CHAPTER IV

Clinical and Electrophysiological Changes after digitalis therapy in Rheumatic Heart Disease with Congestive Heart Failure
RHEUMATIC VALVULAR DISEASE

The Rheumatic fever can be separated in two division:

1. Rheumatic fever with Carditis
2. Mechanical consequences of Rheumatic fever
   i.e. Rheumatic Valvular diseases

4.1 Rheumatic fever with Carditis:

It is the most common cause of heart disease in children. The evidence is 11-30 per 10,000 person per year. In adult 25 percent of heart disease is rheumatic origin (Idem, 1951). In children 5-19 years of age 2-6 per 1,000 (Hill et al, 1949). In India Rheumatic heart disease constitutes 16.5 to 50% of the cardiac patient in hospitals. The prevalence rate of Rheumatic fever in village population 2/1,000 and urban population 2.07/1,000 for women and 1.23/1,000 for men.

4.2 Etiology:

1. It is believed to be an immunological lesion which occurs as a delayed sequelae of Group A haemolytic streptococci infection of the pharynx but not of the skin.
2. The attack rate of Acute Rheumatic fever following streptococcal infection varies with severity of infection, ranging from 0.3 to 3.0% (Park, M.K., 1981).
3. Important predisposing factors are -
   i) High family incidence
   ii) Low socioeconomic status
   iii) Genetic predisposition probably inherited on Mendelian excessive pattern (Ghai, O.P., 1982)
   iv) Age between 5 - 15 years with high peak at 8 years.
4.3 Pathology:

1. The inflammatory lesion is found in many parts of the body notably in the heart, brain, joints and skin.

2. Aschoff bodies in the atrial myocardium are believed to be characteristic of Rheumatic fever.

3. Valvular damage most frequently involves the mitral valve, less commonly aortic and very rarely tricuspid and pulmonary.

   Carditis occurs in 40 - 50% patients.

   Sign of carditis: Usually develops within 1st 2nd weeks of illness.

   Tachycardia (out of proportion for the degree of fever).

   Significant heart murmur.

   a) Apical diastolic murmur
   b) Apical systolic murmur
   c) Aortic diastolic murmur

Other features:

a) Pericarditis
b) Cardiomegaly
c) Signs of congestive heart failure

4.4 Investigations:

1. Complete Blood Count
2. Acute phase reactant (E.S.R.)
3. Asotiter
4. Throat Swab
5. Chest X'ray
6. E.C.G.
4.5 **Treatment of Carditis**:

1. Complete bed rest with orthopneic position
2. Moist oxygen Inhalation
3. Inj. Diazepam (0.2 mg./kg. I.V.)
4. Restriction of Sodium and fluid
5. Prednisolone 6 weeks with gradually tappering doses
6. Digoxin 0.04 mg/kg digitalising dose. 2/3rd of total dose given I.V. in 24 hours. Followed by orally 0.02 mg/kg in 2 divided dose. Frusemide 0.5 mg/kg 8 hourly.
7. Supplemental Potassium Therapy 2 mEq/kg/day.

4.6 **Mitral Stenosis**:

Mitral stenosis of Rheumatic origin results from:

i) Fibrosis of mitral ring

ii) Commissural adhesion

iii) Contracture of valve leaflets, chordae and papillary muscles. It usually takes 5 - 10 years or more for the lesion to become fully established. Sanyal et (1987) has reported mitral valvular disease in India after 4 - 5 years.

The stenosis is considered critical if the valvular orifice is reduced to 25% or less of the expected normal (Nelson et al 1987). Such reduction results in increased pressure and hypertrophy of the left atrium. This increased pulmonary vascular resistance and pulmonary hypertension. Right ventricular and right aerial dilatation and hypertrophy ensure and are followed by right sided heart failure.
4.7 Treatment of Mitral Stenosis:
   a) The management of mitral stenosis is essentially surgical. The patient should be digitalised and given diuretics. Digitalis helps by -
   
   i) Reducing the heart rate and increasing the left ventricular filling
   
   ii) Increasing the left atrial contractibility and
   
   iii) maintaining a slower heart rate on exertion and thus better forward flow. Close mitral valvotomy is the best surgical approach in India.
   
   b) Maintenance of good dental hygiene and antibiotic prophylaxis against infective endocarditis.
   
   c) Prevention of Recurrence of Rheumatic fever with Penicillin
   
   d) Varying degree of restriction of activity.

4.8 Mitral Regurgitation:
   Mitral regurgitation is the commonest manifestation of acute as well as previous Rheumatic carditis. O.P. Ghai has reported 850 patients of Rheumatic heart disease below the age of 12 years, 750 patients exhibited pure or dominant mitral regurgitation. These patients who present with mitral regurgitation during rheumatic carditis have the 25 - 75% chance of disappearing of the murmur within the first decade of attack (Massel, et al, 1961, Wilson et al, 1962).

4.9 Pathology:
   i) Rigidity and shortening of the valve cusps particularly at the post commissure
   
   ii) Mural leflet being displaced into the ventricle and adhering to the wall
iii) The valve tissue is thick with its edges rolled
iv) Chordae Tendinae are contracted through scar formation
v) The valve ring becomes dilated owing to a combination of inflammatory damage and ventricular dilatation
vii) Dilatation of left ventricle and left atrium

4.10 Treatment of M.R.:

1. Preventive measure against infective endocarditis
2. Propyl®Cjfti| against recurrence of Rheumatic fever
3. Anticongestive measures:
   i) Digitalis
   ii) Diuretics (Frusemid)
   iii) Oral Potassium preparation
   iv) Supportive therapy

4.11 Rheumatic Heart Disease:

Digitalis has been studied in the following group of patients:

15 cases of Rheumatic heart disease of which 5 were mitral incompetence and 5 mitral stenosis and 5 cases of acute Rheumatic carditis were studied. In established lesion of Rheumatic heart disease the cause of failure was mechanical. All the cases were treated with Digitalis, diuretics along with antibiotics (Ampicillin) and supportive measures. In cases of Rheumatic carditis steroid was added along with above therapeutic measures.

The detail clinical, radiological and electrophysiological (i.e. E.C.G.) were as follows:
All of them had congestive cardiac failure i.e. biventricular failure. The children usually presented with biventricular failure not as a left or right ventricular failure irrespective of etiology.

4.12 Clinical profile:
All the children are irritable and restless. They have dry hacking, persistent cough, suppression of urine volume for 24 - 36 hours. Examination revealed normal colour except in cases of Rheumatic carditis who are pale looking, extremities are cold in mitral stenosis. Blood pressure is 90 - 110 mm of Hg and 54 - 64 mm of Hg both systolic and diastolic respectively. These are normal for the age. Oedema feet is present in two cases of mitral incompetence. Pulse rate is 140 - 160 per minute and regular, jugular venous pressure is raised. Liver is 3-4 cm below the right costal margin along the mid clavicular line and is tender. Apex beat is 1 - 2.5 cm outside the mild clavicular line over left 5th intercostal space. Heart sounds are soft with gallop rhythm. There are basal creps and ronchi.

4.13 Clinical observation after Decongestant Therapy:
(Digitalis and Diuretics)
Restlessness is controlled after (1/2 - 1 hour) of sedation which is given in the form of chloral hydrate or Diazepam. Hacking cough is controlled within 2 - 21/2 hours. Orthopnoea is controlled within 6 - 8 hours irrespective of etiology. Patient usually passes urine with 2 - 4 hours and gradually urine volume increases and pulse rate settles to 120 - 130 per minute. The Jugular venous pressure is slightly raised and liver size decreases by 12 hours and in cases of carditis it takes longer time
upto 24 hours. There is no change in blood pressure, Gallop rhythm is controlled by 12 - 18 hours and heart rate is settled to 120 - 130 per minute within this time. Liver size comes to normal within 72 - 96 hours, though the size starts decreasing within 3 - 4 hours and disappears within 6 - 12 hours with addition of Amoninophylline. Creps disappears by 24 - 72 hours. In case secondary associated infection it takes 7 days. Jugular venous and hepatojugular reflex is exggerated upto 2 - 3 weeks. There is loss of weight by 1 - 1.5 kg by 3 - 5 days depending on the initial weight.
The Bar diagram showing the heart rate changes after Digitalis therapy in cases of Rheumatic Carditis with congestive heart failure.

The heart rate:
Before digitalis therapy - 130 ± 1.26
After digitalis therapy - 106± 2.19
P value <0.01

The Bar diagram showing the P-R intervals changes after digitalis therapy in cases of Rheumatic Carditis with congestive heart failure.

The P-R intervals (in sec.)
Before Digitalis therapy - 0.106 ± 4.89x10^-7
After Digitalis therapy - 0.12 ± 6.32x10^-7
P value <0.01
Before Digitalis therapy

After Digitalis therapy

Before Digitalis therapy

After Digitalis therapy
E.E.G. changes before and after Digitalis therapy in Rheumatic carditis

Before                  After

ST segment flat
T wave inversion

Before   After

V₄

ST segment elevated
T wave upright

Before   After

V₄

T wave inversion

Before   After

V₅

T wave upright

Before   After

V₅

ST segment depressed

Before   After

aVF

ST segment elevated

Before   After

aVF

ST segment slightly elevated

Before

aVF

ST segment well elevated

After
### Rheumatic Carditis

**4.14 Before Digitalis Therapy:**

<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
<th>S.D.</th>
<th>P.V. Stat</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate</td>
<td>128</td>
<td>130</td>
<td>130</td>
</tr>
<tr>
<td>Axis</td>
<td>+40°</td>
<td>+50°</td>
<td>+30°</td>
</tr>
<tr>
<td>Lie</td>
<td>Semi vertical in all cases</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rotation</td>
<td>Counter Clockwise in all cases</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P wave</td>
<td>Normal in all cases</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P-R interval</td>
<td>.11</td>
<td>.10</td>
<td>.11</td>
</tr>
<tr>
<td>Low voltage</td>
<td>In Lead I, aVF</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ST segment</td>
<td>Flat in Lead I</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ST segment</td>
<td>Depression in Lead II, III, aVF and Chest leads</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T wave</td>
<td>Inversion variable from V₁ to V₆</td>
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<td></td>
</tr>
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</table>

**4.15 After Digitalis therapy:**

<table>
<thead>
<tr>
<th></th>
<th>104</th>
<th>106</th>
<th>110</th>
<th>106</th>
<th>104</th>
<th>106</th>
<th>2.19</th>
<th>&lt;0.01 Sig</th>
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<tbody>
<tr>
<td>Heart rate</td>
<td>60</td>
<td>55</td>
<td>30</td>
<td>30</td>
<td>45</td>
<td>44</td>
<td>12.40</td>
<td></td>
</tr>
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<td>Axis</td>
<td>Semi vertical in all cases</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lie</td>
<td>Counter Clockwise rotation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P wave</td>
<td>Normal in all cases</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P-R interval</td>
<td>.12</td>
<td>.13</td>
<td>.12</td>
<td>.11</td>
<td>.12</td>
<td>0.12</td>
<td>6.32x10⁻³</td>
<td>&lt;0.01 Sig</td>
</tr>
<tr>
<td>ST segment</td>
<td>Voltage improved almost normal</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ST segment</td>
<td>Flat remained in lead</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ST segment</td>
<td>Elevation in Lead II, aVF and Chest Lead</td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T wave</td>
<td>Upright in V₄ and V₆</td>
<td></td>
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<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

S.D. = Standard Deviation, P.V. = P value, Stat = Statistically, Sig = Significant.
4.16 Observation:

The most significant changes in lowering of heart rate. The mean values of heart rate before and after control of failure are 130 and 106 respectively. The P value is \( < 0.01 \). The P-R interval is prolonged. The mean values are 0.106 and 0.12 respectively before and after control of failure and P value is \( < 0.01 \). The other significant changes are the improvement of voltage, elevation of ST segment and disappearance of T wave inversion.

4.17 Discussion:

The digitalis is the drug of choice for congestive heart failure of any etiology i.e. valvular or myocardial disease (Laurance, D.R. 1975). Digitalis and steroid are advised in the treatment of congestive heart failure in Rheumatic myocarditis (Richard, E.B. and Vaughan, V.C. 1987). But the digitalis should be used very cautiously. The main effects of digitalis in the failing heart is the greater stroke and minute output with decreased implying in a shorter which allows a greater diastolic rest for a given cardiac output.

The significant changes observed in E.C.G. is the slowing heart rate. This achieved due to depression of conducting tissue and increased in vagal tone (Laurance, D.R., 1975). There is significant decrease in heart rate with P value \( \leq 0.01 \). The P value of P-R interval is also \( < 0.01 \) which is highly significant. The increased P-R interval is due to slowing of conduction velocity (Laurance, D.R. 1978). The increase in voltage and elevation of ST segment and disappearance of inversion of T wave may be due to combined effect of steriod and digitalis.
E.C.G. changes before and after Digitalis therapy in Mitral Stenosis

Before

After

Before

After

P pulmonale

P pulmonale

P wave - 3 mm

P wave -3.5 mm

V3

V3

AVL

AVL

R - 27 mm

R - 19 mm

S - 11 mm

S - 12 mm

S - 15 mm

S - 14 mm

AVF

AVF

AVR

AVR

R - 19 mm

R -17 mm

R - 6 mm

R - 6 mm

S - 22 mm

S - 23 mm

S - 11 mm

S - 12 mm
Mitral Stenosis

4.18 Before Digitalis therapy:

<table>
<thead>
<tr>
<th>Heart rate</th>
<th>138</th>
<th>130</th>
<th>144</th>
<th>132</th>
<th>140</th>
<th>138</th>
<th>4</th>
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<tbody>
<tr>
<td>Axis</td>
<td>15°</td>
<td>45°</td>
<td>30°</td>
<td>45°</td>
<td>30°</td>
<td>33°</td>
<td>11.22°</td>
</tr>
<tr>
<td>Lie</td>
<td>Semi vertical in all cases</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Rotation</td>
<td>Counter Clockwise</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>P wave</td>
<td>P mitral in all cases</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P-R interval</td>
<td>.10</td>
<td>.10</td>
<td>.10</td>
<td>.11</td>
<td>.10</td>
<td>0.102</td>
<td>4x10^-3</td>
</tr>
<tr>
<td>R wave</td>
<td>In V3 varies from 25-28 mm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>S wave</td>
<td>In V2 - V3 varies from 12-15 mm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>R wave</td>
<td>In V5, V6 varies from 14-16 mm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>S wave</td>
<td>In V5, V6 varies from 22-23 mm</td>
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<td></td>
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4.19 After Digitalis therapy:

<table>
<thead>
<tr>
<th>Heart rate</th>
<th>106</th>
<th>104</th>
<th>108</th>
<th>106</th>
<th>106</th>
<th>106</th>
<th>1.26</th>
<th>&lt;0.01 Sig</th>
</tr>
</thead>
<tbody>
<tr>
<td>Axis</td>
<td>15°</td>
<td>45°</td>
<td>30°</td>
<td>45°</td>
<td>30°</td>
<td>33°</td>
<td>11.22°</td>
<td></td>
</tr>
<tr>
<td>Lie</td>
<td>Semi Vertical</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rotation</td>
<td>Counter Clockwise</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P wave</td>
<td>P mitral in all cases</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P-R interval</td>
<td>.12</td>
<td>.12</td>
<td>.13</td>
<td>.12</td>
<td>.13</td>
<td>0.124</td>
<td>4.89x10^-3 &lt;0.01 Sig</td>
<td></td>
</tr>
<tr>
<td>R wave</td>
<td>In V3 varies from 22-25 mm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>S wave</td>
<td>In V2 - V3 varies from 12-14 mm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>R wave</td>
<td>In V5, V6 varies from 12-14 mm</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>S wave</td>
<td>In V5, V6 varies from 20-23 mm</td>
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</tbody>
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S.D. = Standard Deviation, P.V. = P. Value
St = Statistically, Sig = Significant
## Mitral Stenosis

### 4.18 Before Digitalis therapy:

<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
<th>S.D.</th>
<th>P.V. St.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate</td>
<td>138</td>
<td>130</td>
<td>144</td>
</tr>
<tr>
<td>Axis</td>
<td>15°</td>
<td>45°</td>
<td>30°</td>
</tr>
<tr>
<td>Lie</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rotation</td>
<td>Counter Clockwise</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P wave</td>
<td>P mitral in all cases</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P-R interval</td>
<td>.10</td>
<td>.10</td>
<td>.11</td>
</tr>
<tr>
<td>R wave</td>
<td>In V₃ varies from 25-28 mm</td>
<td></td>
<td></td>
</tr>
<tr>
<td>S wave</td>
<td>In V₂ - V₃ varies from 12-15 mm</td>
<td></td>
<td></td>
</tr>
<tr>
<td>R wave</td>
<td>In V₅, V₆ varies from 14-16 mm</td>
<td></td>
<td></td>
</tr>
<tr>
<td>S wave</td>
<td>In V₅, V₆ varies from 22-23 mm</td>
<td></td>
<td></td>
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</tbody>
</table>

### 4.19 After Digitalis therapy:

<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
<th>S.D.</th>
<th>P.V. St.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate</td>
<td>106</td>
<td>104</td>
<td>108</td>
</tr>
<tr>
<td>Axis</td>
<td>15°</td>
<td>45°</td>
<td>30°</td>
</tr>
<tr>
<td>Lie</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rotation</td>
<td>Counter Clockwise</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P wave</td>
<td>P mitral in all cases</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P-R interval</td>
<td>.12</td>
<td>.12</td>
<td>.13</td>
</tr>
<tr>
<td>R wave</td>
<td>In V₃ varies from 22-25 mm</td>
<td></td>
<td></td>
</tr>
<tr>
<td>S wave</td>
<td>In V₂ - V₃ varies from 12-14 mm</td>
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</tr>
<tr>
<td>R wave</td>
<td>In V₅, V₆ varies from 12-14 mm</td>
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<td></td>
</tr>
<tr>
<td>S wave</td>
<td>In V₅, V₆ varies from 20-23 mm</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

S.D. = Standard Deviation,  
P.V. = P Value,  
St = Statistically,  
Sig = Significant.
4.20 Observation:

There is decrease in heart rate. The mean values before and after control of failure are 138 and 106 respectively and P value is $< 0.01$ which is statistically significant. There is no change in Axis, Lie and Rotation. P value is tall and peaked. The mean value of P-R intervals are (before and after failure) 0.102 and 0.124 respectively and P value is $< 0.01$ which is statistically significant.

There is difference in amplitude of 1-3 mm in chest lead and which is not significant. This could be a simple physiological variation.

4.21 Discussion:

All the mitral stenosis cases, the cause of failure is mechanical. The digitalis is well indicated and mechanical failure (Laurance, D.R. 1975). All the cases have got RVH. The cases were diagnose as RVH according to criteria of Marritt, H.J.V. (1968).

There is significant decrease in heart rate. This increase of heart rate is due to depression of conducting tissue and increased in vagal tone (Laurance, D.R. 1975). The P value of P-R intervals is $< 0.01$ which is highly significant. This is due to effect on the conduction of velocity (Laurance, D. R., 1975). The amplitudes of other waves are not at all affected.

Thus digitalis controls the failure without effecting any structural change of the heart.
MR(1) The Baridiagram showing the heart rate changes with Digitalis therapy in cases of Mitral regurgitation with congestive heart failure.

The heart rate:
Before Digitalis therapy - 133.2 ± 6.01
After Digitalis therapy - 107.8 ± 4.9
P value < 0.01

MR(2) The Baridiagram showing the P-R interval in cases of Mitral regurgitation with congestive heart failure.

The P-R intervals (in sec.)
Before Digitalis therapy - 0.102 ± 4x10⁻³
After Digitalis therapy - 0.116 ± 7.48x10⁻³
P value < 0.05

MS(1) The Baridiagram showing the heart rate changes with Digitalis therapy in cases of Mitral stenosis with congestive heart failure.

The heart rate:
Before Digitalis therapy - 138 ± 4
After Digitalis therapy - 106 ± 1.26
P value < 0.01

MS(2) The Baridiagram showing the P-R intervals changes with Digitalis therapy in cases of Mitral stenosis with congestive heart failure.

P - R intervals
Before Digitalis therapy - 0.102 ± 4x10⁻³
After Digitalis therapy - 0.124 ± 4.89x10⁻³
P value < 0.01
Before Digitalis therapy

After Digitalis therapy

Before Digitalis therapy

After Digitalis therapy
E.C.G. changes before and after Digitalis therapy in Mitral Regurgitation

Before

After

V1
R - 2 mm
S - 8 mm

V1
R - 2 mm
S - 8 mm

V3
R - 8 mm
S - 12 mm

V3
R - 8 mm
S - 12 mm

V6
R - 20 mm
S - 3 mm

V6
R - 20 mm
S - 3 mm

V2

V2

V4
R - 6 mm
S - 14 mm

V4
R - 6 mm
S - 14 mm

V4
R - 5 mm
S - 16 mm

V4
R - 5 mm
S - 16 mm
## Mitral Regurgitation

### 4.22 Before Digitalis Therapy

<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
<th>S.D.</th>
<th>P.V. Stat</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate</td>
<td>138</td>
<td>122</td>
<td>132 138 136 133.2 6.01</td>
</tr>
<tr>
<td>Axis</td>
<td>20</td>
<td>25</td>
<td>15 20 15 19 3.75</td>
</tr>
<tr>
<td>Lie</td>
<td>Vertical in one case. Other cases semivertical</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rotation</td>
<td>Counter Clockwise rotation in all cases</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P wave</td>
<td>Normal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P-R interval</td>
<td>.10</td>
<td>.11</td>
<td>.10 .10 .10 0.102 4x10^-3</td>
</tr>
<tr>
<td>S wave</td>
<td>In $V_1$ varies from 6-8 mm</td>
<td></td>
<td></td>
</tr>
<tr>
<td>R wave</td>
<td>In $V_1$ varies from 2-4 mm</td>
<td></td>
<td></td>
</tr>
<tr>
<td>S wave</td>
<td>In $V_2$ varies from 12-15 mm</td>
<td></td>
<td></td>
</tr>
<tr>
<td>R wave</td>
<td>In $V_2 - V_3$ varies from 15-17 &amp; 3-4 mm</td>
<td></td>
<td></td>
</tr>
<tr>
<td>R wave</td>
<td>In $V_6$ varies from 21-25 mm</td>
<td></td>
<td></td>
</tr>
<tr>
<td>S wave</td>
<td>In $V_6$ varies from 3-4 mm Intrinsicsoid deflection varies from 0.04 - 0.05 mm</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### 4.23 After Digitalis Therapy

<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
<th>S.D.</th>
<th>P.V. Stat</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate</td>
<td>115</td>
<td>100</td>
<td>110 106 108 107.8 4.9 0.01 Sig.</td>
</tr>
<tr>
<td>Axis</td>
<td>20</td>
<td>25</td>
<td>20 25 28 23.6 3.13</td>
</tr>
<tr>
<td>Lie</td>
<td>Vertical in one and semivertical in all cases</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rotation</td>
<td>Counter Clockwise</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P wave</td>
<td>Normal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P-R interval</td>
<td>.12</td>
<td>.11</td>
<td>.12 .11 .13 116 7.48x10^-3 0.05 Sig.</td>
</tr>
<tr>
<td>R wave</td>
<td>In $V_1$ varies from 2-4 mm</td>
<td></td>
<td></td>
</tr>
<tr>
<td>S wave</td>
<td>In $V_1$ varies from 6-8 mm</td>
<td></td>
<td></td>
</tr>
<tr>
<td>S wave</td>
<td>In $V_2$ varies from 6-8 mm</td>
<td></td>
<td></td>
</tr>
<tr>
<td>R wave</td>
<td>In $V_2$ varies from 12-15 mm</td>
<td></td>
<td></td>
</tr>
<tr>
<td>R wave</td>
<td>In $V_6$ varies from 22-25 mm</td>
<td></td>
<td></td>
</tr>
<tr>
<td>S wave</td>
<td>In $V_6$ varies from 3-4 mm Intrinsicsoid deflection varies from 0.04 - 0.05 mm</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*S.D. = Standard Deviation, P.V. = P Value, Stat = Statistically, Sig = Significant.*
4.24 **Observation:**

There is decrease in heart rate. The mean values before and after control of failure are 133.2 and 107.8 respectively and P value is $< 0.01$ which is statistically significant.

The P-R interval is prolonged. The mean values before and after control of failure with digitalis are 0.102 and 0.116 respectively and P value is $< 0.05$ which is statistically significant.

There is no change in axis, lie and rotation. The amplitude of R and S wave in different chest leads remained same. There is increased in intrensicoid deflection 0.04 - 0.05 which indicates ventricular dilatation.

4.25 **Discussion:**

In this present study the cause of failure is mechanical, Digitalis is highly recommended in mechanical failure (Laurance, D.R. 1975). Park, M.K. (1989) has described that the E.C.G. findings in children are either left atrial hypertrophy (LAH or Left Ventricular Hypertrophy). In the present experiment, it was border line of Left Ventricular Hypertrophy as per Estes’ Scoring system (Marrioff, H.J.L., 1968).

There is slight ventricular dilatation as evident by the increase intrensicoid deflection 0.04 - 0.05 (Marriott, H.J.L, 1968). The most significant E.C.G. changes are decrease in heart rate and increase in P-R interval in both cases the P values are $< 0.01$ which is highly significant. The decrease of heart rate is due to depression of conducting tissue and increase in vagal tone (Laurance, D.R., 1975). Increase of P-R interval due to slowing of conducting system (Laurance, D.R., 1975).
There is no changes in amplitude of wave and intrensicoid deflection remains same. This indicates that there is affection of structural changes by digitalis when the failure is controlled due to the physiological effect.