially V\textsubscript{4R}, in patients with typical ECG changes of inferior myocardial infarction.

2. Clinical criteria of RV involvement as described previously, and.

3. Haemodynamic studies on cardiac catheterization.

\textbf{STUDY CHARACTERISTICS}

For the purposes of this study the following characteristics were looked for and analysed:

1. Demographic features (age and gender)
2. Risk factor profile especially for diabetes and previous myocardial infarction.

3. Admission characteristics:
   - Blood pressure: the mean blood pressure on admission was recorded. The mean blood pressure was calculated using the following formula:
     Mean BP = diastolic blood pressure + 1/3 pulse pressure
     (pulse pressure = (systolic – diastolic) blood pressure)
   - Pulse rate: the pulse rate on admission was recorded. If the pulse rate was omitted in the clinical notes, the pulse rate on the admission ECG was used.
   - Cardiogenic shock: as a measure of cardiovascular instability. Here it is defined as severe hypotension with a systolic BP < 80mmHg, associated with poor peripheral circulation.  

4. ECG findings especially with regard to:
   a. area of infarction: according to established criteria on leads involved on the ECG.
   - Inferior: Changes limited to leads II, III and aVF
   - More extensive infarction: Inferior changes as above plus involvement of the anterior or lateral leads.
   b. AVB
c. rhythm abnormalities: all arrhythmias were systemically recorded. The more lethal ventricular tachyarrhythmias were compared to supraventricular tachyarrhythmias. Common ventricular arrhythmias include:

- Ventricular tachycardia (VT): Defined as three (3) or more ventricular beats occurring at a rate of 120 beats per minute or more. The ECG shows a rapid ventricular rhythm with broad (often 0.14s or more), abnormal complexes. The importance of this arrhythmia lies in whether it is sustained (>30 sec) or not.

- Ventricular fibrillation (VF): This is very rapid and irregular ventricular activation with no mechanical effect. The ECG shows shapeless, rapid oscillations and there is no hint of organized complexes.

Important supraventricular arrhythmias include:

- Atrial fibrillation (AF): In this arrhythmia there is continuous, rapid activation of the atria with very little mechanical action, and only a portion of the impulses are conducted to the ventricle. Clinically the patient has a very irregular pulse. The ECG is typical in that there are no clear P waves.

- Atrial flutter (Flutter): The atrial rate is here usually around 300/min. The ECG shows regular saw-tooth like atrial flutter waves between QRST complexes.
Atrial tachycardia (AT): Here the atrial rate is around 150/min. The ECG shows abnormal P waves that occur in front of the QRS complexes.

5. Presence or absence of RV involvement

6. Specific treatment:

a. Trombolytic therapy: Commonly this means the intravenous administration of Streptokinase or tissue plasminogen activator (tPA). Contra-indications to thrombolytic agents were carefully documented, as well as other reasons why patients did not receive this form of therapy.

b. PTCA:

   ♦ Primary PTCA: Patients with contra-indications to thrombolytic therapy or haemodynamically compromised patients received this form of therapy.

   ♦ Rescue PTCA: This was used generally when thrombolytic therapy failed.

7. Rate of CABG

8. Placement of a pacemaker:

   TPM: Temporary pacing was achieved by transvenous insertion of a catheter electrode with the catheter situated in the right ventricular apex and attached to an external generator.
PPM: Permanent pacing leads were inserted transvenously through the subclavian or cephalic vein with the leads positioned in the right atrial appendage for atrial pacing and the right ventricular apex for ventricular pacing.

9. Any therapeutic interventions:
   a. Placement of intra-aortic balloon pump: to augment both the diastolic pressure and cardiac output.
   b. Administration of intravenous inotropic and other agents:
      Inotropic agents in this context mean intravenous infusions of Dobutamine, Dopamine or Adrenaline.
   c. Defibrillation.
   d. Mechanical ventilation

9. Mortality;
   The in-hospital mortality was recorded. The mechanism for death was recorded in all cases as far as possible.
STATISTICAL ANALYSIS

All the patient data was captured on a personal computer and analysed using Microsoft Excel spreadsheets. Data analysis was done using Statistica Kennel version 5.5 (USA). Continuous data are presented as mean ± standard deviation. Frequencies and outcomes were examined and compared in a fourfold tabular analysis. Categorical variables were analysed by Pearson Chi-square and Fisher exact test for small numbers. Continuous variables were compared using the Student's t-test.

When analysing risk factors, variables found to be significant by univariate analysis were entered into the model of stepwise logistical regression to identify independent predictors or subsets of variables that in combination were good predictors. Furthermore, multivariate analysis was performed using Visual GSR (General Stepwise Regression) in the Statistica program. This was done for the following endpoints:

1. Outcome
2. Mortality
3. Heartblock.

Statistical significance was regarded as a p value < 0.05.
ETHICAL CONSIDERATIONS:

This retrospective audit was approved by the institutional ethical committee.
RESULTS

A total of 292 patients with a diagnosis of acute inferior myocardial infarction were admitted to the CCU during the 4 year period described above. Table 1 gives a breakdown of the demographic characteristics of all the patients.

<table>
<thead>
<tr>
<th>DEMOGRAHICS</th>
<th>n=292</th>
<th>(%)</th>
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<tbody>
<tr>
<td><strong>General</strong></td>
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</tr>
<tr>
<td>Age</td>
<td>Mean 57.7 ± 11.2</td>
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<tr>
<td></td>
<td>Range: 22 - 89</td>
<td></td>
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<tr>
<td>Gender: male</td>
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<td>69.2</td>
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<tr>
<td>female</td>
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<tr>
<td>Diabetes Mellitus</td>
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<td>Previous MI</td>
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<td>13.7</td>
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<td><strong>Admission characteristics:</strong></td>
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<tr>
<td>Blood pressure</td>
<td>Mean 90.69 ± 23.0</td>
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<td></td>
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<td>Pulse</td>
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<td></td>
<td>Median: 72.0</td>
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<td>Cardiogenic shock</td>
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<td></td>
<td>55</td>
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*Table 1:* Characteristics of all patients admitted with acute inferior myocardial infarction.

The mean age of all patients was 57.7 with a range of 22-89. Consistent with international observation, significantly more of the admissions were
male (69.2%) compared to female (30.8%). Further, the mean age of the males was 55.7, while that of the females was 62.3. This observation was statistically significant with a $p$ value of 0.0002.

A third (27.1%) of all patients had Diabetes Mellitus. Female patients had a significantly higher incidence of diabetes compared to their male counterparts, 38.9% against 21.8%21. ($p=0.002$)

Previous myocardial infarction was documented in 13.7% of all patients. Significantly more males (16.3%) had prior myocardial infarction compared to the females (7.8%). This result was statistically significant with $p=0.05$.

**Admission characteristics:**

1. Blood pressure: The mean blood pressure in all the patients was 90.69.

2. Pulse rate: The mean pulse rate in all patients was 72.8.

3. Cardiogenic shock: Cardiogenic shock was present in 16.8% of all patients.

**ECG characteristics:**

(a) ECG Diagnosis: A more extensive pattern of infarction as characterised by ECG lead involvement was observed in only 27.7% of
all patients. The majority of patients (72.3%) had inferior myocardial infarction only.

(b) RV involvement: A total of 18.8% of all patients had ECG evidence of RV involvement.

c) Arrhythmia: From the total study population 22.3% had tachyarrhythmias (65 of 292 patients). The majority (78.5%) of patients had ventricular tachyarrhythmias. Nearly half (49.2%) of all these patients had VF that needed defibrillation. The rest were supraventricular arrhythmias, particularly atrial fibrillation (AF) and atrial flutter (Flutt). See Table 2.

<table>
<thead>
<tr>
<th>Tachyarrhythmia:</th>
<th>No AVB</th>
<th>Low degree AVB</th>
<th>High degree AVB</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ventricular</td>
<td>25</td>
<td>2</td>
<td>24</td>
<td>51 (78.5)</td>
</tr>
<tr>
<td>VF</td>
<td>18</td>
<td>2</td>
<td>12</td>
<td>32 (49.2)</td>
</tr>
<tr>
<td>Supraventricular</td>
<td>13</td>
<td>0</td>
<td>1</td>
<td>14 (21.5)</td>
</tr>
<tr>
<td>AF</td>
<td>11</td>
<td>0</td>
<td>1</td>
<td>12 (18.5)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>38</td>
<td>2</td>
<td>25</td>
<td>65 (100)</td>
</tr>
</tbody>
</table>

( ) = %

Low degree AVB = First degree AVB and Wenckebach phenomenon

*Table 2:* Arrhythmias observed in patients with acute inferior myocardial Infarction.
TREATMENT

Table 3 gives a summary of the various forms of treatment all the patients received.

<table>
<thead>
<tr>
<th>Specific intervention:</th>
<th>n=292</th>
<th>(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thrombolysis</td>
<td>128</td>
<td>43.8</td>
</tr>
<tr>
<td>Primary PTCA</td>
<td>37</td>
<td>12.8</td>
</tr>
<tr>
<td>Rescue PTCA</td>
<td>13</td>
<td>4.5</td>
</tr>
<tr>
<td>Urgent CABG</td>
<td>8</td>
<td>2.7</td>
</tr>
<tr>
<td>Supportive treatment:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IABP</td>
<td>16</td>
<td>5.5</td>
</tr>
<tr>
<td>TPM</td>
<td>30</td>
<td>10.3</td>
</tr>
<tr>
<td>PPM</td>
<td>3</td>
<td>1.0</td>
</tr>
<tr>
<td>Inotropic support</td>
<td>50</td>
<td>17.1</td>
</tr>
<tr>
<td>Defibrillation</td>
<td>36</td>
<td>12.3</td>
</tr>
<tr>
<td>Mechanical ventilation</td>
<td>12</td>
<td>4.1</td>
</tr>
<tr>
<td>B-Blocker on discharge</td>
<td>191</td>
<td>65.4</td>
</tr>
</tbody>
</table>

*Table 3:* Various treatment modalities for all the patients admitted with acute inferior myocardial infarction.

Specific interventions:

Revascularisation was attempted in a total of 186 (63.7%) patients. See table 3.

1. Thrombolysis: Of all the patients in the study, 43.8% received a thrombolytic agent.

2. Primary PTCA: Primary PTCA was performed in 12.8% of all patients.

3. Rescue PTCA: Rescue PTCA was performed in 4.5% of the total study population.
4. Urgent CABG: A total of 8 (2.7%) of all patients underwent urgent coronary artery bypass grafting.

Supportive treatment:

1. IABP: A total of 5.5% of all patients had an intra-aortic balloon pump inserted.
2. Temporary pacemaker placement: This procedure was performed in 10.3% of all patients.
3. Permanent pacemaker placement: A total of 1% of all patients had permanent pacemaker placement.
4. Inotropic support: Of all patients 17.1% received inotropic support.
5. Defibrillation: Only 12.3% of all patients were defibrillated.
6. Mechanical ventilation: A total of 4.1% of all patients were ventilated during their hospital admission.

HEART BLOCK

The majority (74.3%) of patients had no AVB. Only 4.1% of all patients had first degree AVB, and even less (2.7%) had Wenckebach block. No patient had Mobitz II block. There were no significant differences between those patients with no AVB and the small number with low degree AVB (first degree AVB and Wenchebach phenomenon). A total of 55 patients (18.8%) had high degree AVB.
<table>
<thead>
<tr>
<th></th>
<th>No AVB</th>
<th>Low degree AVB</th>
<th>High degree AVB</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>General:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (mean)</td>
<td>57.1</td>
<td>56.7</td>
<td>60.7</td>
<td>NS</td>
</tr>
<tr>
<td>Gender: Male</td>
<td>154 (71)</td>
<td>14 (70.0)</td>
<td>34 (61.8)</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>63 (29)</td>
<td>6 (30.0)</td>
<td>21 (38.2)</td>
<td></td>
</tr>
<tr>
<td>Diabetes</td>
<td>53 (24.4)</td>
<td>5 (25)</td>
<td>21 (38.2)</td>
<td>0.04</td>
</tr>
<tr>
<td>Previous MI</td>
<td>33 (15.2)</td>
<td>1 (5.0)</td>
<td>6 (10.9)</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Admission characteristics:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blood pressure</td>
<td>91.6</td>
<td>98.9</td>
<td>83.2</td>
<td>0.008</td>
</tr>
<tr>
<td>Pulse rate</td>
<td>75.8</td>
<td>68.7</td>
<td>62.9</td>
<td>0.001</td>
</tr>
<tr>
<td>Cardiogenic shock</td>
<td>24 (11.1)</td>
<td>1 (5.0)</td>
<td>24 (43.6)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td><strong>ECG Diagnosis:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inferior</td>
<td>153 (70.5)</td>
<td>13 (65.0)</td>
<td>45 (81.8)</td>
<td>NS</td>
</tr>
<tr>
<td>Inferior plus</td>
<td>64 (29.5)</td>
<td>7 (35.0)</td>
<td>10 (18.2)</td>
<td>NS</td>
</tr>
<tr>
<td>RV involvement</td>
<td>34 (15.7)</td>
<td>4 (20.0)</td>
<td>17 (30.9)</td>
<td>0.01</td>
</tr>
<tr>
<td><strong>Specific Intervention:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thrombolysis</td>
<td>95 (43.8)</td>
<td>7 (35.0)</td>
<td>26 (47.3)</td>
<td>NS</td>
</tr>
<tr>
<td>Primary PTCA</td>
<td>25 (11.5)</td>
<td>1 (5.0)</td>
<td>11 (20.0)</td>
<td>NS</td>
</tr>
<tr>
<td>Rescue PTCA</td>
<td>8 (3.7)</td>
<td>0</td>
<td>5 (9.1)</td>
<td>NS</td>
</tr>
<tr>
<td>CABG</td>
<td>6 (2.8)</td>
<td>0</td>
<td>2 (3.6)</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Supportive treatment:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IABP</td>
<td>9 (4.1)</td>
<td>1 (5.0)</td>
<td>6 (10.9)</td>
<td>0.05</td>
</tr>
<tr>
<td>TPM</td>
<td>7 (3.2)</td>
<td>1 (5.0)</td>
<td>22 (40.0)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>PPM</td>
<td>1 (0.5)</td>
<td>0</td>
<td>2 (3.6)</td>
<td>0.03</td>
</tr>
<tr>
<td>Inotropic support</td>
<td>23 (10.6)</td>
<td>4 (20.0)</td>
<td>23 (41.8)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Defibrilation</td>
<td>22 (10.1)</td>
<td>2 (10.0)</td>
<td>12 (21.2)</td>
<td>0.02</td>
</tr>
<tr>
<td>Ventilated</td>
<td>6 (2.8)</td>
<td>1 (5.0)</td>
<td>5 (9.1)</td>
<td>0.04</td>
</tr>
<tr>
<td>B-Blocker</td>
<td>164 (75.6)</td>
<td>8 (40.0)</td>
<td>19 (34.5)</td>
<td>NS</td>
</tr>
<tr>
<td>Mortality</td>
<td>12 (5.5)</td>
<td>0</td>
<td>9 (16.4)</td>
<td>0.003</td>
</tr>
</tbody>
</table>

( ) = %

*Table 4:* Comparative data for the groups without and with various degrees of AVB.
Clarification of abbreviations used in Table 4:

RV: Right ventricle  
PTCA: Percutaneous transluminal coronary angiography  
CABG: Coronary artery bypass graft  
IABP: Intra-aortic balloon pump  
TPM: Temporary pacemaker placement  
PPM: Permanent pacemaker placement

As shown in table 4, the mean ages of these various groups are skewed towards a higher age group for the higher degrees of AVB. As expected, more patients with high grade AVB block had cardiogenic shock, and received more supportive treatments.

MORTALITY

Of the 292 patients admitted with acute inferior myocardial infarction, 21 died. (7.2%) The mortality of those above 65 years was 13.4% compared to 4.8% for those under 65 years. This result was statistically significant. See Table 5. As previously documented, female patients had a higher mortality compared to their male counterparts, 8.9% against 6.4%.
<table>
<thead>
<tr>
<th></th>
<th>TOTAL</th>
<th>DIED</th>
<th>(%)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>MORTALITY</strong></td>
<td>292</td>
<td>21</td>
<td>(7.2)</td>
<td></td>
</tr>
<tr>
<td><strong>General</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;65</td>
<td>82</td>
<td>11</td>
<td>(13.4)</td>
<td>0.01</td>
</tr>
<tr>
<td>&lt;65</td>
<td>210</td>
<td>10</td>
<td>(4.8)</td>
<td></td>
</tr>
<tr>
<td>Gender:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>male</td>
<td>202</td>
<td>13</td>
<td>(6.4)</td>
<td>NS</td>
</tr>
<tr>
<td>female</td>
<td>90</td>
<td>8</td>
<td>(8.9)</td>
<td>NS</td>
</tr>
<tr>
<td>Diabetes Mellitus</td>
<td>79</td>
<td>7</td>
<td>(8.9)</td>
<td>NS</td>
</tr>
<tr>
<td>Previous MI</td>
<td>40</td>
<td>8</td>
<td>(20.0)</td>
<td>0.007</td>
</tr>
<tr>
<td><strong>Admission characteristics:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiogenic shock</td>
<td>49</td>
<td>21</td>
<td>(42.9)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td><strong>ECG characteristics:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inferior</td>
<td>211</td>
<td>6</td>
<td>(2.8)</td>
<td>NS</td>
</tr>
<tr>
<td>Inferior plus</td>
<td>81</td>
<td>9</td>
<td>(11.1)</td>
<td>NS</td>
</tr>
<tr>
<td>RV involvement</td>
<td>55</td>
<td>6</td>
<td>(10.9)</td>
<td>NS</td>
</tr>
<tr>
<td><strong>High degree AVB:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Speciﬁc intervention:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thrombolysis</td>
<td>128</td>
<td>3</td>
<td>(2.3)</td>
<td>0.005</td>
</tr>
<tr>
<td>Primary PTCA</td>
<td>37</td>
<td>4</td>
<td>(10.8)</td>
<td>NS</td>
</tr>
<tr>
<td>Rescue PTCA</td>
<td>13</td>
<td>0</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>Urgent CABG</td>
<td>8</td>
<td>0</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td><strong>Supportive treatment:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IABP</td>
<td>16</td>
<td>2</td>
<td>(12.5)</td>
<td>NS</td>
</tr>
<tr>
<td>TPM</td>
<td>30</td>
<td>8</td>
<td>(26.7)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>PPM</td>
<td>3</td>
<td>0</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>Inotropic support</td>
<td>50</td>
<td>15</td>
<td>(30.0)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Defibrillation</td>
<td>36</td>
<td>9</td>
<td>(25.0)</td>
<td>0.0003</td>
</tr>
<tr>
<td>Mechanical ventilation</td>
<td>12</td>
<td>8</td>
<td>(66.7)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>B-Blocker on discharge</td>
<td>191</td>
<td>0</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Table 5:* Total mortality data in the various categories of all the patients admitted with acute inferior myocardial infarction.
Mortality was also significantly higher (20%) in the 40 patients who had previous myocardial infarctions. Cardiogenic shock (49 patients) was associated with the highest mortality (42.9%), followed by high degree AVB with 16.4%. More extensive ECG changes was associated with 11.1% mortality, followed by RV involvement with 10.9%. All patients who died had cardiogenic shock. No deaths were sudden and unexpected.
Univariate analysis suggested that age (p<0.0001), previous MI (p=0.007), mean admission BP (p=0.008), cardiogenic shock (p<0.0001) and high degree AVB (p=0.03) were all associated with increased mortality. However, multivariate analysis showed that only cardiogenic shock and previous MI were significantly associated with death.

<table>
<thead>
<tr>
<th>Mortality:</th>
<th>F Value</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiogenic shock</td>
<td>120.8</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Previous MI</td>
<td>14.2</td>
<td>0.0002</td>
</tr>
</tbody>
</table>

*Table 6:* Multivariate analysis for increased mortality

**ATTEMPTED REVASCULARISATION**

**Thrombolysis:**

One hundred and twenty eight of the 292 patients (43.8%) received a thrombolytic agent. Only 3 (2.3%) of these patients died. Reasons for not receiving a thrombolytic agent included:

- contra-indications
- late presentations
- cardiogenic shock

The mortality in the 164 patients who did not received a thrombolytic agent was 11%.
<table>
<thead>
<tr>
<th></th>
<th>Total</th>
<th>Thrombolysis</th>
<th>Primary PTCA</th>
<th>Total revascul</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>High degree AVB</strong></td>
<td>55</td>
<td>26</td>
<td>11</td>
<td>37 (67.3)</td>
</tr>
<tr>
<td>Dead</td>
<td>9</td>
<td>1 (3.8)</td>
<td>2 (18.0)</td>
<td>3 (8.1)</td>
</tr>
<tr>
<td><strong>Cardiogenic shock</strong></td>
<td>49</td>
<td>16</td>
<td>11</td>
<td>27 (55.1)</td>
</tr>
<tr>
<td>Dead</td>
<td>21</td>
<td>3 (18.8)</td>
<td>4 (36.4)</td>
<td>7 (25.9)</td>
</tr>
<tr>
<td><strong>Age &gt;65</strong></td>
<td>82</td>
<td>34</td>
<td>6</td>
<td>40 (48.8)</td>
</tr>
<tr>
<td>Dead</td>
<td>11</td>
<td>1 (2.9)</td>
<td>1 (16.6)</td>
<td>2 (5.0)</td>
</tr>
</tbody>
</table>

() = %

*Table 7:* Attempted revascularisation in all high risk groups.

Of the 55 patients with high grade AVB, 26 (47.3%) received a thrombolytic agent. Only 1 (3.8%) of these patients died.

Sixteen of the 49 (32.7%) patients with cardiogenic shock were given a thrombolytic agent of whom only 3 (18.8%) died, compared to the 42.9% mortality in the whole group with cardiogenic shock.

A thrombolytic agent was also given to 34 of 82 patients over the age of 65 (41.5%). In this group only 1 patient died (2.9%). See Table 7 for breakdown.

**Angioplasty:**

Angioplasty was attempted in 50 patients (17.1%). Primary PTCA was attempted in 37 (12.8%) patients and rescue PTCA after failed thrombolysis in 13 (4.5%) patients.
Primary PTCA was attempted in 11 of 49 patients with cardiogenic shock (22.4%), and in 11 of 55 patients with high grade block (20%), reflecting a bias to perform this procedure in sicker patients. Mortality in patients receiving primary PTCA was 18% for high grade AVB, 36.4% for cardiogenic shock and 16.6% for patients older than 65 years. None of the 13 patients in whom rescue PTCA was performed died. The Overall mortality for PTCA was 8% (4 of 50 patients). See Table 7.

Coronary artery bypass grafting:
Urgent CABG was performed in 8 patients in total (2.7%). No patient in this group died.

SUPPORTIVE TREATMENT
1. IABP: Two of the 16 patients in whom an intra-aortic balloon pump was inserted died (12.5%). Six patients with high grade AVB had an IABP inserted, of whom 1 died (16.7%). Likewise, 10 patients with cardiogenic shock had IABP inserted, of whom 2 died (20.0%).
2. TPM: Of the 30 patients in whom this procedure was performed, 8 (26.7%) died. Twenty two of 49 patients with high grade AVB had a TPM inserted, of whom 6 died (12.2%).
3. PPM: A total of 3 patients had permanent pacemakers fitted because of persistent high grade block. No one in this group died.
with high grade block, 23 patients received inotropic support, of whom 8 died (34.8%).

5. Mechanical ventilation: From a total of 12 patients who were ventilated, 8 died (66.7%).

6. Defibrillation: A total of 36 patients were defibrillated of whom 12 died (33.3%). Thirty-two (88.9%) of these patients had VF and 4 (11.1%) had AF that needed cardioversion.

B-Blocker therapy on discharge:

Of the 271 patients who survived, 164 were discharged from the CCU on B-blocker maintenance therapy (60.5%).
DISCUSSION

The results of this study confirm that inferior myocardial infarction is associated with a low mortality (7.2%), and in the majority, the clinical course is benign. However, high degree heart block was associated with a four-fold increase in the mortality of this group of patients to 16.4%
These findings are in agreement with previous studies. 5

HOW DOES HEART BLOCK INFLUENCE MORTALITY?

Patients with high grade AVB were generally older. The admission characteristics for this group of patients were also significantly different, compared to the group without high degree AVB. Their admission blood pressure and pulse rate was significantly lower. Furthermore, close to half of these patients developed cardiogenic shock, either on admission or during their hospital stay.

Surprisingly, only a very few patients with high degree AVB (18.2%) had a more extensive pattern of infarction on ECG criteria. This means that over 80% of these high-risk patients had straightforward inferior myocardial infarctions in this study. A more extensive pattern of infarction on ECG is generally associated with a larger infarct size,
greater myocardial tissue damage and therefore a much higher rate of complications and mortality. This result is different from previous studies, but the reason for this and the explanation therefore is not immediately apparent.

RV involvement, however, was significantly more prevalent in the group of patients with high degree AVB (30.9% vs 16.0%). This would suggest that these patients have a larger infarct size and thus, complication rate. Although the overall incidence of RV involvement in this study is smaller compared to previous studies, it is clear that RV involvement is associated with significant more atrioventricular nodal conduction disturbances and haemodynamic compromise. RV involvement could therefore, in part explain the higher mortality associated with high degree AVB.

The treatment of patients with high degree AVB did not differ significantly from those without high degree AVB. Both groups received thrombolytic therapy at the same rate, but it appears that patients with high degree AVB had more attempts at primary PTCA, (20% vs 11%) and rescue PTCA (9.1% vs 3.4%). It is clear that the patients with high grade AVB were generally sicker and this is reflected by attempts at revascularisation and restoration of coronary circulation, not only to improve the haemodynamics, but also to restore normal sinus rhythm.
The fact that patients with high grade AVB were generally sicker with more cardiovascular instability was brought out by the fact that they receive significantly more in terms of supportive treatment. This reached statistically significant levels across all the categories of supportive treatments surveyed.

It is clear therefore, that high degree AVB is associated with a certain degree of increased haemodynamic instability and increased incidence of cardiogenic shock. This may result in the increased mortality as shown in this study. Thus, high degree AVB increases mortality by increasing the risk of cardiogenic shock. However, high degree AVB is not an independent predictor of mortality as shown by multivariate analysis.

Other mechanisms suggested for increased mortality have not been shown in this study to be independent predictors of mortality. The role of the below mentioned factors, although not entirely clear, can still be appreciated:

- larger infarct size and associated cardiovascular instability
- RV involvement
MORTALITY

Although this study was initiated to show that high grade AVB was an independent predictor of mortality, the results prove this not to be the case. The higher mortality associated with high degree AVB is explained by it's association with cardiogenic shock. A few other prognostic factors were associated with increased mortality and therefore are worth discussing.

Advanced age is not an independent predictor of increased mortality in the setting of inferior myocardial infarction. However, advancing age was shown to be associated with some increases mortality and it should be kept in mind when treating these patients.

Gender also seems to have important mortality association. The results showed that females are more likely to die than males in the setting of inferior myocardial infarction.

More importantly, all patients who died had cardiogenic shock, either on admission or during their hospital stay. It is congruent with the conclusion that death was directly related to pump failure. It appears important to avoid the development of cardiogenic shock in these
patients, and to effectively and promptly reverse the cardiovascular instability once it has developed.

Another interesting point in the treatment of the group of patients who died was that very few received thrombolytic therapy (14.3%). However, compared to the group that survived, more of the patients in the group that died received primary PTCA (19% vs 12.2%). There is very little one can do when there are contra-indications to thrombolysis, or when patients present late, but in the high risk group all efforts should be made in an attempt at early revascularisation. There is a role here for primary PTCA, and all such patients should be offered this form of treatment where available. There is enough evidence to suggest that early treatment with a thrombolytic agent in high-risk patients with inferior myocardial infarction reduces mortality.\(^8\)

As expected there was an increased tendency to use temporary pacemaker placement as a form of treatment in the group with high degree AVB. Despite this, a third of these patients died of cardiovascular compromise. Thus, patients still died of cardiogenic shock that was not avoided by temporary pacing. Similar observations have been made by other independent investigators.\(^7\) Placement of temporary pacemaker appears to be beneficial in patients with high grade block who have a slow heart rate and are haemodynamically unstable. TPM does therefor
not improve outcome in its own right, but it simplifies management in this group of patients.

HIGH RISK GROUP:
Patients at high risk of increased mortality can be identified by the following characteristics;

- Cardiogenic shock
- Previous myocardial infarction

Mortality was lower in high risk patients in whom revascularisation was attempted. Because this was not a randomized, prospective study, conclusions can only be tentative. However, the results suggest that revascularisation should be attempted whenever possible, particularly in high risk groups. Receipt of thrombolytic therapy identified a very low risk group (mortality 2.3%), perhaps because of early presentation.
LIMITATIONS OF THE STUDY

The most obvious limitation of this study and therefore, of its conclusions, is the fact that it is a retrospective/observational analysis. However, even this type of review has some value, particularly:

- As an audit of management practice
- To compare local data with recognised international trend
- As grounds on which prospective trials should be based.

The population studied may in its own right be a limitation. As GSH is the referral centre for other secondary hospitals like GF Jooste Hospital, New Somerset Hospital and Victoria Hospital, the more benign forms of inferior myocardial infarction would have been treated there. Some selection bias inevitably took place for admission to GSH CCU, rather than to GSH medical wards or the other peripheral hospitals. Thus, the group of patients included in this study may not be truly representative of the entire spectrum of patients with inferior myocardial infarction. Future studies involving this subject should probably factor this into the outcome equation to make it not only more representative, but also more meaningful.

Results and conclusions of a study such as this are limited by the non-uniformity within the groups compared. The various or non-uniform
indications for active intervention limit the interpretation of this strategy generally.

Despite this, the feeling is that sufficient numbers of patients were analysed to give a credible account on the effect of high degree AVB on mortality in patients admitted with acute inferior myocardial infarction in GSH CCU.

SUGGESTED FUTURE STUDY

Cardiogenic shock independently increase mortality in patients with acute inferior myocardial infarction. However, a subset of patients with high degree AVB and cardiogenic shock also has high mortality rates. I, therefor suggest that a future study looking at all patients with inferior myocardial infarction and high degree AVB in the presence of cardiogenic shock be studied prospectively, specifically with regard to early revascularisation.

The role of RV infarction in this setting is very speculative and a randomised prospective study addressing this issue is further suggested. A proposed study would be to look at the effect of early revascularisation, either by thrombolytic therapy or primary PTCA, or combination thereof, on mortality in patients with acute inferior myocardial infarction and high degree AVB with and without RV involvement.
CONCLUSIONS

♦ Inferior myocardial infarction generally is associated with low in hospital mortality.

♦ A significant number of patients with high degree AVB also have RV involvement.

♦ Cardiogenic shock is uniformly found to be an independent predictor of both increased mortality and high grade AVB, explaining the increased mortality observed in patients with this rhythm.

♦ Receipt of a thrombolytic drug identifies a group with low mortality, particularly if cardiogenic shock is absent.

♦ Attempts at early revascularisation appear to be important, especially in those who get thrombolytic therapy. Attempts should be made to give thrombolytic therapy timeously.

♦ Patients with diabetes mellitus are more likely to have high degree AVB.

♦ Patients who previously suffered a myocardial infarction are more likely to die in the setting of a new inferior myocardial infarction.

♦ Older age and female sex are also associated with increased mortality, although not independently.
REFERENCES


