CHOLECYSTOCARDIAC LINK INTRA-OPERATIVE EVALUATION DURING BILIARY TRACT SURGERY

Arshad Jamal1 and Jalad Kapoor2

1Assistant Professor, Department of General Surgery, N.C. Medical College, Panipat, Haryana, India.
2Assistant Professor, Department of Anaesthesia, Saraswathi Institute of Medical Sciences, Hapur Rd, Anwarpur, Uttar Pradesh, India.

*Corresponding Author: Dr. Arshad Jamal
Assistant Professor, Department of General Surgery, N.C. Medical College, Panipat, Haryana, India.

ABSTRACT

Introduction: There have been repeated references to reflexes arising from the upper abdominal viscera and especially the biliary tract that could give rise to cardiac arrhythmias, a reduction in coronary blood flow and even catastrophe during surgery of the biliary tract. Various procedures and techniques including paravertebral and/or intercostal block as well as extradural or even intradural block have been tried in past in order to evaluate this phenomena by different workers but with highly variables result. Aims and Objectives: Cholecystocardiac Link Intra-Operative Evaluation During Biliary Tract Surgery. Methods and Material: Sixty adult patients of ASA class I & II (American Society of Anaesthetists) who were posted for elective cholecystectomy and/or common bile duct exploration were taken up for the study. Out of sixty patients 30 patients were normotensive (Group A) and rest of 30 patients were treated hypertensive patients (Group B). Patients with Arrhythmia, IHD and diabetes and with untreated hypertension were excluded from the study. Results: In the present study, patients’ age ranged from 27-45 years (mean 36.33) in Group A and from 25-49 years (mean 35.6) in Group B. Male: Female ratio was 1:14 and 1:9 in Group A and Group B respectively. Pulse rate, heart rhythm, systolic blood pressure, diastolic blood pressure, mean arterial pressure, and rate pressure product during different stages of anaesthesia and surgery in normotensive patients (Gp.A) and treated hypertensive (Gp.B) patients were statistically compared. Conclusion: The measured parameters increased significantly during surgical manipulation. Hemodynamic disturbances secondary to diseased gall bladder and its removal are more marked when cardiovascular system is the seat of proclaimed or latent disorder.

KEYWORDS: Arrhythmia, IHD and diabetes, Cholecystocardiac Link, Intra-Operative Evaluation.

INTRODUCTION

The association of biliary tract and coronary artery disease has been noted clinically for many years. (Babcock R.N. 1909). This relation has been extensively studied in the laboratory. There have been repeated references to reflexes arising from the upper abdominal viscera and especially the biliary tract that could give rise to cardiac arrhythmias, a reduction in coronary blood flow and even catastrophe during surgery of the biliary tract. However the work of several investigators failed to reveal any consistent significant electrocardiographic changes related to manipulation of this area. These changes could probably be due to mesentric traction causing afferent sympathetic stimulation that result in vasodilation of the splanchnic system and venous pooling in splanchnic capacitance vessels. This causes a decrease cardiac return and cardiac output. Parasympathetic nerves are motor to the musculature of gall bladder and bile ducts, but inhibitory to the sphincters. Sympathetic nerves (T7 & T9) are vasomotor and motor to sphincters. Pain from the gall bladder may travel along the vagus, the sympathetic nerves or along the phrenic nerves. It is referred through vagus to stomach, through sympathetic nerves to inferior angle of right scapula, through phrenic nerves to right shoulder. Certain autonomic reflexes from diseased gall bladder are known to bring about circulatory disturbances like decreased coronary blood flow, angina and dysrhythmia (Rains and Riche 1981, Haleem et al, 1991). Various procedure and technique including paravertebral and/or intercostal block as well as extradural or even intradural block have been tried in past in order to evaluate this phenomena by different workers but with highly variables result.
However, contrary to usual belief irritation/stimulation of biliary tract may lead to relative/absolute increase in the sympathetic tone, there by resulting into tachycardia, hypertension and tachyarrhythmias. Orloff in 1981, Katz and Bigger in 1970 suggested that arrhythmias during cholecystectomy can be explained in terms of autonomic nervous system imbalances.

**Pulse and heart rate** observation constitute the simplest monitory aids in the peri-anaesthetic period. Monitoring of pulse during the peri-anaesthetic period tells about the alteration in rate and rhythm of cardiac contractions. The display and the recording of the **electrical changes** within the heart and their interpretation in terms of function and disease is also widely practiced.

In view of the varying results of various investigators, the present study aims to assess and evaluate the cholecystoccardiac link intraoperatively during biliary tract surgery in normotensive and treated hypertensive patients.

**AIMS AND OBJECTIVES**

To evaluate the following parameters preoperatively and during intraoperative and surgical steps of cholecystectomy between normotensive and treated hypertensive patients
1. Pulse Rate
2. Blood Pressure- Systolic and Diastolic
3. Mean Aterial Pressure
4. Rate Pressure Product
5. ECG changes

**REVIEW OF LITERATURE**

The monitoring of the vital functions during anaesthesia in any patient is dependent on the predictability of patients’ responses to drugs, ventilation techniques and other maneuvers associated with anaesthesia, as much as on the responses to the variety of surgical injury. When the course of events is highly predictable, cardio vascular monitoring can be limited to the continous or intermittent non-invasive measurement of certain variables.

**Lennox, Graves et al., 1922:** Studied 48 patients undergoing cholecystectomy, showed no changes in ECG during manipulation of gall bladder.

**Mayo W.J., 1924:** Founded cardiac arrhythmias associated with biliary tract disease.

**Straus and Hamberger 1924:** Reported 4 cases of diseased gall bladder associated with definite cardiac irregularities shown by ECG and pulse. In 3 of these cases, irregularities disappeared post operatively.

**Mendelsohn and Monheit 1956:** Studied 50 patients aged between 24 and 80 years, undergoing biliary tract or upper abdominal surgery. Of 30 patients with cardiac disease preoperatively, 5 developed transient sinus bradycardia and 5 developed ECG changes (Atrioventricular dissociation, Atrial premature beats with lowered T wave, increase ventricular premature beats and recurrence of pre-op transient left bundle branch block).

**Katz and Bigger 1970:** Suggested that arrhythmias are frequently seen even in well managed patients during anaesthesia. The vast majority of arrhythmias do not require drug treatment. It is usually sufficient to eliminate the cause of arrhythmias by lowering the concentration of anaesthetic agent or changing another agent, eliminating excess carbon dioxide by hyperventilation or stopping injection of catecholamines. They observed that most arrhythmias can be explained in terms of autonomic nervous system imbalances.

**Fischer Raab and Von Reumont-J 1983:** Studied the cardio vascular changes during electro stimulation anaesthesia via paravertbral electrodes and halothane anaesthesia and neurolept anaesthesia in 654 patients undergoing for cholecystectomies struma surgery, vein bypass grafting and varicotomies, no difference in heart rate and mean arterial pressure were found during the start of the operation with the different anaesthetic technique.

**J. Magnusson et al.1986:** Studied 30 patients scheduled for cholecystectomy or hernia repair under general anaesthesia with thiopentone-fentanyl and pancuronium. One group (15 patients) received metoprolol tablet 200 mg in slow release form, once daily for two weeks including morning of surgery. The other group received placebo and saline. Metoprolol significantly reduces arterial pressure both during undisturbed anaesthesia in intubation and after extubation. However metaprolol had no effect on variations in systemic vascular resistance.

**Perreault et al. 1990:** Studied 31 patients (18 female 13 male mean age 47 years) undergoing cholecystectomy who received 1 micro gram per kg bolus of sufentanil before induction of anaesthesia with thiopentone. Cardiovascular stability was not achieved in 11 patients who then were given isoflurane. The arterial pressure decreased after sufentanil (P<0.05) (mean BP 108/65 mm Hg & HR 63 beats/m). At 1 minute post incision clinically important hypertension or hypotension did not occur in any patient. I patient receiving beta blocker therapy required atropine to control bradycardia. They concluded that such techniques were valuable to assure good protection of cardiovascular system.
Wester band et al., 1992: Studied the cardiovascular changes in 16 patients undergoing laparoscopic cholecystectomy with serial measurement of mean arterial pressure and HR. Intra peritoneal pressure and expired carbon dioxide were also recorded. Results showed decrease of 30% (P<0.001) in cardiac index and 0.5% (P<0.089) in heart rate, along with increase 15% (P<0.001) in MAP and of 79% (P<0.001) in the calculated total peripheral resistance index. The data suggested that patients with history of cardiac diseases should have preoperative cardiac evaluation and be closely monitored during laparoscopic cholecystectomy.

MATERIAL AND METHOD

The present study entitled Cholecystocardiac link intraoperative evaluation during biliary tract surgery was conducted in Department of General Surgery Jawahar Lal Nehru Medical College, A.M.U., Aligarh. Sixty adult patients of ASA class I & II (American Society of Anaesthesitists) who were posted for elective cholecystectomy and/or common bile duct exploration were taken up for the study. Out of sixty patients 30 patients were normotensive (Group A) and rest of 30 patients were treated hypertensive patients (Group B).

Patients with Arrhythmia, IHD and diabetes and with untreated hypertension were excluded from the study. Patients were evaluated in the ward pre-operatively one day before operation, pulse rate systolic & diastolic blood pressure heart rate and electro cardio gram were recorded. The patients fasted over night and in the morning were pre medicated 1 hr. before surgery with injection buprenorphine (Dose 5-6 micro gram/kg) and promethazine (dose 0.5 mg/kg). After oxygenation for 3-5 minutes, the patients were then induced with thiopentone sodium (dose 4-7 mg/kg) and endotracheal intubation with cuffed endotracheal tube of adequate size was facilitated with suxamethonium (dose 1-2 mg/kg) and anaesthesia was maintained with nitrous oxide (N20) and oxygen (60:40) and pancuronium bromide (dose 0.02 to 0.08 mg/kg) for muscle relaxation.

During perioperative period pulse rate, heart rate blood pressure and ECG monitoring were done continuously with special attention at following times.

- At the time of preanaesthetic check up one day before operation.
- Before induction of anaesthesia.
- After induction of anaesthesia
- At the time of completion of abdominal incision.
- At the time of manipulations and traction of gall bladder during its removal.
- At the time of common bile duct exploration if done.
- At the end of operation.
- At the end of anaesthesia.

From the values thus obtained Mean Arterial Pressure(MAP) was calculated by formula MAP = Diastolic pressure + 1/3 pulse pressure, and Rate Pressure Product (RPP) will be calculated by (Rate pressure product = systolic blood pressure x heart rate).

All patients were followed up post operatively for about 3 months with special attention to blood pressure in hypertensive patients.

The values obtained were tabulated and statistically computed using paired student t-test.

OBSERVATIONS

Table I: Changes in pulse rate in Group A and Group B Patients.

<table>
<thead>
<tr>
<th>Group</th>
<th>Before Induction</th>
<th>After Induction</th>
<th>After Surgical Incision</th>
<th>Cystic Duct Ligation</th>
<th>Cystic Artery Ligation</th>
<th>Gall Bladder Removal</th>
<th>End of Operation</th>
<th>End of Anaesthesia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group A</td>
<td>89.7 ± 11.43</td>
<td>96.6 ± 11.3</td>
<td>89.2 ± 14.98</td>
<td>99.4 ± 15.2</td>
<td>99.9 ± 13.5</td>
<td>100.0 ± 14.08</td>
<td>96.8 ± 13.6</td>
<td>86.03 ± 34</td>
</tr>
<tr>
<td>*P Value</td>
<td>P &lt; .001</td>
<td>P &lt; .001</td>
<td>P &lt; .01</td>
<td>P &lt; .001</td>
<td>P &lt; .001</td>
<td>P &lt; .001</td>
<td>P &lt; .001</td>
<td>P &lt; .001</td>
</tr>
<tr>
<td>Group B</td>
<td>82.56 ± 12.5</td>
<td>91.03 ± 14.87</td>
<td>92.53 ± 12.53</td>
<td>99.46 ± 15.7</td>
<td>94.93 ± 13.5</td>
<td>95.73 ± 11.06</td>
<td>91.86 ± 13.9</td>
<td>85.46 ± 13.8</td>
</tr>
<tr>
<td>*P Value</td>
<td>P &lt; .01</td>
<td>P &lt; .01</td>
<td>P &lt; .01</td>
<td>P &lt; .001</td>
<td>P &lt; .001</td>
<td>P &lt; .001</td>
<td>P &lt; .001</td>
<td>P &lt; .001</td>
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</tbody>
</table>

Table II: Changes in systolic blood pressure in normotensive (Group A) and treated hypertensive patients (Group B).

<table>
<thead>
<tr>
<th>Group</th>
<th>Before Induction</th>
<th>After Induction</th>
<th>After Surgical Incision</th>
<th>Cystic Duct Ligation</th>
<th>Cystic Artery Ligation</th>
<th>Gall Bladder Removal</th>
<th>End of Operation</th>
<th>End of Anaesthesia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group A</td>
<td>124.1 ± 15.18</td>
<td>134.9 ± 16.5</td>
<td>135.0 ± 15.74</td>
<td>143.0 ± 24.9</td>
<td>144.0 ± 16.7</td>
<td>142.0 ± 15.53</td>
<td>131.0 ± 7.5</td>
<td>126.6 ± 14.59</td>
</tr>
<tr>
<td>*P Value</td>
<td>P &lt; .001</td>
<td>P &lt; .001</td>
<td>P &lt; .01</td>
<td>P &lt; .001</td>
<td>P &lt; .001</td>
<td>P &lt; .001</td>
<td>P &lt; .001</td>
<td>P &lt; .001</td>
</tr>
<tr>
<td>Group B</td>
<td>141.0 ± 18.62</td>
<td>161.73 ± 24.02</td>
<td>149.8 ± 21.9</td>
<td>158.36 ± 20.15</td>
<td>156.9 ± 20.1</td>
<td>153.7 ± 20.34</td>
<td>159.4 ± 21.9</td>
<td>124.2 ± 22.7</td>
</tr>
<tr>
<td>*P Value</td>
<td>P &lt; .001</td>
<td>P &lt; .001</td>
<td>P &lt; .01</td>
<td>P &lt; .001</td>
<td>P &lt; .001</td>
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<td>P &lt; .001</td>
<td>P &lt; .001</td>
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</tbody>
</table>
Table III: Changes in diastolic blood pressure in normotensive (Group A) and treated hypertensive patients (Group B).

<table>
<thead>
<tr>
<th>Group</th>
<th>Before Induction</th>
<th>After Induction</th>
<th>After Surgical Incision</th>
<th>Cystic Duct Ligation</th>
<th>Cystic Artery Ligation</th>
<th>Gall Bladder Removal</th>
<th>End of Operation</th>
<th>End of Anaesthesia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group A</td>
<td>89.0 ± 12.89</td>
<td>95.0 ± 12.41</td>
<td>97.4 ± 16.13</td>
<td>99.46 ± 15.7</td>
<td>100.6 ± 15.8</td>
<td>98.6 ± 16.9</td>
<td>106.6 ± 30.9</td>
<td>88.73 ± 10.2</td>
</tr>
<tr>
<td>*P Value</td>
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<td></td>
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</tr>
<tr>
<td>Group B</td>
<td>94.06 ± 10.69</td>
<td>101.9 ± 14.7</td>
<td>106.6 ± 15.23</td>
<td>112.4 ± 15.69</td>
<td>117.7 ± 20.1</td>
<td>108.7 ± 16.3</td>
<td>103.5 ± 9.8</td>
<td>95.06 ± 97.8</td>
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<tr>
<td>*P Value</td>
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Table IV: Changes in Mean Arterial pressure in normotensive (Group A) and treated hypertensive patients (Group B).

<table>
<thead>
<tr>
<th>Group</th>
<th>Before Induction</th>
<th>After Induction</th>
<th>After Surgical Incision</th>
<th>Cystic Duct Ligation</th>
<th>Cystic Artery Ligation</th>
<th>Gall Bladder Removal</th>
<th>End of Operation</th>
<th>End of Anaesthesia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group A</td>
<td>91.12 ± 6.21</td>
<td>98.2 ± 10.1</td>
<td>99.12 ± 13.12</td>
<td>102.13 ± 11.1</td>
<td>104.13 ± 12.0</td>
<td>104.13 ± 12.2</td>
<td>89.2 ± 10.2</td>
<td>94.1 ± 4.21</td>
</tr>
<tr>
<td>*P Value</td>
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<tr>
<td>Group B</td>
<td>97.23 ± 6.14</td>
<td>110.0 ± 7.13</td>
<td>110.23 ± 7.3</td>
<td>118.62 ± 7.23</td>
<td>117.62 ± 8.3</td>
<td>124.34 ± 8.2</td>
<td>108.13 ± 5.16</td>
<td>98.1 ± 4.2</td>
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<tr>
<td>*P Value</td>
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</table>

Table V: Changes in Rate Pressure Product in normotensive (Group A) and treated hypertensive patients (Group B).

<table>
<thead>
<tr>
<th>Group</th>
<th>Before Induction</th>
<th>After Induction</th>
<th>After Surgical Incision</th>
<th>Cystic Duct Ligation</th>
<th>Cystic Artery Ligation</th>
<th>Gall Bladder Removal</th>
<th>End of Operation</th>
<th>End of Anaesthesia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group A</td>
<td>10862.7 ± 7.9</td>
<td>13031.1 ± 11.0</td>
<td>10892.7 ± 7.9</td>
<td>14157.0 ± 10.1</td>
<td>14385.6 ± 13.5</td>
<td>14385.6 ± 9</td>
<td>10870.7 ± 6.0</td>
<td>10826.2 ± 8.6</td>
</tr>
<tr>
<td>*P Value</td>
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<tr>
<td>Group B</td>
<td>10324.2 ± 7.2</td>
<td>11640.9 ± 11.2</td>
<td>14310.3 ± 16.9</td>
<td>14516.1 ± 17.1</td>
<td>14795.1 ± 18.1</td>
<td>14841.2 ± 14.2</td>
<td>11016.0 ± 10.9</td>
<td>10589.1 ± 10</td>
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<tr>
<td>*P Value</td>
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</table>

Table VI: Electrocardiographic changes during cholecystectomy in normotensive (Group A) and treated hypertensive patients (Group B).

<table>
<thead>
<tr>
<th>Group</th>
<th>Before Induction</th>
<th>After Induction</th>
<th>After Surgical Incision</th>
<th>Cystic Duct Ligation</th>
<th>Cystic Artery Ligation</th>
<th>Gall Bladder Removal</th>
<th>End of Operation</th>
<th>End of Anaesthesia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group A</td>
<td>Regular</td>
<td>Regular</td>
<td>Regular</td>
<td>Regular</td>
<td>Regular</td>
<td>Regular</td>
<td>Regular</td>
<td>Regular</td>
</tr>
<tr>
<td>Group B</td>
<td>Regular</td>
<td>Irregular T-wave depression (1)</td>
<td>Irregular P-wave disappearance (1)</td>
<td>Irregular Extrasystole (3)</td>
<td>Irregular Extrasystole (2)</td>
<td>Irregular Extrasystole (4)</td>
<td>Irregular Bradycardia (1)</td>
<td>Regular</td>
</tr>
</tbody>
</table>
DISCUSSION
In the present study, patients' age ranged from 27-45 years (mean 36.33) in Group A and from 25-49 years (mean 35.6) in Group B. Male: Female ratio was 1:14 and 1:9 in Group A and Group B respectively. In this study we observed pulse rate, heart rhythm, systolic blood pressure, diastolic blood pressure, mean arterial pressure, and rate pressure product during different stages of anaesthesia and surgery in normotensive patients (Gp.A) and treated hypertensive (Gp.B) patients. The observation in both these groups for the various above mentioned parameters have been analyzed.

PULSE RATE
a) Induction of Anaesthesia
In our study it was observed that pulse rate was significantly increased after induction of anaesthesia in normotensive (7% p <0.01) and treated hypertensive patients (10% p<0.01). Our result was consistent with the findings of Esten and Li (1955) and Coleman et al. (1972).

Halter, P. Fling and Porte (1971), and Engquest et al (1980) suggested that surgical stimulation under general anaesthesia produced an increase in activity of sympathetic nervous system and rise in plasma concentration of circulating catecholamines that resulted in cardiovascular and metabolic changes. The cardiovascular stimulation produced rise in heart rate and hence increase in cardiac work. Our observations were in agreement with observation of the above workers.

b) Surgical Incision
We observed that surgical incision causes significant increase in pulse rate in normotensive (0.56% P<0.01) and treated hypertensive patients (12.08% p<0.01).

Child (1984) found significant rise in heart rate 15 minutes after skin incision in normotensive group but no changes in patients treated with labetalol group. Our observations were in accordance with observations of the above workers.

c) Biliary tract Manipulation
In our study there was significant rise in pulse rate in normotensive and treated hypertensive patients during cystic duct ligation (10.8%, 20.47% p<0.01) cystic artery ligation (11.3%, 14.9% p<0.01) and gall bladder removal. (11.7%, 15.9% p<0.001).

Bettman and Rubinfeld (1935) also observed an increase in heart rate due to the effects of pressure on the clamping and traction on the gall bladder. Seltzer et al (1985) also noted similar result.

d) End of Surgery
In our study pulse rate was significantly increased at the end of surgery in normotensive (8.76% p<0.01) and treated hypertensive groups (11.26% p<0.001). This observation in our study was in accordance with workers like Engquest (1971) who pointed out that surgical stimulation under anaesthesia was related to increased activity of the sympathetic nervous system and increased catecholamine level leading to increased heart rate and cardiac work.

e) End of Anaesthesia
There was slight increase of pulse rate after the recovery from anaesthesia in normotensive (4.9% p<0.001) and treated hypertensive groups (3.5% p<0.001). This can possibly be explained by the fact that when patient is reversed from anaesthesia, instrumentation in form of laryngoscopy, extubation and suctioning of the pharynx and larynx causes induced stress in the patient leading to increased catecholamine levels and thereby causing tachycardia. (Halter and Fling, 1971).

BLOOD PRESSURE
a) Induction of Anaesthesia
In our study there was significant increase in systolic blood pressure (8.71%, 15.2% p<0.001) and diastolic blood pressure (6.74%, 8.3% P<0.01) after induction of anaesthesia in normotensive and treated hypertensive patients. Esten and Li (1955) found that induction of anaesthesia with thiopentone sodium caused hypotension and decrease in cardiac output. Our observations were not consistent with observation of above workers.

b) Surgical Incision
In our study we observed significant increase in systolic blood pressure (8.78% p<0.001) 10.2%), p<0.001 and diastolic blood pressure (9.47%, 13.3 % p<0.00) during surgical incision. Our observations were in agreement with Halter, P. Fling and Porte (1971) and Engquest et. al. (1960) who found that surgical stimulation under general anaesthesia produces an increase in activity of sympathetic nervous system.

c) Biliary tract Manipulations
We also observed significant increase in systolic blood pressure (8.78%, 15.23% P. < 0.001) and diastolic blood pressure (2.01%, 11.9% P. < 0.001) during cystic duct ligation, cystic artery ligation (13.03%, 25.1% P < .001) and during gall bladder removal (2.03, 4.9% P. .001).

Heleem et. al (1990) reported an increase in systolic blood pressure and diastolic blood pressure during surgical, manipulation of gall bladder.

However, Seltzer et. al. (1985), Dripps RD, Ecken hoff, Vandam (1982), Strumin (1980) Kanfman (1980), and Batchelder BM, (1987) reported decrease in systemic blood pressure during traction on abdominal mesentry. They suggested that mesentric traction causes an apparent sympathetic stimulation that results in vasodilation of splanchnic system and venous pooling in splanchnic capacitance vessels. This splanchnic pooling...
is assumed to result in decrease venous return to the heart and decrease in stroke volume and cardiac output.

d) End of Operation
In our study there was less significant increase in systolic blood pressure (2.01%, 10.9%, P. < .01) and diastolic blood pressure (2.03%, 4.9%, P. < .001) at the end of surgery in normotensive and treated hypertensive patients. However these blood pressure changes come to preinduction level after the reversal of anaesthesia in normotensive and treated hypertensive patients. This increase in systolic and diastolic blood pressure can be explained by comparing the study of Haleem et al 1990 where an increase in systolic and diastolic blood pressure was noted and values returned to pre induction values after end of operative procedure.

Mean Arterial Pressure

a) Induction Of Anaesthesia And Surgical Incision
In our study there was significant increase in mean arterial pressure after induction of anaesthesia (7.1%, 13.13%, P. < .001) and during surgical incision (0.9%, 13.3%, P. < .001) in normotensive and treated hypertensive patients. Our results are consistent with Child (1984). In our study increase in mean arterial pressure after skin incision can be explained by the fact that surgical stimulus resulting in pain in early anaesthetic period causes increase in sympathetic activity which leads to increase in systolic and diastolic pressure thereby increasing the mean arterial pressure.

b) Biliary Tract Manipulation
In our study it was found that mean arterial pressure increased significantly during cystic duct ligation, (12.1%, 23.4% P. <.001) cystic artery ligation (14.27%, 20.9%, P.<.001) and during gall bladder removal (14.18%, 27.7%). Haleem et. al. (1990) reported increase in mean arterial pressure during surgical manipulation of gall blader. Westerband et. al. (1992) also observed 15% increase in mean arterial pressure in patients undergoing laproscopic cholecystectomy during surgical manipulation.

c) End Of Surgery
There was less significant increase in Mean arterial pressure at the end of surgery in normotensive (2.01%, P.<.001) and treated hypertensive (11.1%, P.<.001) patients at the end of surgery. These changes came to pre-induction level after reversal of anaesthesia. Our results were in consistent with study of Haleem et al 1990.

Rate Pressure Product

a) Induction Of Anaesthesia
In our study the rate pressure product was significantly increased after induction of anaesthesia in normotensive group (19.96%, P.<.001) and treated hypertensive groups (12.47% P.<.001) of patients. Increase in rate pressure product is obvious as the sympathetic stimulation causes rise in both heart rate and systolic blood pressure (Halter P. Fling 1971).

b) Surgical Incision
After surgical incision rate pressure product also increased in normotensive (1.08%, P.<.001) and treated hypertensive patients (10.9%, P.<.01). This may be because the effect of increase in sympathetic activity is greater than that of infusion of adrenaline and it is accompanied by release of noradrenaline and also the potency of labetalol at alpha receptor is less than that at beta receptor.

c) Biliary Tract Manipulations
In our study rate pressure product was also increased during cystic duct ligation (24.71%, 30.3%, P.<.001) cystic artery ligation (27.09%, 32.43%, P.<.001) and gall bladder removal (27.4%, 32,01%, P.< .001) in normotensive and treated hypertensive patients.

Child in (1984) suggested that labetalol did prevent the rise in rate pressure product which occurred in response to surgical stimulation and this may be useful in patients with Ischemic heart disease or hypertension. Our observations conformed to the observations of Child (1984).

d) End of Surgery
At the end of surgery the rate pressure product was increased in normotensive group (.71%, P.<.001) and in treated hypertensive patients (5.36%, P.<.001). Rate pressure product comes to preinduction level in normortensive group of patients (.33%, P. <.001) while it was significantly increased in treated hypertensive patients (9.03%, P.<.01) after recovery from anaesthesia. This study is in accordance with the study of Haleem et al 1990 who found similar result.

Common Bile duct Exploration
Pulse rate was significantly increased during common bile duct exploration in normotensive (12.21%, P.<.001) and treated hypertensive patients (8.89%, P.<.001). Systolic blood pressure, (5.2%, 6.02%, P.<.001) diastolic blood pressure (7.3%, 9.6%, P.<0.01), mean arterial pressure (14.3%, 35.1%, P.<.001) and rate pressure product (2.92%, and 9.7%, P.<.001) were also found to be increased during common bile duct exploration.

However, Buchbinder, 1930 and Scott and Ivy, 1932 suggested that incision of gall bladder with spillage of bile into upper peritoneal cavity causes reflex inhibition of heart. Carlson in (1930) found the same type of stimulation in the turtle causes acceleration of heart. Scott and Ivy (1923), Schrager (1928) found that distension of gall bladder may cause either inhibition or acceleration of heart rate. The observations of above authors were in agreement with our observation.
Electro-cardiographic changes

In our study electro-cardiographic changes were observed in normotensive and treated hypertensive patients. In our study no electro-cardiographic changes were observed in normotensive patients during surgical manipulation of gall bladder. But in treated hypertensive patients, one patient developed T-wave depression after induction of anaesthesia while other patient develops P-wave disappearance after surgical incision.

Our findings of present study were in agreement with those reported by other workers. Bettman and Rubinfeld in 1935 also reported that three patients developed Cardiac irregularities during gall bladder surgery. 2 patients developed ectopic beat and one sinus arrhythmia. Two of the patients developed ventricular extra systole, 1 during ligation and cutting of cystic duct and artery, the other during pressure on gall bladder. They suggested that these changes were due to reflex effects on the heart from operative manipulation. Mendelson and Monheit (1956) found electrocardiographic changes in six patients out of 20 normotensive patients during gall bladder manipulation. In 30 patients with cardiac disease pre-operatively electrocardiographic changes were developed in 15 patients during surgical manipulations. They found that changes bore a temporal relationship to surgical manipulation which suggested that they were reflex in origin.

Cady et. al., 1948, Owen 1933, Greene 1975, Gilbert Mc 1942, Winbury MM, and Green 1952 found that reflexes arising from biliary tract can give rise to cardiac arrhythmias and reduction in coronary blood flow that leads to catastrophic changes during biliary tract surgery.

CONCLUSION

It was concluded from the present study that.

1. Pulse rate rises significantly during induction and surgical manipulation of gall bladder, at the end of operation and anaesthesia the values returned to pre induction level.
2. Systolic Blood pressure, during induction and surgical manipulations increases significantly and the values comes to pre-induction levels at end of anaesthesia.
3. Diastolic blood pressure increases significantly during induction and surgical manipulation of gall bladder and the values come back to normal levels at the end of anaesthesia.
4. Mean arterial pressure increases significantly during surgical manipulation and at the end of operation the values come back to pre-induction level.
5. Rate pressure product also increases significantly during surgical manipulation and at the end of anaesthesia, the values come back to pre-induction level.
6. ECG changes develop in treated hypertensive patients during surgical manipulation and at the end of operation these changes come back to normal levels.

The present study confirmed that hemodynamic disturbances secondary to diseased gall bladder and its removal are more marked when cardiovascular system is the seat of proclaimed or latent disorder.

REFERENCES