CHAPTER-V

SECTION - A

HAEMATOLOGY
The mallards exposed to mining effluents (M.E.) showed various alterations in the concentrations of haemoglobin, RBC Counts, WBC counts, Differential counts, blood platelet counts, Erythroyte sedimentation rates and in addition brought about significant changes in the serum constituents levels depending upon the concentrations of mining effluents and the durations of exposures to M.E.

I) ALTERATIONS IN HAEMOGLOBIN:

The changes in the haemoglobin (Hb) concentrations of blood under the influence of mining effluents (M.E.) at various time intervals are given in Table No.24.0 and are graphically presented in Graph No. 24. The Hb concentrations are expressed as grams percent. The control animals showed Hb concentration equivalent to 15.3 ± 1.2 grams percent. The 100% M.E. induced maximum reduction in Hb concentration at the end of twenty-four hours. All the concentrations of the mining effluents promoted reduction in the haemoglobin concentration.

The ducks exposed to 0.01% M.E. did not show any significant change in the Hb level up to the end of twenty-nine days but at the end of thirty days (seven hundred and twenty hours), the Hb concentration dropped to 12.54 ± 0.272 grams and at the end of forty-five days (one thousand and eighty hours) it further reduced to 11.94 ± 0.135 grams percent.

On exposure to 0.1% M.E. the ducks showed fluctuations in the Hb concentrations. By the end of six, twelve, twenty-four, forty-eight, seventy-two, ninetysix and one twenty hours, the Hb concentrations were equivalent to 11.0 ± 0.583, 13.02 ± 0.213, 10.60 ± 0.389, 13.96 ± 0.205, 12.0 ± 0.141, 12.20 ± 0.424 and 11.80 ± 0.141 grams respectively.

Under the influence of 1.0% M.E. the Hb concentrations of the ducks fluctuated between 11.26 gms to 15.02 gms. The Hb concentrations observed at the end of six, twelve, twenty-four, forty-eight, seventy-two, ninetysix and one
twenty hours were equivalent to 15.02 ± 0.354, 11.98 ± 0.132, 11.26 ± 0.326, 12.0 ± 0.282, 13.98 ± 0.213, 14.02 ± 0.312 and 12.12 ± 0.348 grams respectively.

The ducks subjected to 5% M.E. showed reduction in Hb concentrations at all the time intervals under study but, the reduction was not uniform. The Hb concentrations of blood were equivalent to 13.02 ± 0.481, 13.14 ± 0.417, 12.46 ± 0.361, 11.26 ± 0.338, 11.60 ± 0.393, 14.04 ± 0.287 and 11.44 ± 0.344 gram percent at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix, and one hundred and twenty hours respectively.

The 10.0% M.E. induced decrease in the Hb concentrations of the mallards for all the time intervals under study and the degree of decrement was neither M.E. concentration dependent nor exposure time dependent. At the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix, one hundred and twenty hours, the Hb concentrations of the blood were equivalent to 11.40 ± 0.236, 12.02 ± 0.16, 12.0 ± 0.616, 11.92 ± 0.172, ± 12.44 ± 0.32, 14.16 ± 0.392 and 12.96 ± 0.241 grams percent respectively.

The exposure to 50.0% M.E. promoted decrease in the Hb concentration but the decrement did not reflect any relevance to the high M.E. concentration. By the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix, one twenty hours, the Hb concentrations of the blood were equivalent to 13.98 ± 0.132, 13.60 ± 0.346, 13.10 ± 0.678, 14.0 ± 0.141, 13.36 ± 0.50, 12.36 ± 0.257 and 12.34 ± 0.265 gram percent respectively.

The 100% M.E. caused maximum decrease in the Hb concentration at the end of twentyfour hours but Hb concentration increased a little subsequently through fortyeight and seventytwo hours. Thus, the Hb concentrations of the ducks were equivalent to 14.46 ± 0.205, 13.90 ± 0.340, 9.98 ± 0.494, 12.0 ± 0.141, 12.50 ± 0.109, 11.84 ± 0.162 and 12.60 ± 0.303 grams percent at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one hundred and twenty hours respectively.
TABLE NO. 24.0 : ALTERATIONS IN HAEMOGLOBIN CONCENTRATIONS OF DUCKS EXPOSED TO MINING EFFLUENTS.

<table>
<thead>
<tr>
<th></th>
<th>6 Hours</th>
<th>12 Hours</th>
<th>24 Hours</th>
<th>48 Hours</th>
<th>72 Hours</th>
<th>96 Hours</th>
<th>120 Hours</th>
<th>720 Hours</th>
<th>1080 Hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>15.30 ±1.20</td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>0.01% M.E.</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>12.54 ±0.2727</td>
</tr>
<tr>
<td>0.1% M.E.</td>
<td>11.0 ±0.583</td>
<td>13.02 ±0.213</td>
<td>10.60 ±0.389</td>
<td>13.96 ±0.205</td>
<td>12.0 ±0.141</td>
<td>12.20 ±0.424</td>
<td>11.80 ±0.141</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td>1% M.E.</td>
<td>15.02 ±0.354</td>
<td>11.98 ±0.132</td>
<td>11.26 ±0.326</td>
<td>12.0 ±0.282</td>
<td>13.98 ±0.213</td>
<td>14.02 ±0.312</td>
<td>12.12 ±0.348</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td>5% M.E.</td>
<td>13.02 ±0.481</td>
<td>13.14 ±0.417</td>
<td>12.46 ±0.361</td>
<td>11.26 ±0.338</td>
<td>11.60 ±0.393</td>
<td>14.04 ±0.287</td>
<td>11.44 ±0.344</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td>10% M.E.</td>
<td>11.40 ±0.236</td>
<td>12.02 ±0.16</td>
<td>12.0 ±0.616</td>
<td>11.92 ±0.172</td>
<td>12.44 ±0.32</td>
<td>14.16 ±0.392</td>
<td>12.96 ±0.241</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td>50% M.E.</td>
<td>13.98 ±0.132</td>
<td>13.6 ±0.346</td>
<td>13.10 ±0.678</td>
<td>14.0 ±0.141</td>
<td>13.36 ±0.500</td>
<td>12.36 ±0.257</td>
<td>12.34 ±0.265</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td>100% M.E.</td>
<td>14.46 ±0.205</td>
<td>13.90 ±0.340</td>
<td>9.98 ±0.494</td>
<td>12.0 ±0.141</td>
<td>12.50 ±0.109</td>
<td>11.84 ±0.162</td>
<td>12.60 ±0.303</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
</tbody>
</table>

Note: Unit: Haemoglobin in gms %.
N.D.: Not Determined.

All alterations are statistically significant - P < 0.01.
GRAPH NO. 24.0: ALTERATIONS IN HAEMOGLOBIN CONCENTRATIONS OF DUCKS EXPOSED TO MINING EFFLUENTS.

Units: gms %.

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Controls: 0 6 12 24 48 72 96 120 720 1080 Hours
II] ALTERATION IN RBC. COUNTS:

The Changes in the total red blood corpuscles (RBC) counts are compiled in Table No.25 and Graphically presented in Graph No. 25. The RBC counts are expressed as millions per cubic millimeter. The control ducks showed the RBC count equivalent to 5.3 ± 0.017 millions per cubic millimeter.

From the table and graph it appears that under the influence of mining effluents (M.E.) the ducks exhibited reduction in erythropoises as evidenced by the reduced total RBC counts. But the M.E. induced fluctuations in the RBC counts, though the total RBC count was less than that observed in the Control. Under the influence of only 5.0 % M.E. at the end of ninetysix hours a significant recovery in the total RBC count was observed. The 100% M.E. induced the maximum reduction in the total RBC count at the end of twentyfour hours only.

When the ducks were exposed to 0.01% M.E. there was no significant change in the total RBC counts up to the end of twentynine days but at the end of sevenhundred and twenty hours (thirty days) there was reduction in the total RBC count which continued to decrease up to the end of fortyfive days (one thousand and eighty hours). By the end of seven twenty and onethousand and eighty hours the total RBC counts were equivalent to 4.304 ± 0.010 and 3.948 ± 0.043 millions per cubic millimeter respectively.

The ducks subjected to 0.1% M.E. exhibited sharp decrease in the total RBC counts at the end of six and twenty four hours.

The total RBC count was equivalent to 3.688 ± 0.139, 4.28 ± 0.102, 3.59 ± 0.285, 4.626 ± 0.053, 4.016 ± 0.049, 4.16 ± 0.115 and 4.024 ± 0.050 millions per cubic millimeter at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours respectively.
The 1.0% M.E. induced pronounced reductions in the total RBC counts at the end of twelve and twentyfour hours. At the end of six, twelve, twentyfour, forty eight, seventytwo, ninetysix and one twenty hours the total RBC count was equivalent to 4.92 ± 0.136, 3.924 ± 0.149, 3.614 ± 0.270, 4.05 ± 0.157, 4.232 ± 0.154, 4.76 ± 0.086 and 4.008 ± 0.109 millions per cubic millimeter respectively.

Under the influence of 5.0% M.E. the marked decrease and recovery in the total RBC count was observed at the end of seventytwo hours and ninetysix hours respectively. But at the end of one twenty hours there was once again sharp decrease in the total RBC count. The total RBC count at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours was equivalent to 4.36 ± 0.115, 4.326 ± 0.243, 4.19 ± 0.185, 4.526 ± 0.060, 3.776 ± 0.053, 5.20 ± 0.070 and 3.948 ± 0.038 millions per cubic millimeter respectively.

The ducks exposed to 10.0% M.E. showed profound decrease in the total RBC count at the end of six hours and a significant recovery in the total RBC count was observed at the end of ninetysix hours. By the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours the total RBC count was equivalent to 3.904 ± 0.034, 4.008 ± 0.038, 3.962 ± 0.035, 3.98 ± 0.035, 4.38 ± 0.029, 4.88 ± 0.213 and 4.04 ± 0.185 millions per cubic millimeter respectively.

The exposure of ducks to 50% M.E. promoted decrease in the total number of erythrocytes and though the erythrocyte count was low it fluctuated between 4.2 and 4.69 millions per cubic millimeter. By the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours the total RBC count was equivalent to 4.69 ± 0.037, 4.578 ± 0.054, 4.664 ± 0.037, 4.632 ± 0.029, 4.62 ± 0.031, 4.30 ± 0.013 and 4.20 ± 0.059 millions per cubic millimeter respectively.
The ducks subjected to 100% M.E. showed pronounced decrease in the total number of erythrocytes at the end of twentyfour hours. The total RBC count at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours was equivalent to $4.346 \pm 0.020$, $4.28 \pm 0.016$, $3.118 \pm 0.013$, $4.158 \pm 0.035$, $4.416 \pm 0.010$, $3.55 \pm 0.040$ and $4.184 \pm 0.010$ millions per cubic millimeter respectively.
**TABLE NO: 25: THE ALTERATIONS IN TOTAL R.B.C COUNTS UNDER THE INFLUENCE OF MINING EFFLUENTS.**

<table>
<thead>
<tr>
<th></th>
<th></th>
<th>6 Hours</th>
<th>12 Hours</th>
<th>24 Hours</th>
<th>48 Hours</th>
<th>72 Hours</th>
<th>96 Hours</th>
<th>120 Hours</th>
<th>720 Hours</th>
<th>1080 Hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td></td>
<td>5.30</td>
<td>± 0.017</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.01%</td>
<td>M.E.</td>
<td>3.688</td>
<td>±0.139</td>
<td>4.28</td>
<td>±0.102</td>
<td>3.59</td>
<td>±0.285</td>
<td>4.626</td>
<td>±0.053</td>
<td>4.016</td>
</tr>
<tr>
<td>0.1%</td>
<td>M.E.</td>
<td>4.92</td>
<td>±0.136</td>
<td>3.924</td>
<td>±0.149</td>
<td>3.614</td>
<td>±0.270</td>
<td>4.05</td>
<td>±0.157</td>
<td>4.232</td>
</tr>
<tr>
<td>1%</td>
<td>M.E.</td>
<td>4.36</td>
<td>±0.115</td>
<td>4.326</td>
<td>±0.243</td>
<td>4.19</td>
<td>±0.185</td>
<td>4.526</td>
<td>±0.060</td>
<td>3.776</td>
</tr>
<tr>
<td>5%</td>
<td>M.E.</td>
<td>3.904</td>
<td>±0.034</td>
<td>4.008</td>
<td>±0.038</td>
<td>3.962</td>
<td>±0.035</td>
<td>3.98</td>
<td>±0.035</td>
<td>4.38</td>
</tr>
<tr>
<td>10%</td>
<td>M.E.</td>
<td>4.69</td>
<td>±0.037</td>
<td>4.578</td>
<td>±0.054</td>
<td>4.664</td>
<td>±0.037</td>
<td>4.632</td>
<td>±0.029</td>
<td>4.62</td>
</tr>
<tr>
<td>50%</td>
<td>M.E.</td>
<td>4.346</td>
<td>±0.020</td>
<td>4.28</td>
<td>±0.016</td>
<td>3.118</td>
<td>±0.013</td>
<td>4.158</td>
<td>±0.035</td>
<td>4.416</td>
</tr>
<tr>
<td>100%</td>
<td>M.E.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

**Note.:** Unit : Million /mm$^3$.

N.D. : Not Determined.

All alterations are statistically significant - $P < 0.01$. 

**Unit - Million /mm$^3$.**
GRAPH NO. 25: THE ALTERATIONS IN TOTAL RBC COUNT UNDER THE INFLUENCE OF MINING EFFLUENTS.

Units: million/mm³.
III) ALTERATIONS IN WBC COUNTS:

The alterations in the total leucocytes (WBC) counts are given in Table No.26 and are graphically presented in Graph No. 26. The total WBC counts are expressed as the number per cubic millimeter. The control animals had total leucocyte count equivalent to 6800 ± 20.0 cells per cubic millimeter. From the table and the Graph it appears that the mining effluents (M.E.) induced increase in the number of leuocytes but the increase was neither M.E. concentration dependent nor exposure period dependent. The maximum increase in the total number of leucocytes was observed at the end of twentyfour hours under the influence of 50 percent mining effluents (M.E.). Though, the M.E. induced increase in leucocyte numbers over the normal range, there were wide fluctuations in the WBC counts for different time intervals.

When the ducks were expsoed to 0.01% M.E. there were no significant changes in the total leucocyte counts till the end of twentynine days. But at the end of seven hundred and twenty hours (thirty days) there was sharp increase in the leucocyte numbers which increased very marginally (nonsignificantly) at the end of fortyfive days (one thousand and eighty hours). Thus at the end of seven hundred and twenty hours and one thousand and eighty hours the total WBC count was equivalent to 9213 ± 31.24 and 9268 ± 34.81 cells per cubic millimeter.

On exposure to 0.1% M.E., the total WBC count of ducks raised to a high level at the end of ninetysix hours and then dropped subsequently. But in general the 0.1% M.E. induced increase in the number of leucocytes. Thus, the total WBC count at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours was equivalent to 10309.0 ± 35.06, 10251.0 ± 53.64, 11805.0 ± 16.41, 9199.0 ± 15.09, 9211.0 ± 43.08, 12904.0 ± 31.5 and 8927.0 ± 40.05 cells per cubic millimeter respectively.
The ducks treated with 1.0% M.E. showed fluctuations in the total WBC count for various time intervals under study and at the end of twelve hours exposure, there was a pronounced increase in the number of leucocytes. Thus, at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours, the total WBC count was equivalent to 9197.0 ± 12.70, 12301.0 ± 12.56, 10323.0 ± 29.75, 10310.0 ± 28.47, 9924.0 ± 36.77, 9424.0 ± 26.747 and 8598.0 ± 19.27 cells per cubic millimeter respectively.

The animals subjected to 5.0% M.E. exhibited significant increase in the total WBC count at all the time intervals in comparison with that observed in the controls. By the end of six and twelve hours there was prominent increase in the total number of leucocytes and then the leucocyte numbers exhibited considerable fluctuations. Thus, at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and onetwenty hours, the total WBC count was equivalent to 10599.0 ± 16.49, 10800 ± 42.85, 9205 ± 18.75, 10147 ± 32.13, 9841 ± 48.10, 8614 ± 31.90 and 9219 ± 32.25 cells per cubic millimeter respectively.

The exposure to 10.0% M.E. promoted sharp increase in the leucocyte number and the leucocyte population remained over eleven thousand from the end of six hours to the end of fortyeight hours. The total W.B.C. count at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours was equivalent to 11605.0 ± 15.5, 11828.0 ± 30.8, 11916.0 ± 33.5, 11225.0 ± 29.74, 10640.0 ± 81.51, 10617.0 ± 31.53 and 9200.0 ± 26.40 cells per cubic millimeter respectively.

Under the influence of 50.0% M.E., the ducks exhibited the highest number of leucocytes at the end of twentyfour hours and the leucocyte population remained over eleven thousand for all the time intervals under study. Thus, at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one hundred and twenty hours the leucocyte count was
equivalent to 11219.0 ± 36.80, 12909.0 ± 29.11, 20868.0 ± 42.91, 14617.0 ± 38.48, 11719.0 ± 37.90, 15218.0 ± 37.83 and 15623.0 ± 33.60 cells per cubic millimeter respectively.

The 100% M.E. induced increase in leucocyte population but the increase was not as high as found under the influence of 50% M.E. Thus, at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one hundred and twenty hours, the total W.B.C. count was equivalent to 9836.0 ± 182.16, 9868.0 ± 39.043, 10752.0 ± 39.69, 10887.0 ± 45.305, 8800.0 ± 316.2, 10694.0 ± 54.921 and 11914.0 ± 71.03 cells per cubic millimeter respectively.
### TABLE NO. 26: THE CHANGES IN TOTAL WBC COUNTS UNDER THE INFLUENCE OF MINING EFFLUENTS.

<table>
<thead>
<tr>
<th></th>
<th>6 Hours</th>
<th>12 Hours</th>
<th>24 Hours</th>
<th>48 Hours</th>
<th>72 Hours</th>
<th>96 Hours</th>
<th>120 Hours</th>
<th>720 Hours</th>
<th>1080 Hours</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Control</strong></td>
<td>6800 ± 20</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>0.01% M.E.</strong></td>
<td>10309 ± 35.06</td>
<td>10251 ± 53.64</td>
<td>11805 ± 16.41</td>
<td>9199 ± 15.09</td>
<td>9211 ± 43.08</td>
<td>12904 ± 31.5</td>
<td>8927 ± 40.05</td>
<td>9213 ± 31.24</td>
<td>9268 ± 34.81</td>
</tr>
<tr>
<td><strong>0.1% M.E.</strong></td>
<td>9197 ± 12.70</td>
<td>12301 ± 12.56</td>
<td>10323 ± 29.75</td>
<td>10310 ± 28.47</td>
<td>9924 ± 36.77</td>
<td>9424 ± 26.747</td>
<td>8598 ± 19.27</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td><strong>1% M.E.</strong></td>
<td>10599 ± 16.49</td>
<td>10800 ± 42.85</td>
<td>9205 ± 18.75</td>
<td>10147 ± 32.13</td>
<td>9841 ± 48.10</td>
<td>8614 ± 31.90</td>
<td>9219 ± 32.25</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td><strong>5% M.E.</strong></td>
<td>11605 ± 15.5</td>
<td>11828 ± 30.8</td>
<td>11916 ± 33.5</td>
<td>11225 ± 29.74</td>
<td>10640 ± 81.51</td>
<td>10617 ± 31.53</td>
<td>9200 ± 26.40</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td><strong>10% M.E.</strong></td>
<td>11219 ± 36.80</td>
<td>12909 ± 29.110</td>
<td>20868 ± 42.91</td>
<td>14617 ± 38.48</td>
<td>11719 ± 37.90</td>
<td>15218 ± 37.83</td>
<td>15623 ± 33.60</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td><strong>50% M.E.</strong></td>
<td>9836 ± 182.16</td>
<td>9868 ± 39.043</td>
<td>10752 ± 39.69</td>
<td>10887 ± 45.305</td>
<td>8800 ± 316.2</td>
<td>10694 ± 54.921</td>
<td>11914 ± 71.03</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td><strong>100% M.E.</strong></td>
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</tr>
</tbody>
</table>

**Note:** Unit: WBC Count /mm³.

N.D. : Not Determined.

All alterations are statistically significant - P < 0.01.
GRAPH NO. 26: THE CHANGES IN TOTAL LEUCOCYTE COUNT UNDER THE INFLUENCE OF MINING EFFLUENTS.
Units: Total WBC / mm³
IV) ALTERATIONS IN THE LEUCOCYTE DIFFERENTIAL COUNT:

The alterations in the differential counts of leucocytes are given in Table No. 27 and are graphically presented in Graph No. 27.1, 27.2 and 27.3. The differential count envisages the occurrences of different types of leucocytes in specific percentages. Normally in the animals blood the leucocytes such as neutrophils, lymphocytes, eosinophils, basophils and monocytes are found in specific ranges. The occurrences of these individual leucocyte types are expressed as percents of total number of leucocytes. The control birds exhibited the presence of neutrophils, lymphocytes and eosinophils while the basophils and monocytes were absent. The proportions of neutrophils, lymphocytes and eosinophils were equivalent to 38.0 ± 2.08%, 60.0 ± 2.0% and 2.0 ± 0.87% respectively.

A) THE CHANGES IN NEUTROPHIL COUNT:

The exposure to 0.01% M.E. did not produce any significant change in the neutrophil count up to the end of twenty-nine days but at the end of thirty days (seven hundred & twenty hours) there was significant increase in the neutrophil count which further increased by the end of forty-five days (one thousand and eighty hours). Thus, at the end of thirty and forty-five days exposure to 0.01% M.E., the neutrophil percentage was equivalent to 50.0 ± 1.72 and 60.0 ± 2.5 percent respectively.

The 0.1% M.E. induced fluctuations in the neutrophil counts and the maximum increase in the neutrophil count was observed at the end of forty-eight hours. Thus, at the end of six, twelve, twenty-four, forty-eight, seventy-two, ninetysix and one twenty hours the neutrophil count was equivalent to 41.0 ± 2.4, 53.0 ± 2.5, 40.0 ± 2.05, 69.0 ± 2.4, 60.0 ± 1.7, 40.0 ± 2.1 and 44.0 ± 3.1 percent respectively.
Under the influence of 1.0% M.E. there was significant reduction in the neutrophil count at the end of twelve and ninetysix hours while at the end of seventytwo hours there was sharp rise in the neutrophil number. Thus, at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours the neutrophil count was equivalent to 45.0 ± 2.6, 31.0 ± 1.7, 50.0 ± 3.6, 42.0 ± 2.1, 55.0 ± 2.6, 32.0 ± 3.7 and 47.0 ± 2.1 percent respectively.

The animals subjected to 5.0% M.E. showed increase in the neutrophil percentage at the end of six hours and the neutrophil count went on increasing up to the end of twelve hours and subsequently the neutrophil count dropped gradually for a period from fortyeight hours to ninetysix hours. Then, once again the neutrophil count rose prominently. By the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours, the neutrophil count was equivalent to 54.0 ± 3.2, 61.0 ± 1.62, 48.0 ± 1.01, 41.0 ± 2.4, 40.0 ± 3.8, 40.0 ± 2.1 and 59.0 ± 1.8 percent respectively.

The exposure of birds to 10% M.E. induced successive rise in the neutrophil count from the end of twentyfour hours to onetwenty hours. Initially at the end of six and twelve hours exposure, the bird showed marginal increase in the neutrophil count.

Thus, at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours the neutrophil count was equivalent to 40.0 ± 2.0, 42.0 ± 2.13, 39.0 ± 1.72, 40.0 ± 2.13, 48.0 ± 2.6, 55.0 ± 2.6 and 64.0 ± 2.87 percent respectively.

On exposure to 50.0% M.E., the ducks exhibited sharp decrease in the neutrophil count at the end of twentyfour and fortyeight hours. The neutrophil count remained low up to seventytwo hours and then increased at the end of ninetysix hours. Thus, at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and onetwenty hours the neutrophil count was equivalent
to 33.0 ± 2.607, 35.0 ± 2.82, 22.0 ± 3.26, 22.0 ± 2.99, 25.0 ± 3.1, 45.0 ± 3.4, and 40.0 ± 1.95 percent respectively.

The 100% M.E. promoted maximum increase in the neutrophil count at the end of one twenty hours and at the end of seventytwo hours the neutrophil count was marginally lower than that observed in the control animals. Thus, at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours, the neutrophil count was equivalent to 46.0 ± 1.32, 43.0 ± 1.326, 46.0 ± 2.28, 43.0 ± 2.56, 37.0 ± 1.624, 39.0 ± 2.1 and 71.0 ± 1.78 percent respectively.
### Table No. 27.0: Alterations in the Neutrophil Counts Under the Influence of Mining Effluents.

<table>
<thead>
<tr>
<th></th>
<th>6 Hours</th>
<th>12 Hours</th>
<th>24 Hours</th>
<th>48 Hours</th>
<th>72 Hours</th>
<th>96 Hours</th>
<th>120 Hours</th>
<th>720 Hours</th>
<th>1080 Hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>M.E.</td>
<td>38% (\pm 2.08)</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>0.01%</td>
<td>M.E.</td>
<td>41% (\pm 2.4)</td>
<td>53% (\pm 2.5)</td>
<td>40% (\pm 2.05)</td>
<td>69% (\pm 2.4)</td>
<td>60% (\pm 1.7)</td>
<td>40% (\pm 2.1)</td>
<td>44% (\pm 3.1)</td>
<td>50% (\pm 1.72)</td>
</tr>
<tr>
<td>0.1%</td>
<td>M.E.</td>
<td>45% (\pm 2.6)</td>
<td>31% (\pm 1.7)</td>
<td>50% (\pm 3.6)</td>
<td>42% (\pm 2.1)</td>
<td>55% (\pm 2.6)</td>
<td>32% (\pm 3.7)</td>
<td>47% (\pm 2.1)</td>
<td>N.D. (N.D.)</td>
</tr>
<tr>
<td>1%</td>
<td>M.E.</td>
<td>45% (\pm 3.2)</td>
<td>61% (\pm 1.62)</td>
<td>48% (\pm 1.01)</td>
<td>41% (\pm 2.4)</td>
<td>40% (\pm 3.8)</td>
<td>40% (\pm 2.1)</td>
<td>59% (\pm 1.8)</td>
<td>N.D. (N.D.)</td>
</tr>
<tr>
<td>5%</td>
<td>M.E.</td>
<td>40% (\pm 2.0)</td>
<td>42% (\pm 2.13)</td>
<td>39% (\pm 1.72)</td>
<td>40% (\pm 2.13)</td>
<td>48% (\pm 2.6)</td>
<td>40% (\pm 2.6)</td>
<td>55% (\pm 2.87)</td>
<td>N.D. (N.D.)</td>
</tr>
<tr>
<td>10%</td>
<td>M.E.</td>
<td>33% (\pm 2.607)</td>
<td>35% (\pm 2.82)</td>
<td>22% (\pm 3.26)</td>
<td>22% (\pm 2.99)</td>
<td>25% (\pm 3.1)</td>
<td>45% (\pm 3.4)</td>
<td>40% (\pm 1.95)</td>
<td>N.D. (N.D.)</td>
</tr>
<tr>
<td>50%</td>
<td>M.E.</td>
<td>46% (\pm 1.32)</td>
<td>43% (\pm 1.326)</td>
<td>46% (\pm 2.28)</td>
<td>43% (\pm 2.56)</td>
<td>37% (\pm 1.624)</td>
<td>39% (\pm 2.1)</td>
<td>71% (\pm 1.78)</td>
<td>N.D. (N.D.)</td>
</tr>
<tr>
<td>100%</td>
<td>M.E.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

Note: Unit: Neutrophils in %.
N.D.: Not Determined.

All alterations are statistically significant \(- P < 0.01\).
GRAPH NO. 27: ALTERATIONS IN THE NEUTROPHIL COUNT UNDER THE INFLUENCE OF MINING EFFLUENTS.
Units: Neutrophils in %
B) THE CHANGES IN LYMPHOCYTE COUNT:

The control animals had sixty percent lymphocytes in the blood among the leucocytes. The exposure of ducks to 0.01% M.E. could not alter the lymphocyte count up to the end of twenty-nine days but at the end of thirty days the lymphocyte count reduced significantly and kept on decreasing up to the end of forty-five days. Thus, at the end of thirty days (seven hundred and twenty hours) and forty-five days (one thousand and eighty hours), the lymphocyte count was equivalent to 41.0 ± 2.2 and 32.0 ± 1.4 percent respectively.

Under the influence of 0.1% M.E., there was, in general reduction in the lymphocyte count. The acute reduction in the lymphocyte number was observed at the end of twelve hours and then the lymphocyte count increased gradually from twenty-four hours to ninety-six hours but it remained below that observed in control bird. Thus, at the end of six, twelve, twenty-four, forty-eight, seventy-two, ninety-six, and one-twenty hours, the lymphocyte count was equivalent to 32.0 ± 2.1, 25.0 ± 2.6, 27.0 ± 1.9, 30.0 ± 1.7, 30.0 ± 1.6, 43.0 ± 3.6 and 34.0 ± 2.4 percent respectively.

The exposure to 1.0% M.E. caused decrease in lymphocyte count. The relatively high decrease in lymphocyte count was observed at the end of seventy-two hours and then it recorded significant elevation at the end of ninety-six hours. At the end of six, twelve, twenty-four, forty-eight, seventy-two, ninety-six and one-twenty hours, the lymphocyte count was equivalent to 42.0 ± 2.5, 38.0 ± 1.6, 34.0 ± 3.3, 41.0 ± 1.8, 32.0 ± 1.8, 56.0 ± 2.4 and 44.0 ± 2.8 percent respectively.

The 5.0% M.E. promoted acute decline in lymphocyte number at the end of twelve hours and a significant recovery in the lymphocyte percentage was observed at the end of ninety-six hours. By the end of six, twelve, twenty-four, forty-eight, seventy-two, ninety-six, and one-twenty hours the lymphocyte count
was equivalent to 29.0 ± 2.7, 27.0 ± 1.41, 40.0 ± 1.4, 39.0 ± 1.6, 37.0 ± 1.01, 54.0 ± 3.0 and 30.0 ± 1.42 percent respectively.

The ducks treated with 10.0% M.E. exhibited reduction in the lymphocyte count and it fluctuated between thirty percent and thirty eight percent. By the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours the lymphocyte count was equivalent to 36.0 ± 1.93, 33.0 ± 2.9, 36.0 ± 2.4, 38.0 ± 2.2, 37.0 ± 2.0, 30.0 ± 1.9 and 38.0 ± 1.7 percent respectively.

The 50% M.E. induced maximum increase in the lymphocyte count the end of twenffour hours while at the rest of the time intervals under study there was reduction in the lymphocyte number. Thus, at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours, the lymphocyte count was equivalent to 43.0 ± 2.8, 44.0 ± 2.03, 74.0 ± 2.31, 47.0 ± 1.72, 55.0 ± 2.4, 40.0 ± 3.16 and 42.0 ± 2.8 percent respectively.

The ducks exposed to 100% M.E. showed acute reduction in the lymphocyte count at the end of one twenty hours and the lymphocyte count remained lower to that observed in the control animals at all the time intervals under study. By the end of six, twelve, twentyfour, fortyeight, seventy two, ninetysix, and one twenty hours the lymphocyte count was equivalent to 50.0 ± 1.41, 55.0 ± 1.6, 42.0 ± 1.4, 47.0 ± 2.13, 56.0 ± 1.9, 53.0 ± 2.13 and 21.0 ± 1.93 percent respectively.
### ALTERATIONS IN THE LYMPHOCYTE COUNT UNDER THE INFLUENCE OF MINING EFFLUENTS.

<table>
<thead>
<tr>
<th></th>
<th>6 Hours</th>
<th>12 Hours</th>
<th>24 Hours</th>
<th>48 Hours</th>
<th>72 Hours</th>
<th>96 Hours</th>
<th>120 Hours</th>
<th>720 Hours</th>
<th>1080 Hours</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Control</strong></td>
<td>M.E. 60% ±2.0</td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td><strong>0.01%</strong></td>
<td>M.E.</td>
<td>32% ±2.7</td>
<td>25% ±2.6</td>
<td>27% ±1.9</td>
<td>30% ±1.72</td>
<td>30% ±1.6</td>
<td>43% ±3.6</td>
<td>34% ±2.4</td>
<td>41% ±2.2</td>
</tr>
<tr>
<td><strong>0.1%</strong></td>
<td>M.E.</td>
<td>42% ±2.5</td>
<td>38% ±1.6</td>
<td>34% ±3.31</td>
<td>41% ±1.8</td>
<td>32% ±1.8</td>
<td>56% ±2.4</td>
<td>44% ±2.8</td>
<td>N.D. N.D.</td>
</tr>
<tr>
<td><strong>1%</strong></td>
<td>M.E.</td>
<td>29% ±2.7</td>
<td>27% ±1.4</td>
<td>40% ±3.31</td>
<td>39% ±1.8</td>
<td>37% ±1.8</td>
<td>54% ±2.4</td>
<td>30% ±1.42</td>
<td>N.D. N.D.</td>
</tr>
<tr>
<td><strong>5%</strong></td>
<td>M.E.</td>
<td>36% ±1.93</td>
<td>33% ±2.9</td>
<td>36% ±2.4</td>
<td>38% ±2.2</td>
<td>37% ±2.0</td>
<td>30% ±1.9</td>
<td>38% ±1.7</td>
<td>N.D. N.D.</td>
</tr>
<tr>
<td><strong>10%</strong></td>
<td>M.E.</td>
<td>43% ±2.8</td>
<td>44% ±2.03</td>
<td>74% ±2.31</td>
<td>47% ±1.72</td>
<td>55% ±2.4</td>
<td>40% ±3.16</td>
<td>42% ±2.8</td>
<td>N.D. N.D.</td>
</tr>
<tr>
<td><strong>50%</strong></td>
<td>M.E.</td>
<td>50% ±1.414</td>
<td>55% ±1.6</td>
<td>42% ±1.4</td>
<td>47% ±2.13</td>
<td>56% ±1.9</td>
<td>53% ±2.13</td>
<td>21% ±1.93</td>
<td>N.D. N.D.</td>
</tr>
<tr>
<td><strong>100%</strong></td>
<td>M.E.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Note:** Unit: Lymphocytes in %.

**N.D.** : Not Determined.

All alterations are statistically significant - P < 0.01.
GRAPH NO. 27: ALTERATIONS IN LYMPHOCYTE COUNT UNDER THE INFLUENCE OF MINING EFFLUENTS.

Units: Lymphocytes in %

- 80%
- 70%
- 60%
- 50%
- 40%
- 30%
- 20%
- 10%
- 0%

Control 6 12 24 48 72 96 120 720 1080

Hours

Legend:
- 0.01% M.E.
- 0.1% M.E.
- 1% M.E.
- 5% M.E.
- 10% M.E.
- 50% M.E.
- 100% M.E.
C) THE ALTERATIONS IN THE EOSINOPHIL COUNT:

The control animals exhibited just 2.0% eosinophils in the blood. The 0.01% M.E. did not induce any significant change in the eosinophil count up to the end of twenty-nine days but at the end of thirty and forty-five days the eosinophil number increased significantly. The eosinophil count was 10.0 ± 2.0 percent at the end of thirty and forty-five days.

The ducks exposed to 0.1% M.E. exhibited wide fluctuations in the eosinophil count. The acute increase in eosinophil count was observed at the end of twenty-four hours. By the end of six, twelve, twenty-four, forty-eight, seventy-two, ninety-six and one twenty hours the eosinophil count was equivalent to 20.0 ± 1.01, 16.0 ± 1.74, 31.0 ± 1.7, 9.0 ± 1.01, 11.0 ± 1.6, 16.0 ± 1.2 and 20.0 ± 1.3 percent respectively.

Under the influence of 1.0% M.E. the ducks showed an acute eosinophilia at the end of twelve hours and then gradually the eosinophil count decreased up to the end of seventy-two hours in comparison with what was observed at the end of twelve hours. By the end of six, twelve, twenty-four, forty-eight, seventy-two, ninety-six and one twenty hours, the eosinophil count was equivalent to 12.0 ± 1.4, 29.0 ± 1.6, 14.0 ± 2.7, 12.0 ± 1.4, 10.0 ± 0.7, 12.0 ± 2.5 and 8.0 ± 1.01 percent respectively.

The ducks subjected to 5.0% M.E. showed the eosinophil count in the range of nine to twenty-two percent. At all the time intervals under study, the eosinophil count was higher than that observed for controls. By the end of six, twelve, twenty-four, forty-eight, seventy-two, ninety-six and one twenty hours the eosinophil count was equivalent to 14.0 ± 2.1, 12.0 ± 1.41, 9.0 ± 1.7, 11.0 ± 1.72, 22.0 ± 1.3, 9.0 ± 1.6 and 10.0 ± 1.72 percent respectively.

The exposure to 10.0% M.E. promoted pronounced eosinophilia. At the end of six, twelve and twenty-four hours the eosinophil count was about twelve
fold higher than that observed in the controls. The eosinophil count was equivalent to 24.0 ± 2.2, 25.0 ± 3.3, 25.0 ± 1.42, 21.0 ± 1.8, 15.0 ± 2.7, 15.0 ± 1.3 and 8.0 ± 1.0 percent at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours respectively.

Under the influence of 50.0% M.E. acute eosinophilia was observed at the end of fortyeight hours and in general the eosinophil count was higher than that observed in the control animals. The eosinophil count was equivalent to 22.0 ± 3.0, 21.0 ± 1.72, 7.0 ± 2.0, 29.0 ± 3.0, 17.0 ± 1.8, 16.0 ± 1.41 and 18.0 ± 0.74 percent at the end of six, twelve twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours respectively.

The 100.0 % M.E. induced relatively less increase in eosinophil counts as compared to that produced by the other concentrations of M.E. At the end of six, twelve, twentyfour fortyeight, seventytwo, ninetysix, and one twenty hours, the eosinophil count was equivalent to 4.0 ± 0.748, 2.0 ± 0.748, 12.0 ± 1.9, 11.0 ± 1.41, 10.0 ± 1.16, 9.0 ± 1.32 and 8.0 ± 1.01 percent respectively.
TABLE NO. 27: ALTERATIONS IN THE EOSINOPHIL COUNTS UNDER THE INFLUENCE OF MINING EFFLUENTS.

<table>
<thead>
<tr>
<th></th>
<th>6 Hours</th>
<th>12 Hours</th>
<th>24 Hours</th>
<th>48 Hours</th>
<th>72 Hours</th>
<th>96 Hours</th>
<th>120 Hours</th>
<th>720 Hours</th>
<th>1080 Hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>M.E. 2%</td>
<td>±0.87</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.01% M.E.</td>
<td>20% ±1.01</td>
<td>16% ±1.74</td>
<td>31% ±1.01</td>
<td>9% ±1.6</td>
<td>11% ±1.2</td>
<td>16% ±1.3</td>
<td>20%</td>
<td>10% ±2.0</td>
<td>10% ±2.0</td>
</tr>
<tr>
<td>0.1% M.E.</td>
<td>12% ±1.4</td>
<td>29% ±1.6</td>
<td>14% ±2.7</td>
<td>12% ±1.4</td>
<td>10% ±0.7</td>
<td>12% ±2.5</td>
<td>8%</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td>1% M.E.</td>
<td>14% ±2.1</td>
<td>12% ±1.41</td>
<td>9% ±1.72</td>
<td>11% ±1.3</td>
<td>22% ±1.6</td>
<td>9% ±1.6</td>
<td>10% ±1.3</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td>5% M.E.</td>
<td>24% ±2.2</td>
<td>25% ±1.42</td>
<td>25% ±1.8</td>
<td>21% ±2.7</td>
<td>15% ±1.3</td>
<td>8%</td>
<td>N.D.</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td>10% M.E.</td>
<td>22% ±3.0</td>
<td>21% ±1.72</td>
<td>7% ±2.0</td>
<td>29% ±3.0</td>
<td>17% ±1.8</td>
<td>16% ±1.4</td>
<td>18% ±0.74</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td>50% M.E.</td>
<td>4% ±0.748</td>
<td>2% ±1.948</td>
<td>12% ±1.41</td>
<td>11% ±1.16</td>
<td>10% ±1.32</td>
<td>9% ±1.01</td>
<td>8%</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td>100% M.E.</td>
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</tbody>
</table>

Note: Unit: Eosinophils in %.
N.D.: Not Determined.

All alterations are statistically significant - P < 0.01.
GRAPH NO. 27.2: ALTERATIONS IN THE EOSINOPHIL COUNT UNDER THE INFLUENCE OF MINING EFFLUENTS.

Units: Eosinophils in %

<table>
<thead>
<tr>
<th>Time (Hours)</th>
<th>Control</th>
<th>6</th>
<th>12</th>
<th>24</th>
<th>48</th>
<th>72</th>
<th>96</th>
<th>120</th>
<th>720</th>
<th>1080</th>
</tr>
</thead>
<tbody>
<tr>
<td>0%</td>
<td></td>
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</tr>
<tr>
<td>10%</td>
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<td></td>
<td></td>
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<tr>
<td>20%</td>
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<tr>
<td>30%</td>
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<tr>
<td>40%</td>
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</tr>
</tbody>
</table>

Legend:
- 0.01% M.E.
- 0.1% M.E.
- 1% M.E.
- 5% M.E.
- 10% M.E.
- 50% M.E.
- 100% M.E.
V) THE ALTERATIONS IN BLOOD PLATELET COUNT:

The changes in the blood platelet count are compiled in Table No. 28 and are graphically presented in Graph No. 28. The blood platelet counts are expressed as cells per cubic millimeter. The control animals had blood platelet count equivalent to 153000 ± 2000 cells per cubic millimeter. From the table and graph it appears that the changes in the blood platelet counts were neither M.E. concentrations dependent nor exposure period dependent. The 1.0% M.E. induced acute increase in the blood platelet count at the end of six hours and no other concentration of M.E. could induce such as pronounced increase in blood platelet count at any time interval.

When the animals were exposed to 0.01% M.E. there was no significant change in the blood platelets count up to the end of twentynine days but at the end of thirty days (seven hundred and twenty hours) there was pronounced increase in the platelet count which showed progressive increase up to the end of fortyfive days (one thousand and eighty hours). Then, at the end of thirty and fortyfive days the blood platelet count was equivalent to 260,000 ± 1000 and 275,000 ± 2000 cells per cubic millimeter respectively.

The birds exposed to 0.1% M.E. showed increase in the blood platelet counts but the platelet count fluctuated for different periods. By the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours the blood platelet count was equivalent to 226, 000 ± 3000, 260,000 ± 1230, 202,000 ± 984, 230,000 ± 1000, 215,000 ± 1400, 219,000 ± 1000 and 206,000 ± 1000 cells per cubic millimeter respectively.

Under the influence of 1.0% M.E. the ducks showed highest increase in the blood platelet number at the end of six hours and then the platelet count went on decreasing gradually up to the end of fortyeight hours. Then the platelet count kept on increasing up to the end of ninetysix hours. Thus, at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty
hours, the blood platelet count was equivalent to 360,000 ± 2000, 285,000 ± 592, 282,000 ± 973 and 216,000 ± 2010, 295000 ± 1200, 298000 ± 2700 and 265000 ± 2000 cells per cubic millimeter respectively.

The ducks subjected to 5.0% M.E. exhibited increase in the number of blood platelets at the end of six hours and then it kept on increasing up to the end of twentyfour hours. The platelet count fluctuated during the subsequent time intervals. The blood platelet count was equivalent to 208,000 ± 1000, 210,000 ± 792, 260,000 ± 1000, 190,000 ± 1500, 220,000 ± 2500, 215000 ± 2000 and 240,000 ± 1340 cells per cubic millimeter at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours respectively.

The 10.0% M.E. induced fluctuations in the blood platelet count. At the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours, the blood platelet count was equivalent to 216,000 ± 2000, 219,000 ± 1500, 217,000 ± 889, 230,000 ± 1400, 225,000 ± 1000, 185,000 ± 1000 and 190,000 ± 1930 cells per cubic millimeter respectively.

On exposure to 50% M.E. the ducks exhibited acute increase in the blood platelet numbers and the platelet count marginally fluctuated from the end of six hours to the end of fortyeight hours. By the end of seventytwo hours the platelet count dropped a little but improved subsequently. Thus, at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours the blood platelet count was equivalent to 295,000 ± 1670, 298,000 ± 1800, 296,000 ± 1000, 299,000 ± 757, 268,000 ± 1397, 300,000 ± 1300 and 295,000 ± 1500 cells per cubic millimeter respectively.

The ducks treated with 100.0% M.E. showed elevation of blood platelet count for all the time intervals as compared with the blood platelet count observed for the control birds. The blood platelet number exhibited fluctuations but remained almost same for six, ninetysix and one twenty hours. Thus, the blood platelet count was equivalent to 250,000 ± 1000, 245,000 ± 1500,
216,000 ± 892, 265,000 ± 937, 292,000 ± 1000, 250,000 ± 2,000 and 250,000 ± 796 cells per cubic millimeter at the end of six, twelve, twenty-four, forty-eight, seventy-two, ninetysix and one twenty hours respectively.
TABLE NO. 28 : ALTERATIONS IN THE PLATELET COUNT UNDER THE INFLUENCE OF MINING EFFLUENTS.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th>6 Hours</th>
<th>12 Hours</th>
<th>24 Hours</th>
<th>48 Hours</th>
<th>72 Hours</th>
<th>96 Hours</th>
<th>120 Hours</th>
<th>720 Hours</th>
<th>1080 Hours</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Control</strong></td>
<td>153,000</td>
<td></td>
<td></td>
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<td></td>
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<td></td>
<td></td>
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<tr>
<td></td>
<td>± 2,000</td>
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<td></td>
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</tr>
<tr>
<td><strong>0.01% M.E.</strong></td>
<td></td>
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<tr>
<td></td>
<td>226,000 ± 3,000</td>
<td>260,000 ± 1,230</td>
<td>202,000 ± 984</td>
<td>230,000 ± 1,000</td>
<td>215,000 ± 1,400</td>
<td>219,000 ± 1,000</td>
<td>206,000 ± 1,000</td>
<td>N.D.</td>
<td>N.D.</td>
<td></td>
</tr>
<tr>
<td><strong>0.1% M.E.</strong></td>
<td></td>
<td></td>
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<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td></td>
<td>285,000 ± 2,000</td>
<td>282,000 ± 592</td>
<td>216,000 ± 2,010</td>
<td>295,000 ± 1,200</td>
<td>298,000 ± 2,700</td>
<td>265,000 ± 2,000</td>
<td>N.D.</td>
<td>N.D.</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>1% M.E.</strong></td>
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<tr>
<td></td>
<td>208,000 ± 1,000</td>
<td>210,000 ± 792</td>
<td>260,000 ± 1,000</td>
<td>190,000 ± 1,500</td>
<td>220,000 ± 2,500</td>
<td>215,000 ± 2,000</td>
<td>240,000 ± 1,340</td>
<td>N.D.</td>
<td>N.D.</td>
<td></td>
</tr>
<tr>
<td><strong>5% M.E.</strong></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>216,000 ± 2,000</td>
<td>219,000 ± 1,500</td>
<td>217,000 ± 889</td>
<td>230,000 ± 1,400</td>
<td>225,000 ± 1,000</td>
<td>185,000 ± 1,000</td>
<td>190,000 ± 1,930</td>
<td>N.D.</td>
<td>N.D.</td>
<td></td>
</tr>
<tr>
<td><strong>10% M.E.</strong></td>
<td></td>
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<td></td>
</tr>
<tr>
<td></td>
<td>295,000 ± 1,670</td>
<td>298,000 ± 1,800</td>
<td>296,000 ± 1,000</td>
<td>299,000 ± 757</td>
<td>268,000 ± 1,397</td>
<td>300,000 ± 1,300</td>
<td>295,000 ± 1,500</td>
<td>N.D.</td>
<td>N.D.</td>
<td></td>
</tr>
<tr>
<td><strong>50% M.E.</strong></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>250,000 ± 1,000</td>
<td>245,000 ± 1,500</td>
<td>216,000 ± 892</td>
<td>265,000 ± 937</td>
<td>292,000 ± 1,000</td>
<td>250,000 ± 2,000</td>
<td>250,000 ± 796</td>
<td>N.D.</td>
<td>N.D.</td>
<td></td>
</tr>
</tbody>
</table>

**Note:** Unit: Platelet Counts / mm³.
N.D.: Not Determined.
All alterations are statistically significant - P < 0.01.
GRAPH NO. 28: ALTERATIONS IN THE PLATELET COUNT UNDER THE INFLUENCE OF MINING EFFLUENTS.
Units: Platelet Count / mm$^3$

Control  6  12  24  48  72  96  120  720  1080 Hours
VI) THE ALTERATIONS IN THE ERYTHROCYTE SEDIMENTATION RATE (E.S.R).:

The alterations in the Erythrocyte Sedimentation Rate (E.S.R) are given in Table No. 29 and are graphically expressed in Graph No. 29. The E.S.R. is expressed as millimeter per hour. The control birds had E.S.R. equivalent to 2.0 ± 0.00 millimeter per hour.

From the table and graph it appears that the changes in the E.S.R. were neither M.E. concentration dependent nor exposure period dependent. The 0.1 and 1.0% M.E. reduced the E.S.R. by the end of six hours. The maximum increase in E.S.R. was observed under the influence of 5.0 and 100.0% M.E. at the end of ninety-six and one twenty hours respectively.

The exposure of mallards to 0.01% M.E. did not change the E.S.R. up to the end of twenty-nine days but at the end of thirty days (seven hundred and twenty hours) the E.S.R. increased marginally but the forty-five days exposure promoted over two fold increase in E.S.R. if compared with what was obtained for the control.

Under the influence of 0.1% M.E. the ducks exhibited fifty percent reduction in E.S.R. for six and twelve hours exposures. The ducks showed acute increase in E.S.R. under the influence of 0.1% M.E. at the end of twenty-four and seventy-two hours. Thus, at the end of six, twelve, twenty-four, forty-eight, seventy-two, ninety-six and one twenty hours, the E.S.R. was equivalent to 2.0 ± 0.00, 2.0 ± 0.00, 10.0 ± 2.0, 5.0 ± 1.0, 10.0 ± 2.0, 3.0 ± 0.00 and 8.0 ± 1.0 millimeter per hour respectively.

The ducks subjected to 1.0% M.E. exhibited fifty percent reduction in E.S.R. at the end of six hours and then showed pronounced and steady increase in E.S.R. from the end of twelve hours to the end of forty-eight hours. Then, once again the E.S.R. decreased sharply at the end of seventy-two and
ninetysix hours. The E.S.R. was equivalent to $2.0 \pm 0.00$, $8.0 \pm 2.0$, $10.0 \pm 0.00$, $12.0 \pm 2.0$, $5.0 \pm 0.00$, $3.00 \pm 0.00$ and $8.0 \pm 1.0$ millimeter per hour at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours respectively.

The 5.0% M.E. promoted sharp increase in E.S.R. at the end of six and twelve hours but the highest rise in E.S.R. was observed at the end of ninetysix hours. The E.S.R. observed at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours was equivalent to $12.0 \pm 1.0$, $14.0 \pm 1.0$, $5.0 \pm 0.00$, $10.0 \pm 1.0$, $5.0 \pm 0.00$, $16.0 \pm 2.0$ and $10.0 \pm 1.0$ millimeter per hour, respectively.

The exposure of ducks to 10.0% M.E. caused increase in E.S.R. The E.S.R. increased predominantly at the end of six, twelve, twentyfour and fortyeight hours. At the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours, the E.S.R. was equivalent to $10.0 \pm 1.0$, $12.0 \pm 1.0$, $11.0 \pm 1.0$, $15.0 \pm 1.0$, $10.0 \pm 1.0$, $8.0 \pm 0.00$ and $9.0 \pm 0.00$ millimeter per hour, respectively.

The ducks treated with 50.0% M.E. showed time dependent increase in E.S.R. from the end of six hours to the end of fortyeight hours but the E.S.R. decreased slightly by the end of seventy two hours. Then, onceagain the E.S.R. increased a little at the end of ninetysix hours and remained unchanged up to the end of one twenty hours. Thus, at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours, the E.S.R. was equivalent to $8.0 \pm 1.0$, $9.0 \pm 1.0$, $12.0 \pm 1.10$, $15.0 \pm 1.0$, $10.0 \pm 1.0$, $12.0 \pm 1.0$ and $12.0 \pm 1.0$ millimeters (per hour), respectively.

The 100.0% M.E. induced very acute increase in E.S.R. at the end of twentyfour and one twenty hours. For the rest of the time intervals the E.S.R. remained above what was obtained for the control animals. Thus, at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty
hours the E.S.R. was equivalent to 10.0 ± 1.0, 10.0 ± 2.0, 15.0 ± 1.0, 12.0 ± 1.0, 10.0 ± 1.0, 8.0 ± 1.0 and 16.0 ± 2.0 millimeters (per hour) respectively.
### TABLE NO. 29: ALTERATIONS IN THE ERYTHROCYTE SEDIMENTATION RATE (ESR) UNDER THE INFLUENCE OF MINING EFFLUENTS.

<table>
<thead>
<tr>
<th></th>
<th>6 Hours</th>
<th>12 Hours</th>
<th>24 Hours</th>
<th>48 Hours</th>
<th>72 Hours</th>
<th>96 Hours</th>
<th>120 Hours</th>
<th>720 Hours</th>
<th>1080 Hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>2.0 ±0.00</td>
<td></td>
<td></td>
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<tr>
<td>0.01% M.E.</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>0.1% M.E.</td>
<td>2 ±0.00</td>
<td>2 ±0.00</td>
<td>10 ±2.0</td>
<td>5 ±1.0</td>
<td>10 ±2.0</td>
<td>3 ±0.00</td>
<td>8 ±1.0</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td>1% M.E.</td>
<td>2 ±0.00</td>
<td>8 ±2.0</td>
<td>10 ±0.00</td>
<td>12 ±0.0</td>
<td>5 ±0.00</td>
<td>3 ±0.00</td>
<td>8 ±1.0</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td>5% M.E.</td>
<td>12 ±1.0</td>
<td>14 ±1.0</td>
<td>5 ±0.00</td>
<td>10 ±0.0</td>
<td>5 ±0.00</td>
<td>16 ±2.0</td>
<td>10 ±1.0</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td>10% M.E.</td>
<td>10 ±1.0</td>
<td>12 ±1.0</td>
<td>11 ±1.0</td>
<td>15 ±1.0</td>
<td>10 ±1.0</td>
<td>8 ±0.00</td>
<td>9 ±0.00</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td>50% M.E.</td>
<td>8 ±1.0</td>
<td>9 ±1.0</td>
<td>12 ±1.10</td>
<td>15 ±1.0</td>
<td>10 ±1.0</td>
<td>12 ±1.0</td>
<td>12 ±1.0</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td>100% M.E.</td>
<td>10 ±1.0</td>
<td>10 ±2.0</td>
<td>15 ±1.0</td>
<td>12 ±1.0</td>
<td>10 ±1.0</td>
<td>8 ±2.0</td>
<td>16 ±2.0</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
</tbody>
</table>

**Note:** Unit: mm/hour.

**N.D.:** Not Determined.

All alterations are statistically significant - P < 0.01.
GRAPH NO. 29: CHANGES IN E. S. R. UNDER THE INFLUENCE OF MINING EFFLUENTS

Units: mm/h
CHAPTER-V
SECTION - B
SERUM
1) PROTEINS:

The alterations in the serum protein levels are compiled in Table No. 30 and are graphically presented in Graph No. 30. The serum protein level (Total proteins) is expressed as grams per litre of serum. The control birds showed the serum protein concentration equivalent to 3.4 ± 0.31 grams per litre. From the graph and table it appears that all the concentrations of mining effluents (M.E.) at all the time intervals (except 1.0% M.E. at the end of seventytwo hours) induced rise in the serum protein level. But the rise in the concentrations of the serum proteins was neither M.E. concentration dependent nor exposure time dependent.

The exposure of ducks to 0.01% M.E. did not induce any significant change in the serum protein levels up to the end of twenty nine days but at the end of thirty days there was significant increase in the serum protein concentrations which continued to increase up to the end of fortyfive days. Thus the serum protein concentrations were equivalent to 4.3 ± 0.2 and 6.6 ± 0.17 grams per litre at the end of thirty and fortyfive days respectively.

Under the influence of 0.1% M.E. the ducks showed the rise in the concentrations of the serum proteins at the end of six hours and it continued to rise up to the end of twenty four hours and then subsequently decreased gradually up to the end of ninety six hours but at the end of one twenty hours the serum protein concentrations increased once again. The serum protein concentrations were equivalent to 4.62 ± 0.193, 4.9 ± 0.141, 6.26 ± 0.149, 5.36 ± 0.30, 5.20 ± 0.141, 4.80 ± 0.263 and 5.32 ± 0.231 grams per litre at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours respectively.

The ducks subjected to 1.0% M.E. showed maximum reduction in the serum protein concentrations at the end of seventy two hours while a sharp increase in the serum protein level was shown at the end of one twenty hours. The serum protein level at the end of six, twelve, twentyfour, fortyeight,
seventytwo, ninetysix and one twenty hours was equivalent to 5.68 ± 0.172, 4.30 ± 0.2, 5.32 ± 0.213, 5.10 ± 0.178, 3.32 ± 0.231, 6.10 ± 0.389 and 6.70 ± 0.275 grams per litre, respectively.

The exposure to 5.0% M.E. promoted increase in the serum protein concentrations but the rise in serum protein levels had no correlation with the exposure period. The serum protein concentrations were equivalent to 5.18 ± 0.172, 5.10 ± 0.140, 5.98 ± 0.132, 5.24 ± 0.162, 5.54 ± 0.101, 6.56 ± 0.185 and 4.16 ± 0.215 grams per litre at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours respectively.

The 10.0% M.E. induced fluctuations in the serum protein concentrations but in general, the serum levels of the proteins were higher than those observed in the controled animals. The serum protein concentrations were equivalent to 5.28 ± 0.116, 5.58 ± 0.132, 5.82 ± 0.271, 4.5 ± 0.141, 5.66 ± 0.220, 5.10 ± 0.141 and 5.60 ± 0.140 grams per litre at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours respectively.

On exposure to 50.0% M.E. there was acute rise in the serum protein levels but the rise in serum protein concentrations had no correlation with the exposure period. Infact, the serum protein levels exhibited fluctuations. Thus at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours the serum concentrations of the proteins were equivalent to 5.80 ± 0.089, 5.95 ± 0.182, 5.88 ± 0.1, 6.62 ± 0.17, 6.22 ± 0.172, 6.84 ± 0.108 and 5.82 ± 0.074 grams per litre, respectively.

The exposure to 100% M.E. caused maximum increase in the serum protein concentrations at the end of fortyeight hours and at the rest of the time intervals, the serum protein levels remained above that found in the control. By the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours, the serum protein concentrations were equivalent to 5.88 ± 0.097, 6.44 ± 0.241, 6.52 ± 0.213, 7.10 ± 0.209, 6.94 ± 0.10, 6.20 ± 0.167 and 5.76 ± 0.137 grams per litre, respectively.
### TABLE NO. 30 : ALTERATIONS IN SERUM PROTEINS INDUCED BY MINING EFFLUENTS.

<table>
<thead>
<tr>
<th></th>
<th>6 Hours</th>
<th>12 Hours</th>
<th>24 Hours</th>
<th>48 Hours</th>
<th>72 Hours</th>
<th>96 Hours</th>
<th>120 Hours</th>
<th>720 Hours</th>
<th>1080 Hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control M.E.</td>
<td>3.4</td>
<td>±0.31</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.01% M.E.</td>
<td>4.62</td>
<td>±0.193</td>
<td>4.90</td>
<td>±0.141</td>
<td>6.26</td>
<td>±0.149</td>
<td>5.36</td>
<td>±0.300</td>
<td>5.20</td>
</tr>
<tr>
<td>0.1% M.E.</td>
<td>5.68</td>
<td>±0.172</td>
<td>4.30</td>
<td>±0.2</td>
<td>5.32</td>
<td>±0.213</td>
<td>5.10</td>
<td>±0.178</td>
<td>3.32</td>
</tr>
<tr>
<td>1% M.E.</td>
<td>5.18</td>
<td>±0.172</td>
<td>5.10</td>
<td>±0.14</td>
<td>5.98</td>
<td>±0.132</td>
<td>5.24</td>
<td>±0.162</td>
<td>5.54</td>
</tr>
<tr>
<td>5% M.E.</td>
<td>5.28</td>
<td>±0.116</td>
<td>5.58</td>
<td>±0.132</td>
<td>5.82</td>
<td>±0.271</td>
<td>4.50</td>
<td>±0.141</td>
<td>5.66</td>
</tr>
<tr>
<td>10% M.E.</td>
<td>5.80</td>
<td>±0.089</td>
<td>5.95</td>
<td>±0.182</td>
<td>5.88</td>
<td>±0.1</td>
<td>6.62</td>
<td>±0.172</td>
<td>6.22</td>
</tr>
<tr>
<td>50% M.E.</td>
<td>5.88</td>
<td>±0.097</td>
<td>6.44</td>
<td>±0.241</td>
<td>6.52</td>
<td>±0.213</td>
<td>7.10</td>
<td>±0.209</td>
<td>6.94</td>
</tr>
</tbody>
</table>

**Note:** Unit: Serum protein in gm/L.

N.D.: Not Determined.

All alterations are statistically significant - P < 0.01.
GRAPH NO. 30: ALTERATIONS IN SERUM PROTEIN LEVELS UNDER THE INFLUENCE OF MINING EFFLUENTS.

Units: gm/L

Hours

gm/L
2) ACID PHOSPHATASE ACTIVITY:

The alterations in the serum acid phosphatase activity are given in Table No.31 and are graphically expressed in Graph No. 31. The serum acid phosphatase activity is expressed as units per hundred millilitre of serum. The control animals showed acid phosphatase activity equivalent to 310.50 ± 3.08 units per hundred millilitre.

From the table and the graph it appears that the serum acid phosphatase activity showed M.E. concentration dependent (from 0.1 to 50% M.E.) increase for fortyeight hours time interval, while for the M.E. concentrations like 10.0 and 50.0 percent it exhibited exposure time dependent rise from the end of six hours to the end of forty eighty hours.

When the ducks were exposed to 0.01% M.E. the serum acid phosphatase activity did not exhibit any significant change up to the end of twenty nine days but it increased significantly at the end of thirty days (seven hundred and twenty hours) and continued to increase up to the end of fortyfive days (one thousand and eighty hours). Thus, at the end of thirty and fortyfive days the acid phosphatase activity was equivalent to 411.24 ± 13.772 and 678.96 ± 8.638 units per hundred millilitre of serum respectively.

The exposure to ducks to 0.1% M.E. induced marginal increase in the acid phosphatase activity at the end of six and twelve hours but subsequently it increased significantly and fluctuated considerably. At the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours, the acid phosphatase activity was equivalent to 387.78 ± 6.693, 324.576 ± 10.948, 422.556 ± 1.642, 485.208 ± 11.495, 545.10 ± 13.399, 570.492 ± 10.225 and 584.016 ± 13.358 units per hundred millilitre of serum respectively.

The 1.0% M.E. induced sharp increase in the acid phosphatase activity at the end of six hours and the enzyme activity decreased a little by the end of twelve hours. Then, the acid phosphatase activity increased significantly at
the end of twentyfour hours and subsequently went on increasing steadily up to
the end of one twenty hours. Thus, at the end of six, twelve twentyfour
fortyeight, seventy two, ninetysix and one twenty hours the acid phosphatase
activity was equivalent to 451.26 ± 11.440, 438.012 ± 10.984, 582.084 ±
1.756 units per hundred millilitre of serum respectively.

The exposure of ducks to 5.0% M.E. promoted acute decrease and
increase in the acid phosphatase activity at the end of seventy two and one
twenty hours respectively. By the end of six, twelve, twentyfour, fortyseight,
seventytwo, ninetysix and one twenty hours the serum acid phosphatase
activity was equivalent to 592.848 ± 1.464, 521.916 ± 6.669, 708.216 ± 8.917,
678.96 ± 9.399, 70.704 ± 11.914, 759.276 ± 1.435 and 837.66 ± 1.711 units per
hundred millilitre of serum, respectively.

The ducks treated with 10.0% M.E. exhibited sharp increase in the
serum acid phosphatase activity at the end of six hours and subsequently the
enzyme activity increased steadily up to the end of fortyeight hours. The
maximum increase in the enzyme activity was observed at the end of one
twenty hours. Thus, the acid phosphatase activity was equivalent to 538.752 ±
7.384, 594.54 ± 10.764, 674.82 ± 68.379, 834.072 ± 12.513, 815.304 ± 59.450,
730.02 ± 5.682 and 994.704 ± 10.192 units per hundred millilitre of serum at
the end of six, twelve, twentyfour, fortyseight, seventytwo ninetysix and one
twenty hours respectively.

The ducks subjected to 50% M.E. exhibited increase in the serum acid
phosphatase activity at the end of six hours and the enzyme activity went on
increasing up to the end of fortyeight hours. By the end of six, twelve,
twentyfour, fortyseight, seventytwo, ninetysix and one twenty hours the acid
phosphatase activity was equivalent to 387.78 ± 7.521, 523.848 ± 11.298,
664.608 ± 9.729, 909.42 ± 7.645, 374.256 ± 10.294, 452.916 ± 5.682 and
304.428 ± 9.894 units per hundred millilitre of serum respectively.
The 100% M.E. exposure caused reduction in the acid phosphatase activity at all the time intervals under study except six hours. At the end of six hours the serum acid phosphatase activity raised sharply. The maximum reduction in the acid phosphatase activity was observed at the end of twentyfour hours. The serum acid phosphatase activity was equivalent to 597.264 ± 6.720, 302.772 ± 10.488, 150.42 ± 1.386, 166.98 ± 4.14, 154.836 ± 9.494, 269.376 ± 12.42 and 208.656 ± 8.914 units per hundred millilitre of serum at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours respectively.
TABLE NO. 31: ALTERATIONS IN THE SERUM ACID PHOSPHATASE ACTIVITY UNDER THE INFLUENCE OF MINING EFFLUENTS.

<table>
<thead>
<tr>
<th></th>
<th>6 Hours</th>
<th>12 Hours</th>
<th>24 Hours</th>
<th>48 Hours</th>
<th>72 Hours</th>
<th>96 Hours</th>
<th>120 Hours</th>
<th>720 Hours</th>
<th>1080 Hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>310.5 ± 3.08</td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>0.01% M.E.</td>
<td>387.78 ± 6.693</td>
<td>324.576 ± 10.948</td>
<td>422.556 ± 1.642</td>
<td>485.208 ± 11.495</td>
<td>545.10 ± 13.399</td>
<td>570.492 ± 10.225</td>
<td>584.016 ± 13.358</td>
<td>411.24 ± 13.772</td>
<td>678.96 ± 8.638</td>
</tr>
<tr>
<td>1% M.E.</td>
<td>592.848 ± 1.464</td>
<td>521.916 ± 6.669</td>
<td>708.216 ± 8.917</td>
<td>678.96 ± 9.399</td>
<td>70.704 ± 11.914</td>
<td>759.276 ± 1.435</td>
<td>837.66 ± 1.711</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td>5% M.E.</td>
<td>538.752 ± 7.364</td>
<td>594.54 ± 10.764</td>
<td>674.82 ± 68.379</td>
<td>834.072 ± 12.513</td>
<td>815.304 ± 59.450</td>
<td>730.02 ± 5.682</td>
<td>994.704 ± 10.192</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
</tbody>
</table>

Note: Unit: μmoles/100 ml.
N.D.: Not Determined.

All alterations are statistically significant - P < 0.01.
GRAPH NO. 31: SERUM - ACID PHOSPHATASE.
Units: μmoles/100 ml.

- Control
- 6 hours
- 12 hours
- 24 hours
- 48 hours
- 72 hours
- 96 hours
- 120 hours
- 720 hours
- 1080 hours

Concentrations:
- 0.01% M.E.
- 0.1% M.E.
- 1% M.E.
- 5% M.E.
- 10% M.E.
- 50% M.E.
- 100% M.E.
3] ALKALINE PHOSPHATASE:

The changes in the serum alkaline phosphatase activity under the influence of mining effluents are compiled in Table No. 32 and are graphically presented in Graph No. 32. The alkaline phosphatase activity is expressed as mmol units per hundred millilitre of serum. The control animals had the serum alkaline phosphatase (AIP) activity equivalent to $3.16 \pm 0.537$ mmoles per hundred millilitre of serum. From the table and graph it appears that the increase in serum alkaline phosphatase activity was concentration dependent for 0.1 to 10% M.E. concentrations at the end of six and twentyfour hours only. The maximum increase in the AIP activity was observed at the end of six hours under the influence of 50% M.E. while the highest reduction in the AIP activity was observed at the end of one twenty hours under the influence of 100% M.E.

The ducks exposed to 0.01% M.E. did not show significant changes in the serum AIP activity up to the end of twenty nine days but at the end of thirty days (seven hundred and twenty hours) the serum AIP activity increased marginally and continued to increase further up to the end of fortyfive days. Thus, the serum AIP activity was equivalent to $4.0 \pm 0.120$ and $6.80 \pm 0.136$ mmoles per hundred millilitre at the end of thirty and fortyfive days.

Under the influence of 0.1% M.E. the serum AIP activity exhibited exposure time dependent increase. By the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours the serum AIP activity was equivalent to $3.85 \pm 0.068$, $4.0 \pm 0.073$, $4.15 \pm 0.109$, $4.30 \pm 0.092$, $4.60 \pm 0.100$, $5.0 \pm 0.048$ and $5.55 \pm 0.098$ mmoles per hundred millilitre of serum respectively.

The exposure of ducks to 1.0% M.E. promoted steady increase in the serum AIP activity at all the time intervals under study except at the end of forty eight hours. The acute increase in the serum AIP activity was observed at the end of one twenty hours. By the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours, the serum AIP activity was
equivalent to 5.10 ± 0.034, 5.25 ± 0.077, 6.50 ± 0.089, 5.45 ± 0.084, 7.20 ± 0.051, 7.40 ± 0.114 and 8.35 ± 0.125 mmol/100 ml

On exposing the ducks to 5.0% M.E. the serum AIP activity increased at the end of six hours and continued to increase further up to the end of twentyfour hours, but the serum AIP activity dropped significantly by the end of fortyeight hours. Then, subsequently the AIP activity showed steady rise up to the end of ninetysix hours. Thus, at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one hundred and twenty hours, the serum AIP activity was equivalent to 5.45 ± 0.104, 8.55 ± 0.108, 9.05 ± 0.086, 5.20 ± 0.121, 5.75 ± 0.056, 6.50 ± 0.071 and 6.45 ± 0.068 mmol/100 ml respectively.

The 10.0% M.E. induced fluctuations in the serum AIP activity at different time intervals. A very high AIP activity was observed at the end of fortyeight hours. By the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one hundred and twenty hours, the serum AIP activity was equivalent to 7.70 ± 0.090, 13.0 ± 0.103, 10.0 ± 0.103, 20.40 ± 0.107, 14.80 ± 0.128, 15.40 ± 0.086 and 17.0 ± 0.117 mmol/100 ml respectively.

When the ducks were introduced to 50.0% M.E. the highest serum AIP activity was observed at the end of six hours. The AIP activity decreased gradually from the end of fortyeight hours to the end of one hundred and twenty hours. By the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix, one hundred and twenty hours, the serum AIP activity was equivalent to 23.60 ± 0.145, 7.0 ± 0.087, 16.80 ± 0.092, 12.90 ± 0.106, 10.20 ± 0.107, 5.45 ± 0.062 and 4.35 ± 0.103 mmol/100 ml respectively.

The exposure of ducks to 100% M.E. caused progressive reduction in the serum AIP activity from the end of twelve hours to the end of forty eight hours after the initial rise in AIP activity at the end of six hours. The maximum
reduction in the serum AIP was observed at the end of one hundred and twenty hours. Thus, at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix, one hundred and twenty hours, the serum AIP activity was equivalent to $17.70 \pm 0.154$, $10.20 \pm 0.196$, $8.25 \pm 0.137$, $7.90 \pm 0.075$, $10.20 \pm 0.808$, $4.30 \pm 0.104$ and $2.60 \pm 0.0876$ mmoles per hundred millilitres of serum respectively.
**TABLE NO. 32: ALTERATIONS IN THE SERUM ALKALINE PHOSPHATASES ACTIVITY UNDER THE INFLUENCE OF MINING EFLUENTS.**

<table>
<thead>
<tr>
<th></th>
<th>6 Hours</th>
<th>12 Hours</th>
<th>24 Hours</th>
<th>48 Hours</th>
<th>72 Hours</th>
<th>96 Hours</th>
<th>120 Hours</th>
<th>720 Hours</th>
<th>1080 Hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>3.16</td>
<td>± 0.537</td>
<td></td>
<td></td>
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<td></td>
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<td></td>
<td></td>
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<tr>
<td>0.01% M.E.</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>4.0</td>
<td>±0.120</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.10% M.E.</td>
<td>3.85</td>
<td>±0.068</td>
<td>4.0</td>
<td>±0.073</td>
<td>4.15</td>
<td>±0.109</td>
<td>4.30</td>
<td>±0.092</td>
<td>4.60</td>
</tr>
<tr>
<td></td>
<td>5.0</td>
<td>±0.100</td>
<td>5.0</td>
<td>±0.048</td>
<td>5.55</td>
<td>±0.098</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1% M.E.</td>
<td>5.10</td>
<td>±0.034</td>
<td>5.25</td>
<td>±0.077</td>
<td>6.50</td>
<td>±0.089</td>
<td>5.45</td>
<td>±0.084</td>
<td>7.20</td>
</tr>
<tr>
<td></td>
<td>7.40</td>
<td>±0.114</td>
<td>7.40</td>
<td>±0.125</td>
<td>8.35</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5% M.E.</td>
<td>5.45</td>
<td>±0.104</td>
<td>8.55</td>
<td>±0.108</td>
<td>9.05</td>
<td>±0.086</td>
<td>5.20</td>
<td>±0.121</td>
<td>5.75</td>
</tr>
<tr>
<td></td>
<td>6.50</td>
<td>±0.071</td>
<td>6.50</td>
<td>±0.068</td>
<td>6.45</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10% M.E.</td>
<td>7.70</td>
<td>±0.090</td>
<td>13.0</td>
<td>±0.103</td>
<td>10.0</td>
<td>±0.103</td>
<td>20.40</td>
<td>±0.107</td>
<td>14.80</td>
</tr>
<tr>
<td></td>
<td>15.40</td>
<td>±0.128</td>
<td>15.40</td>
<td>±0.117</td>
<td>17.0</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>50% M.E.</td>
<td>23.60</td>
<td>±0.145</td>
<td>7.0</td>
<td>±0.087</td>
<td>16.80</td>
<td>±0.092</td>
<td>12.90</td>
<td>±0.106</td>
<td>10.20</td>
</tr>
<tr>
<td></td>
<td>5.45</td>
<td>±0.062</td>
<td>5.45</td>
<td>±0.103</td>
<td>4.35</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>100% M.E.</td>
<td>17.70</td>
<td>±0.154</td>
<td>10.20</td>
<td>±0.196</td>
<td>8.25</td>
<td>±0.137</td>
<td>7.90</td>
<td>±0.075</td>
<td>10.20</td>
</tr>
<tr>
<td></td>
<td>4.30</td>
<td>±0.104</td>
<td>4.30</td>
<td>±0.087</td>
<td>2.60</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Note:** Unit: mmoles/100 ml.

**N.D.:** Not Determined.

All alterations are statistically significant - $P < 0.01$. 
GRAPH NO. 32: SERUM - ALKALINE PHOSPHATASE.
Units: mmoles/100 ml.

Graph showing the concentration of serum alkaline phosphatase over time for various concentrations. The y-axis represents mmoles/100 ml, and the x-axis represents hours from 0 to 1080.
enzyme activity. By the end of fortyeight hours the duck showed a marginal
decrease in the nonspecific esterase activity while the glomeruli and the other
tubules did not exhibit any esterase (nonspecific) activity. In a few cases the
walls of Bowman's capsule showed a very weak enzyme activity (Plate 64 : Fig 1). Subsequently, at the end of seventytwo and ninetysix hours the ducks showed gradual increase in the enzyme activity in about sixty percent tubules, but by the end of one twenty hours the ducks showed increase in the enzyme activity in comparison to that seen at the end of fortyeight hours and showed marginally less than that observed at ninetysix hours. The other forty percent tubules showed the absence of enzyme activity (Plate 64 : Fig 2). The glomeruli and collecting tubules also showed absence of esterase (nonspecific) activity.

Under the influence of 10.0% M.E. the ducks showed a sharp increase in the enzyme activity in about sixty percent tubules and a few of these tubules showed the brush/luminal border esterase (nonspecific) activity. About thirty eight percent tubules showed absence of enzyme activity, while about two percent tubules showed weak, membrane bound esterase activity (Plate 64 : Fig 3). By the end of twelve hours the sixty percent tubules showed slight reduction in the enzyme activity while about thirty eight percent tubules showed absence of enzyme activity and around two percent tubules exhibited weak, membrane bound, esterase activity. The wall of Bowman's capsule showed mild esterase activity (Plate 64 : Fig 4). At the end of twentyfour hours the ducks exhibited further decrease in the nonspecific esterase activity in about sixty percent tubules (proximal and distal). These tubules occasionally showed weak, membrane bound, esterase activity. The other tubules did not show any activity (Plate 65 : Fig 1). The glomeruli and collecting tubules showed absence of enzyme activity. By the end of fortyeight hours the nonspecific esterase activity from about sixty percent tubules increased sharply while the remaining tubules showed absence of enzyme activity. The glomeruli did not
CAPTIONS TO FIGURES

PLATE 65:

Fig 1: 10.0% M. E. - 24 hours - Esterase (Nonspecific) - Indoxyl acetate.
(20 x 5)
Note further reduction in the enzyme activity from the tubules and the occurrence of membrane esterase at the nucleus (solid arrow). Also note the absence of enzyme activity from other tubules (dashed arrow).

Fig 2: 10.0% M. E. - 48 hours - Esterase (Nonspecific) - Indoxyl acetate.
(20 x 5)
Note increase in esterase activity in the tubules (solid arrow) and absence of activity in other tubules (dashed arrow) as well as in glomerulus (GL). Also note weak esterase in the luminal debris. (D).

Fig 3: 10.0% M. E. - 72 hours - Esterase (Nonspecific) - Indoxyl acetate.
(10 x 5)
Note further increase in the esterase activity (solid arrow) and absence of esterase activity in other tubules.

Fig 4: 10.0% M. E. - 120 hours - Esterase (Nonspecific) - Indoxylacetate.
(20 x 5)
Note intense staining in the tubules (DT, solid arrow) and absence of enzyme activity in other tubules (dashed arrow).
exhibit any enzyme activity but the debris in the lumen showed weak enzyme activity. The interstitium was without esterase activity (Plate 65: Fig 2). By the end of seventytwo hours the ducks exhibited further increase in the nonspecific esterase activity from about sixty percent tubules, while the remaining forty percent tubules showed absence of enzyme activity (Plate 65: Fig 3). The glomeruli and collecting tubules showed no enzyme activity. At the end of ninetysix hours, the ducks showed increase in the esterase activity from about sixty percent tubules while at the end of one twenty hours there was no significant change in the enzyme activity if compared to that obtained at ninetysix hours. Some of the tubules from the sixty percent group showed very intense enzyme activity and the luminal debris showed weak enzyme activity. The other tubules (about forty percent) did not show any enzyme activity. The interstitium was without any enzyme activity (Plate 65: Fig 4).

When the mallards were exposed to 50% M.E. a few tubules showed sharp increase in the enzyme activity and in some cases they showed brush/luminal border esterase activity. The other tubules, glomeruli and collecting tubules showed absence of esterase (nonspecific) activity. The interstitium showed no enzyme activity (Plate 66: Fig 1). Subsequently, the ducks showed gradual reduction in the enzyme activity at the end of twelve and twentyfour hours. But by the end of fortyeight hours the ducks showed increase in the esterase activity in about sixty percent tubules, while the remaining tubules, glomeruli and collecting tubules exhibited absence of enzyme activity. The luminal debris showed esterase activity (Plate 66: Fig 2). Then, at the end of seventy two hours, once again the nonspecific esterase activity reduced sharply from the sixty percent tubules and increased by the end of ninetysix hours. By the end of one twenty hours the ducks showed very intense enzyme activity in about sixty percent tubules and a few of these tubules showed brush/luminal border esterase (nonspecific) activity. The
CAPTIONS TO FIGURES

PLATE 66:

Fig 1: 50.0% M. E. - 6 hours - Esterase (Nonspecific) - Indoxyl acetate. (20 x 5)
Note intense staining in few tubules (solid arrow) and reduced esterase activity in some tubules (DT). Also note the absence of enzyme activity in other tubules (dashed arrow). The collecting tubules (CT) show absence of enzyme activity. Glomerulus (G) shows no enzyme activity.

Fig 2: 50.0% M. E. - 48 hours - Esterase (Nonspecific) - Indoxyl acetate. (10 x 5)
Note increase in the enzyme activity in some tubules (solid arrow). Also note the absence of esterase activity in other tubules (dashed arrow) and collecting tubules (CT).

Fig 3: 50.0% M. E. - 120 hours - Esterase(Nonspecific) - Indoxyl acetate. (10 x 5)
Note intense staining in the tubules (solid arrow) and absence of enzyme activity in other tubules (dashed arrow) and glomerulus (G).

Fig 4: 100.0% M. E. - 24 hours - Esterase (Nonspecific) - Indoxyl acetate. (20 x 5)
Note increase in the esterase activity in some tubules (DT) and absence of enzyme activity in other tubules (dashed arrow).
CAPTIONS TO FIGURES

PLATE 67:

Fig 1: 100.0% M. E. - 72 hours - Esterase (Nonspecific) - Indoxyl acetate. (10 x 5)
Note further increase in the enzyme activity and absence of esterase activity in glomerulus (G) and collecting tubules (CT).

Fig 2: 100.0% M. E. - 120 hours - Esterase (Nonspecific) - Indoxyl acetate. (40 x 5)
Note intense staining in the tubules (DT) and the esterase activity in the luminal debris. Also note absence of enzyme activity in other tubules (dashed arrow).
The ultrastructural changes induced by M.E. in the proximal tubules are like disruption of brush border, emptying of lysosomal vesicles, pycnosis of tubule nuclei, accumulation of cytoplasmic and nuclear debris in the lumina, vacuolisation of tubule cytoplasm, destruction of mitochondria, deposition of heavy metals in the tubule cytoplasm. The intensity of necrotic alterations appear to depend upon the exposure period and the concentrations of M.E. The necrotic changes in the proximal tubules may be due to the deposition of heavy metals, activation of lysosomes and lysosomal enzymes which promote degenerative changes and in addition the depletion of energy due to the destruction of mitochondria may activate lysosomes to promote degeneration of tubule structure. Prasad Rao et. al., (1989) have reported accumulation of lipid droplets, lysosomal bodies and membrane bound vacuoles in methyl mercury treated ducks while lead exposed birds exhibit large number of secondary lysosomes and swollen mitochondria in proximal tubules. These observations support the findings in the present investigation up to some extent but the ultrastructural changes observed under the influence of M.E. are more pronounced. The ducks do not show any ultra structural change probably till the heavy metal retentions reach a threshold level to promote any toxic action.

The ultrastructural changes in the distal tubules are like vacuolisation of cytoplasm, swelling of Golgi bodies, heavy metal depositions, pycnosis of nuclei, lipid depositions in the cytoplasm, swelling of distal tubules, reduction in lumina depositions of tissue debris in the lumina, proliferations of Golgi bodies, formation of autophagic vacuoles, widening of intercellular space, disruption of basal membrane, heavy metal depositions in the nuclei of tubule cells, destruction of mitochondria, etc. These ultrastructural changes are observed at the end of six hours and at later hours the degree of necrosis increases depending upon the exposure period and the concentrations of M.E. Prasad Rao et. al., (1989) report the lipid depositions in the proximal tubules of ducks exposed to heavy metals like methyl mercury, lead and cadmium but in the
The present study shows that the lipid depositions occur in the cytoplasm of distal tubule cells. The ultrastructural changes observed in the present investigations under the influence of various concentrations of M.E. are quite pronounced and have not been reported thus far. The presence of heavy metal depositions in the tubule cell cytoplasm and nucleoplasm clearly indicate the possibility of heavy metals' involvement in inducing necrotic changes.

The changes in the ultrastructure of collecting tubules under the influence of M.E. are quite pronounced and are like distortion of basal membranes, pycnosis of nuclei, depositions of heavy metals in the nuclei, vacuolisation of mitochondria, heavy deposition of lipid droplets, disruption of luminal borders, swelling of Golgi bodies, proliferation of endoplasmic reticulum, ciliary processes, Golgi profiles. The 100.0% M.E. promote acute proliferations of E.R., ciliary processes and Golgi profiles into the lumina.

The degree of acuteness of the ultrastructural changes depend upon the exposure period and the concentration of M.E. The mitochondrial and endoplasmic reticular damages along with the destruction of Golgi could lead to severe alterations/disruptions in the metabolism of the tubules and such alterations may develop energy crisis. The destruction of mitochondria may lead to shortage of energy which may activate lysosomes leading to the degenerations of the tissue.

Some researchers have reported heavy metal promoted renal lesions to the fishes (Saxena and Parashari, 1981; Thiyagarajah et al., 1989). These observations support the involvement of heavy metals in nephrotoxicity. Thus, in the present investigation the heavy metals could be associated with the nephrotoxicity observed in the ducks.
C) HISTOCHEMICAL CHANGES:

I) ACID PHOSPHATASE:

The studies on acid phosphatase of the kidneys of control ducks revealed the presence of two types of renal tubules. One type shows the presence of brush/luminal border and cytoplasmic acid phosphatase activity while the other type shows only cytoplasmic activity. Nearly sixty percent tubules bear brush/luminal border as well as cytoplasmic acid phosphatase activity while, forty percent tubules contain only cytoplasmic activity. It is very difficult to adjudge on the basis of acid phosphatase activity, if the tubules are reptilian type or mammalian type. On exposure to mining effluents the ducks show the acid phosphatase activity bound to the membranes in addition to brush/luminal border and cytoplasmic acid phosphatase activity.

Therefore, the present investigation reveals the presence of three types of acid phosphatases based on the locations, a - brush/luminal border, cytoplasmic and membrane bound. The kidney sections of the controls show brush/luminal border acid phosphatase activity and a few glomeruli show acid phosphatase activity. These glomeruli could be mammalian type as mammalian glomeruli are known to bear acid phosphatase activity (Farquhar, 1961, Dingle and Fell, 1969).

The ducks exposed to 0.01% M.E. bear the increased acid phosphatase activity at the end of thirty days which increases further up to the end of fortyfive days. The increase in acid phosphatase activity indicates activations of lysosomes and hence the increase in the degenerative changes.

By the end of six hours the acid phosphatase activity increases as the M.E. concentration increases, indicating higher the concentrations of M.E., higher is the enzyme activity. As the degree of necrosis increases with the M.E. concentration naturally the proportionate rise in acid phosphatase activity
is justified as the acid phosphatase is a lysosomal enzyme and the lysosomes are responsible for autophagy viz-a-viz degenerations of tissues.

The 0.1, 5.0, and 100.0% M.E. concentrations induce decrease in the acid phosphatase activity at the end of twelve hours while 1.0, 10.0 and 50.0 percent concentrations of M.E. promote increase in the acid phosphatase activity from the brush/luminal borders. The 50.0% M.E. induce cytoplasmic granular lysosomal staining.

The M.E. concentrations of 1.0, 5.0, 10.0 and 100.0 percent induce increase in acid phosphatase activity at the end of twenty four hours while 50% M.E. promote decrease in brush/luminal border enzyme activity but at the same time cause increase in glomerular and cytoplasmic acid phosphatase activity. The 5.0% M.E. induce decrease in the cytoplasmic enzyme activity. By the end of fort yeight hours, the 0.1, 1.0 and 10.0 percent M.E. induce increase in the enzyme activity while 50 percent M.E. promote decrease in brush/luminal border acid phosphatase activity and increase in cytoplasmic enzyme activity. The 100% M.E. cause decrease in the cytoplasmic acid phosphatase activity. At the end of seventy two hours the 0.1 and 1.0 percent M.E. promote increase in acid phosphatase activity at the brush/luminal borders and also increase the brush/luminal border enzyme activity in the tubules previously showing cytoplasmic enzyme activity. The 100 percent M.E. induce decrease in brush/luminal border activity but at the same time increase in the cytoplasmic acid phosphatase activity.

The M.E. concentrations of 0.1 and 50.0 percent induce increase in brush/luminal border acid phosphatase activity at the end of ninetysix hours while 1.0, 5.0, 10.0 and 100 percent M.E. promote decrease in the acid phosphatase activity but at the same time 10.0% M.E. induce increase in the cytoplasmic granular enzyme activity. By the end of one hundred and twenty hours the 0.1, 50.0 and 100% M.E. cause reduction in the acid phosphatase
activity. The 1.0 and 5.0% M.E. concentrations induce increase in the enzyme activity.

It is interesting to note that under the influence of M.E. the acid phosphatase activity change the locations/sites and appears at the membranes such as Bowman's wall, basement membrane and nuclear membranes. Even the cytoplasmic and nuclear debris in the lumina show acid phosphatase activity.

Acid phosphatase is a lysosomal enzyme and generally the lysosomes of the cells of the proximal tubules are responsible for the absorption of proteins and other high molecular weight substances from the glomerular filtrate which come to the lumina of the tubules (Maunsbach, 1973). The proximal and distal tubule lysosomes are responsible for the degradation of cytoplasmic components including mitochondria-like structures or organelle complexes (Straus and Oliver, 1955; Sellers, 1955; Squire et al., 1962; Pitts, 1963; Hamburger et al., 1968; Rhodin, 1954; Novikoff, 1959; Miller, 1960, 1962; Miller and Palade, 1964; Ericsson, 1964; Trump and Bulger, 1965 as cited by Dingle and Fell, 1969). During the necrosis of the glomeruli all the above mentioned components are released and then they come to the renal tubules. The renal tubules also receive injury and under such pathological conditions, to carry out the usual physiological functions the lysosomal acid phosphatase may be increasing up to certain extent but in the period of acute necrosis the enzyme activity reduces as it gets excreted through urine and passes down the concentration gradient to the extracellular spaces and blood (Merill et al., 1956).

The lysosomes of intercapillary cells are responsible for sweeping and unclogging the basement membrane (Farquhar, 1961; Farquhar and Palade, 1962 as cited by Dingle and Fell, 1969) and the degradation of plasma proteins which have passed through glomerular basement membrane (Dixon et al., 1950; Michael et al., 1977). The acid phosphatases appearing in the
glomeruli may probably have some role in the degradation of proteins or absorbed residues. The rise in acid phosphatase activity may be for dealing with the degradable debris and the loss may be due to its excretion through urine or release into the blood and extracellular spaces. The luminal debris may be showing enzyme activity of cytoplasmic origin or may be released from the brush/luminal borders for the degradation of debris. The appearance of membrane acid phosphatases in Bowman's capsule may be for degrading the exudents in the Bowman's space as well as for degradation of damaged membrane components. Similarly, the nuclear membrane acid phosphatase may be responsible for the degradation of nucleoplasmic debris.

II) ALKALINE PHOSPHATASE:

The duck kidney bears the typical granular lysosomal alkaline phosphatase activity. The staining reactions of the kidney sections reveal the two types of tubules: one darkly stained and the other lightly stained. The darkly staining proximal and distal tubules show moderate granular alkaline phosphatase activity with occasional weak brush/luminal border enzyme activity as well as membrane alkaline phosphatase activity at the nuclear membranes. The light staining tubules bear just the fine granular (lysosomal) enzyme activity.

The 0.01% M.E. promote increase in the alkaline phosphatase (AIP) activity at the end of thirty and forty-five days when the necrosis is progressing. The AIP activity from all the renal tubules increases at the end of six hours in the M.E. concentration dependent manner i.e the rise in AIP activity was proportionate to the increase in M.E. concentration. The histopathological studies have revealed that the degree of necrosis increases as the M.E. concentration rises. Therefore, the rise in AIP activity may be for degrading the degenerative products/tissue debris as AIP is a lysosomal enzyme and the lysosomes are responsible for the degradation of cytoplasmic components.
including mitochondria or organelle complexes (Dingle and Fell, 1969). All the concentrations of M.E. induce increase in AIP activity at all the time intervals under study except at a few occasions. Especially, 1.0 and 100.0% M.E. at the end of 12 hours, 10.0, 50.0 and 100.0% M.E. at the end of twenty four hours, 5.0% M.E. at the end of fortyeight hours; 10.0, 50.0 and 100.0% M.E. at the end of seventy two hours; 10.0% at the end of ninetysix hours and 100.0% M.E. at the end of one hundred and twenty hours, induce reduction in the AIP activity. The glomerular AIP activity appears to increase according to the degree of necrosis. The exudents exhibit increased AIP activity and the enzyme activity may be cytoplasmic in origin or probably secreted from the brush/luminal borders. A pronounced increase in the granular lysosomal staining observed in the glomeruli and the cytoplasm can be taken as the activation of lysosomes, thereby promoting the release of AIP. Maunsbach (1973) has postulated that the glomerular filtrates under necrotic conditions contain proteins as well as high molecular weight substances which come to the tubules' lumina and the lysosomes of the cells of the tubules, especially of proximal tubules are responsible for the absorption of proteins and high molecular weight substances. In the present study the rise in lysosomal staining from the cytoplasm of both the proximal and distal tubules as well as elevated brush/luminal border, AIP activity may probably be responsible for the absorption of material from the tubules' lumen as suggested by Maunsbach (1973). Besides, the rise in the lysosomal staining may be for sweeping and unclogging of the basement membranes (Farquhar, 1961; Farquhar and Palade, 1962). The reduction in the AIP activity at a few time intervals under the influence of some concentrations of M.E. may be due to the acute necrosis of the proximal and distal tubules causing exudation of tissue debris into the lumen and excretion of AIP through urine as well as passing down the concentration gradient to the extracellular spaces and blood. (Merill et al., 1956). The appearance of AIP at the membranes may be due to the loss of
cytoplasmic AIP as these membrane AIPs may be useful for degrading the exudents from the Bowman's space or nucleoplasm or interstitium or even for degradation of damaged membrane proteins.

It is interesting to note that the collecting tubules did not show acid phosphatase activity but do exhibit the alkaline phosphatase activity and the alkaline phosphatase being a lysosomal enzyme may be considered responsible for the degradation of the tissue debris/products of necrosis. The AIP in the luminal exudents may be of cytoplasmic origin as well as from the brush/luminal border release. The membrane AIP of collecting tubules may be playing the role ascribed to it for proximal and distal tubules under necrosis. The enzyme activation/inhibition studies confirm that AIP is a true lysosomal enzyme.

III) NONSPECIFIC ESTERASE:

The histochemical studies of esterase (nonspecific) with indoxyl acetate method reveal the presence of two types of renal tubules on the basis of distribution of esterase activity, in the kidney of control and experimental ducks. The first group comprising of about sixty percent tubules bear the fine granular lysosomal activity and the other group comprising of about forty percent tubules are without any esterase activity. The sixty percent collecting tubules bear very weak lysosomal enzyme activity and the remaining have no esterase activity. The glomeruli do not have any esterase activity.

The 0.01% M.E. treatment induces increase in the esterase (nonspecific) activity at the end of thirty and forty five days. This increase in esterase activity shows its association with progression of necrosis. The histochemical studies of esterase (nonspecific) clearly reveal the increase of esterase activity in association with the rise in concentrations of M.E., especially in the 60 percent of proximal and distal tubules at the end of six
hours and the enzyme activity is mostly lysosomal (granular) but in a few occasions brush luminal/border and membrane esterases are present.

The 0.1% M.E. induces appearances of esterases in the second group of tubules where normally no enzyme activity is seen. This concentration of M.E. also promotes occurrence of glomerular and membrane esterases in Bowman's wall in a few cases. But the collecting tubules, glomeruli and forty percent proximal and distal tubules bear no esterase activity under the influence of M.E. In general, mostly all the concentrations of M.E. induce rise in the esterase activity from sixty percent tubules for all time intervals except a few reductions observed under the influence of a few M.E. concentrations for a few time intervals. These inclines and declines in the esterase activity appear to depend upon the degree of necrosis and the particular stage of necrosis (early or late). In a very few occasions the collecting tubules show the esterase activity in the form of membrane bound enzyme. Thus, it appears that the esterase does not contribute to the necrosis or degradation of necrotic products of collecting tubules. The exudents in the lumina of the renal tubules contain esterase which may be of cytoplasmic or luminal border in origin and may be considered as responsible for the degradation of the debris or being eliminated with debris. The rise in nonspecific esterase activity from the proximal tubules may be considered essential for absorption of material from the lumen (Maunsbach, 1973), as damaged glomeruli are likely to pass on the proteins and other high molecular weight substances to the renal tubules. Besides, the rise in non-specific esterase from the necrotic proximal and distal tubules may be considered essential for degradation of cytoplasmic components including mitochondria and organelle complexes (Straus and Oliver, 1955; Sellers, 1955; Squire et al., 1962; Pitts, 1963; Hamburger et al., 1968; Rhodin, 1954; Novikoff, 1959; Miller, 1960, 1962; Miller and Palade, 1964; Ericsson, 1964; Trump and Bulger, 1965 as cited by Dingle and Fell, 1969). The reduction in the esterase activity from the renal tubules may probably be due to the
excretion of estrases through urine and its passing down the concentration gradient to the extracellular spaces and blood (Merill et al., 1956). The appearance of esterase in glomeruli may be required for unclogging of basement membrane as suggested by Farquhar (1961) and Farquhar & Palade (1962). Besides, the lysosomal esterase activity from glomeruli be considered essential for the degradation of plasma proteins which are passing through glomerular basement membrane (Dixon et al., 1950; Michael et al., 1977). The appearance of membrane esterase in Bowman's wall could be for degradation of exudents from the Bowman's space or for the degradation of membrane proteins released during membrane disruptions. Similarly, the membrane esterase from the collecting tubules be considered essential for degradation of tissue debris in the lumina or for the degradation of exudents in the interstitium or intercellular spaces.

Thus, the histochemical studies of AP, AIP, and nonspecific esterase reveal that the degradation of necrotic contents of the collecting tubules is entrusted to the alkaline phosphatase and to a little extent to other two enzymes as and when they appear either in the cytoplasm or membranes.

BIOCHEMICAL STUDIES:

I) ACID PHOSPHATASE:

The acid phosphatase activity increases in association with the increase in the concentrations of mining effluents at the end of six hours except for 10.0% M.E. The 0.01 and 0.1% M.E. induce progressive increase in the acid phosphatase activity as the exposure period increases but 1.0, 5.0, 10.0, 50.0% M.E. induce reduction in the acid phosphatase activity at the end of a few time intervals, though otherwise these M.E. concentrations induce progressive increase in the acid phosphatase activity in association with the
increase in the degree of necrosis. Particularly at the end of one hundred and twenty hours, the 0.1, 1.0, 5.0, 10.0 and 50.0 percent M.E. induce increase in the acid phosphatase activity beyond the level present at the end of six hours. The 100.0% M.E. induces maximum increase in the acid phosphatase activity at the end of six hours and the activity keeps on decreasing from the end of seventy two hours to the end of one hundred and twenty hours. The rise and falls in the acid phosphatase activity appear to be correlated with the progress and state of necrotic activity of the kidney.

Slater and Greenbaum (1963) reported that the increase in acid phosphatase (AP) activity of the kidney is proportional to the degree of necrosis and the AP activity does not increase before the onset of renal injury. Therefore, in the present investigation the AP activity does not increase under the influence of 0.01% M.E. up to the end of twenty nine days, as, till this time, there is no onset of renal injury but in the later periods the acid phosphatase activity increase in proportion with the progress of necrosis. Under the influence of other concentrations of M.E. the AP activity increases in proportion with the degree of necrosis induced by M.E. for a specific exposure period.

The other researchers have also reported this enzyme in necrosis in other tissues. Becker and Barron (1961) have reported an increase in AP activity in the injured neurons. Gould and Holt (1961), Kawai (1963), Holtzman and Novikoff (1965) and Friede (1966) have reported increase in the AP activity in the axons undergoing changes subsequent to injury. Dianzani (1963) have reported an increase in AP activity subsequent to the liver injury: The relationship between the elevation of AP activity and necrosis is well established and a vast literature is available on this aspect. (Lysosomes in Biology and Pathology Vol. 1 to 5 edited by Dingle and Fell Dean. Vol I - 1969, Vol II - 69, Vol III - 1973, Vol. IV - 1975 and Vol. V - 1976).

In the present investigation the enhancement of AP activity under the influence of : 0.1% M.E. from the end of six to the end of one hundred and
twenty hours; 1.0% M.E. for six, twentyfour, seventy two and one hundred and twenty hours; 5.0% M.E. from six, twenty four, fortyeight, ninetysix and one twenty hours; 10.0% M.E. at the end of six, twelve, twenty four, fortyeight, ninetysix and one twenty hours; 50% M.E. at the end of six, twelve, nientysix and 100% M.E. at the end of six hours, follow the degree of necrosis. By the time the kidney exhibits maximum tubular necrosis, the AP activity increases far above the control level and above the level observed at the end of six hours (except for 100% M.E.) as the exposure period of one twenty hours approaches. The histological studies have revealed the progression of necrosis and involvement of more tubules in necrosis as the exposure period increases. These observations indicate the relationship between the progress of necrosis and progressive increase of AP activity. The AP activity increases in renal nephritis to destroy or to break down the damaged cell constituents (Sztriha et. al., 1975). Dingle and Fell (1969) have reported the scavanging role of AP in liver necrosis. Kobayashi et. al., (1971) have reported high level of AP activity in diseased tissue. The elevation of AP activity in the damaged portion of the kidney is an important factor in the metabolism of necrotic kidney. The same may be true in the present case as the rise in the acid phosphatase runs parallel to the rise in the necrosis of the kidney.

It may possibly be that during necrosis some of the lysosomes get lost or the lysosomal enzymes from the cytoplasm may get passed on to the lumen along with the cytoplasmic debris. Histochemical studies support this as the luminal debris show the lysosomal staining. Hence, this may be the reason of decrease in the AP activity at a few time intervals. In the rat proximal tubules cytosomes and cytosegresomes are found and both these bodies are responsible for high specific AP activity, at least in part (Straus, 1956). The renal damage may probably be responsible for the loss of such bodies which may atleast partly be related to the decrease in AP activity, especially for very large M.E. concentrations which cause acute necrosis. This may also happen
for the M.E. concentrations above 1.0% M.E. when they induce acute necrosis at a few time intervals, may be in a few tubules. But it needs further investigation to throw adequate light on this aspect. The higher renal clearance of AP also supports the above suggestion. Merill et. al., (1956) put up a hypothesis of passing down of enzymes against the concentration gradient into the extracellular space and blood. In the present studies the reduction in the AP activity may be due to such a phenomenon as suggested by Merill et. al., (1956). In the present investigation the serum shows an increase in AP activity and this increase can be correlated to the decrease in the kidney AP up to certain extent. Thus, the decrease in AP activity in the kidney may be due to its renal clearance and passing down in to the serum as well as into the extracellular spaces. But these correlations are by no means constant as it depends upon the degree of necrosis, passing of the enzyme down the concentration gradient into the extracellular space and blood. The intermittent fluctuations in the enzyme activity can be attributed to the lysosomes which become very active after the cytoplasmic exudation which take with them some of the lysosomes to the debris.

II) ALKALINE PHOSPHATASE:

The alkaline phosphatase (AIP) activity increases in association with the increase in the concentrations of mining effluents (from 0.1 to 50%) at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninety six and one hundred and twenty hours barring a few time intervals/instances. The alkaline phosphatase activity fluctuates for some time intervals but remains above that obtained for the control. The fluctuations in the AIP activity correlate partly with the degree of necrosis, number of renal tubules involved in necrosis and partly with the renal clearance of AIP as well as its passing down the concentration gradient in to the extracellular space and blood, (Merill et. al., 1956). But a perfect stable correlation in the fluctuations of AIP activity with the
aforementioned factors cannot be obtained. There is some correlation between the degree of necrosis and the enhancement of the AIP activity. The higher concentrations of M.E. induce acute necrosis and hence under such conditions enhancement of AIP activity is obtained. The AIP activity does not increase before the onset of injury. Therefore, these observations are in agreement with the observations of Slater and Greenbaum (1963) for acid phosphatase. Here, one must remember that like AP, AIP is also a lysosomal enzyme. Under the influence of 0.01% M.E. the AIP activity does not increase up to the end of twenty-nine days as the necrosis is not set on. But once the necrosis sets on, the AIP activity increases gradually. The other concentrations of M.E. also do not induce AIP enhancement up to the beginning of six hours period as the necrosis does not begin. The enhancement in AIP activity in proportion to the degree of necrosis may be justifiable as such a correlation has been obtained by many researchers with various necrotic tissues for AP activity (Becker and Barron, 1961; Gould and Holt, 1961; Kawai, 1963; Holtzman and Novikoff, 1965; Friede, 1966; Dianzani, 1963 and Dingle and Fell, 1969).

III) NON SPECIFIC ESTERASE :

The renal nonspecific esterase activity does not increase before the onset of necrosis but soon after the beginning of necrosis, a slight increase in the esterase activity is seen and when the necrosis progresses, then only significant increase in the esterase activity is seen. Almost all the concentrations of M.E. except 5.0% induce significant increase in the esterase activity at the end of six hours. Histopathological studies have revealed that as the exposure period increases, the necrosis progresses and more number of tubules are involved in the necrosis. In the present studies also it is observed that, as the necrosis progresses and more number of tubules are becoming necrotic, the esterase activity increases up to certain extent, especially at the end of one twenty hours when more tubules are necrotic, the enhancement in
esterase activity over that level seen at six hours, is obtained. The fluctuations in the enzyme activity may probably be due to the passing of esterases down the concentration gradient into the extracellular space and blood (Merill et. al., 1956) and partly may be due to the renal clearance of the enzyme through urine as well as due to the passing of cytosomes and cytosegresomes in to the debris (Straus, 1956). The elevation of serum esterase activity supports Merill et. al., (1956) hypothesis and the elevation of esterase activity in proportion to the increase in necrosis can be considered as in agreement with the observation of some researches for AP in kidney and other tissues (Sztriha et. al., 1975; Dingle and Fell, 1969; Kobayashi et. al., 1971; Becker and Barron, 1961; Gould & Holt, 1961; Kawai, 1963; Holtzman & Novikoff, 1965; and Friede, 1966), as esterase is also lysosomal enzyme.

IV) PROTEINS:

The present investigation shows that the mining effluents (M.E.) reduce the kidney protein levels. Under the influence of all the M.E. concentrations the protein concentrations decrease and the decrease in the protein level appears to be dependent upon the degree of necrosis which is dependent upon the M.E. concentration as well as exposure periods. For very low concentrations of M.E. there are no changes in protein levels which indicates that there is no renal damage. The histopathological observations also show that the very low concentrations of M.E. do not induce renal necrosis unless the animals are exposed to M.E. for a longer period. In the present investigation the non alterations of the amount of proteins in the kidney up to the end of twenty nine days under the influence of 0.01% M.E. clearly shows that there is no renal damage. But after thirty days exposure the kidney shows reduction in the amount of proteins indicating the onset of necrosis.

Therefore, the reductions in the amount of proteins may be due to necrosis. Under the influence of larger M.E. concentrations, the kidney tubules
undergo acute necrosis and the tubules lose their integrity and many tubule cells are damaged and sloughed off. Their cytoplasmic constituents get broken down by the action of lysosomal enzymes. Besides these, cytoplasmic debris is exuded in the lumina or interstitial spaces and the proteins pass down the urine and also escape into the circulation. The increase in serum protein levels and urine protein levels substantiate this. Thus, the necrosis of renal tubules, loss of proteins through urine and its passing down the concentration gradient into the extracellular spaces as well as blood may be responsible for the reduction of protein levels.

The recovery in protein levels at a few time intervals may be due to the attempts of the kidney to regenerate the lost cells/tissue. Such incidents of recovery of protein levels appear to occur under the influence of low M.E. concentrations especially for longer exposure periods but such recovery is induced only for 0.1, 1.0 & 5.0% M.E. concentrations at one or two time intervals. Probably at these time intervals the protein synthesis might be stepped up but it needs further investigation to throw more light on this aspect.

V) UREA:

The studies on the kidney urea levels show that the mining effluents profoundly influence the urea levels and very significantly increase the urea concentrations. Mining effluents to a large extent, induce exposure period and concentration dependent rise in the kidney urea concentrations and in a few occasions the urea levels decline slightly but the urea never comes to the control level. These observations suggest that the M.E. promote enhancement of urea synthesis though the birds are basically uricotelic. Under the influence of M.E. the kidney undergoes necrosis and the lysosomal enzymes may be promoting degradation of proteins. These excess of proteins or their degraded products vis-a-vis the amino acid enter the circulations as evidenced by the rises in serum protein levels, and these may be taken to the liver for
deamination and transamination. During such processes, more ammonia may be getting produced which eventually may be detoxified by its conversion into urea and this excess urea may be passed on to the kidney for elimination. This hypothesis is supported by the observations of urea elevations in the serum, kidney and urine. But it needs further investigation to throw more light in it. It may be of great interest to know how exactly the protein metabolism is stepped up. It may be of interest to find out if M.E. promote rise in the Arginine levels and if the enzymes involved in the ornithine cycle escalate their activities. The slight decline in the urea level at a few time intervals may be due to the passing of urea into the urine and blood.

VI) URIC ACID:

The studies of the kidney uric acid levels indicate that very low concentrations of M.E. (0.01%) induce elevation of uric acid concentration if the animals are exposed to M.E. for a very long period. Similarly, 0.1 and 1.0% M.E. promote successive rise in uric acid concentrations from the end of fortyeight hours to the end of one twenty hours, while the 0.1, 1.0, 5.0 and 10.0% M.E. concentrations induce reduction (except at the end of twelve hours for 1.0%) in the uric acid level up to end of twenty four hours. The higher concentrations of M.E. induce elevations of uric acid levels. Though a perfect relationship between the kidney concentrations and the renal clearance is not established, some correlation is seen. When the kidney concentrations of uric acids are high under the influence of 0.1% M.E. at the end of fortyeight, seventy two and ninety six hours, the urine concentrations of uric acid are low. Similar situation is observed for 10.0% M.E. at the end of fortyeight and one twenty hours. But the lower concentrations of kidney uric acid and high renal clearance of uric acid are induced by : 0.1% and 1.0% M.E. at the end of six, twelve, twentyfour hours ; 5.0% M.E. at the end of all the time intervals under investigation ; 10.0% M.E. at the end of six, twelve, twentyfour and ninetysix
hours; 50.0% M.E. at the end of twelve hours. Interestingly, another type of correlation between kidney uric acid level and renal clearance (high kidney concentration and high renal clearance) is found under the influence of: 0.01% M.E. at the end of thirty and fortyfive days; 0.1% M.E. at the end of one twenty hours; 1.0% M.E. at the end of fortyeight, seventytwo, ninetysix and one twenty hours; 10.0% M.E. at the end of seventytwo hours; 50.0% M.E. at the end of six, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours and by 100% M.E. at the end of all the time intervals under investigation.

Therefore, the reduction in the kidney uric acid level could be partly due to the necrosis of the kidney leading to the exudations of cytoplasmic debris and passing out of uric acid through urine. The birds' kidneys are known to produce nearly twice as much uric acid as the liver (Chou, 1972) and hence, reduction of uric acid synthesis under necrotic conditions may partly be responsible for the decline in uric acid level. The increase in the uric acid concentrations may be due to the increased purine metabolism leading to uric acid synthesis by the kidney under the influence of mining effluents. Similarly, the liver uric acid synthesis may be increased under the influence of M.E. and the synthesised uric acid may be passed on to the renal tubules through blood for its elimination. This may partly elevate the kidney uric acid concentrations. But this needs further investigation to throw more light on this aspect.

VII) CREATININE:

The present investigation indicates that the mining effluents (M.E.) of all the concentrations tested induce increase in the creatinine concentrations of the kidney at all the time intervals under investigation (except 1.0% M.E. at the end of twentyfour and seventytwo hours). The relatively low concentrations of M.E. induce elevation of creatinine amounts when birds are exposed to them for longer periods while higher concentrations can induce sharp rise in creatinine as early as completion of six hours. The M.E. promote fluctuations in
the creatinine levels between the end of six hours and one hundred and twenty hours. In general the creatinine level appears to be high.

Creatinine represents the waste products of creatine metabolism and it arises in the body from the spontaneous break down of creatine phosphate. It serves practically no apparent function in the body. The creatinine level increases due to excessive tissue destruction releasing creatinine or due to failure of creatine being properly phosphorylated (Chatterjee, 1972).

In the present studies the elevation of creatinine in the renal tissue may be due to the tissue destruction promoted by mining effluents. Besides the destruction of renal tissue, the M.E. may be promoting destructions of other tissues in the body as well as may be interferring in the process of phosphorylation of creatine in the muscles which may step up the conversion of creatine to creatinine and this excess of creatinine may be passed on to the kidney for elimination. This may also promote the elevation of creatinine in the kidney up to certain extent. The fluctuations in the kidney creatinine may be related to the fluctuations in the destructions of renal tissues as well as other tissues promoted by the mining effluents. The elevation of kidney tissue creatinine in proportion to the renal necrosis substantiates Chatterjee's (1972) view of relations between the tissue damage and creatinine level. The acute reduction in creatinine level can be interpreted on the basis of its excessive elimination through urine or its passing down the concentration gradient into the extracellular space or blood as suggested by Merill et. al., (1956).

VIII) CALCIUM:

The M.E. of different concentrations induce concentration dependent elevation in the calcium levels of the kidney at the time intervals of six, twelve, and twenty four hours but for the subsequent periods such a relationship is not obtained. Only 0.1% M.E. induce reduction in the renal calcium level at the end of twentyfour hours while, rest of the M.E. concentrations promote increase
in calcium level above that present in the control animals. The mining effluents induce fluctuations in the calcium levels. The rise in the renal calcium level may be due to the necrosis which cause damage to several cytoplasmic organelle thereby releasing the calcium bound to the organelle and proteins. Besides this, the M.E. may be inducing damage to other organs of the animal, thereby releasing more calcium in the blood. This extracalcium may be passed on to the urine for excretion and may lead to the elevation and fluctuations of calcium. In addition if M.E. is inducing release of parathyroid hormone (PTH) then, under the influence of PTH the distal tubular reabsorption of calcium may be promoted which may increase the kidney retention of calcium.

Although the determinants of ionized calcium concentrations on the cytoplasm of cells are largely unknown, the cytosolic, ionized calcium concentration is known to change with changes in membrane calcium fluxes. In some mammalian cells, PTH has been shown to increase cellular calcium (Howard, 1989). In similar studies calcitonin is shown to induce renal cellular calcium content.

Therefore the elevations and fluctuations in the renal calcium contents may also be due to the changes in membrane calcium fluxes, PTH influence, as well as due to calcitonin. It is difficult to say which of the mechanism is contributing more to the elevation of calcium. At present it is safe to propose that the elevation in renal calcium contents may be partly due to necrosis causing release of bound calcium, partly by increased mobilization of calcium, by passing of calcium by blood to kidney by the action of PTH and calcitonin and partly due to the changes in membrane calcium fluxes. The M.E. may also influence another regulatory route of β - adrenergic which may activate phospholipase C and inositol triphosphate or the IP3 system which increases intracellular calcium. Therefore, this may lead to accumulation of Ca++. Depending upon the increase or decrease of the operation of any one
mechanism, the renal calcium contents may fluctuate and acute decline in a few of these mechanisms may reduce the renal calcium contents.

IX) CHLORIDE:

Under the influence of 0.1 and 1.0% M.E. the renal chloride contents reduce sharply while the higher concentrations of M.E. induce increase in the renal chloride contents. The 100% M.E. induce highest increase in the renal chloride contents. The renal chloride contents undergo some fluctuations as the exposure period increases but a perfect correlation between the M.E. concentration/exposure period and renal chloride contents is not seen. In general the chloride contents of the kidney increase under the influence of mining effluents barring few exceptions.

Several lines of evidence strongly suggest that Na⁺ and Cl⁻ are cotransported across the luminal membranes by a carrier (Ericsson and Spring, 1982; Frizzell et. al., 1979). The coupled carriers are capable of producing uphill transport of one partner and the other partner moves down hill through a large enough gradient (i.e. secondary active transport). The coupled carrier scheme suggests that Cl⁻ can be transported uphill into the cell. In the present investigation the M.E. may be promoting the enhancement of these coupled carriers which may then elevate the renal Cl⁻ contents. Probably under the influence of M.E. this coupled carrier system is activated which may lead to more transport of chloride ions. Now it is known that the second messengers in the sequence of regulatory events within the cell include cAMP and calcium ions. Cyclic AMP may activate a protein kinase that phosphorylates and opens closed chloride channels, thereby increasing the chloride transport. Therefore, mining effluents may be activating cAMP synthesis and activating protein kinases to promote phosphorylation and opening of chloride channels. The M.E. may also influence another regulatory route of β-adrenergic which may activate phospholipase C and inositol triphosphate, or the IP₃ system, which
increases intracellular calcium. A rise in intracellular calcium has been shown to open Cl⁻ and K⁺ channels (Sterling, 1989). In the present investigation, the kidney is shown to retain more calcium under the influence of mining effluents and this elevated calcium may open the chloride channels as suggested by Sterling (1989) leading to the elevation of chloride contents of the kidney. But it needs further investigation to throw more light on this aspect. The chloride contents may fluctuate in association with the rise and decline in the chloride transport mediated either by coupled carrier system or by cAMP activation of protein kinase or by raising intracellular calcium levels. In the present context as the calcium level is seen elevated in the kidney the calcium mediated transport of chlorides and its accumulation seems more logical. The reduction in kidney Cl⁻ contents may be due to its passing down the concentration gradient into blood and its passing down to the urine.

II) SECTION B - URINE ANALYSIS

ENZYMES : I) ACID PHOSPHATASE (AP):

It is now an established fact that enzymes appear in the urine after the renal insult, leading to the renal injury. Besides, variety of enzymes are eliminated through urine under pathological conditions developed due to renal impairments. In the present investigation the control animals do not excrete any enzyme or protein through urine indicating non existence of any renal damage.

From the present investigation it appears that the mining effluents induce enzyme-urea, a pathological state where enzymes and urea appear in the urine. The various concentrations of M.E. induce concentration dependent rise in the renal clearance of AP at the end of twelve, fortyeight and ninetysix
hours and the 50% M.E. promote the maximum clearance of AP at the end of fortyeight hours. The renal clearances of AP elevate progressively as the exposure periods increase, but at few times the AP activity gets reduced.

Normally, urine does not show presence of acid phosphatase. Only in pathological urines the lysosomal enzymes are known to occur. The variations in the renal clearances of acid phosphatase are dependent upon the degree of necrosis but a perfect correlation between the necrotic state and renal clearance is not evident. The lysosomes from the cytoplasm may come into the lumina along with the cytoplasmic exudents and then may pass into the urine. Thus, the acid phosphatase comes into the urine from the injured/damaged renal tubules. But this may not be the only source. The mining effluents are likely to induce injury to the alimentary tract, especially to the stomach intestine and also to the liver or muscles depending upon the depositions of heavy metals of the M.E. origin into them. The acid phosphatase of these tissue origin is likely to be passed on to the blood during circulation thereby elevating the blood AP. The animal body may tend to dispose off this excess AP which might promote serum protein degradation, through urine. Therefore, the urinary AP may also be due to the contribution of the injured tissues other than the kidney. Therefore, the elevation and the decrement of acid phosphatase from the serum and the injured organs may be affecting the renal clearance of AP. This may be the reason for fluctuations in urinary AP.

II) ALKALINE PHOSPHATASE (AIP):

All the concentrations of mining effluents induce concentration depended elevation in the renal clearance of AIP at the end of twelve and ninetysix hours, while 0.1 to 50% M.E. promote increase in urinary excretion of AIP in accordance with the rise in the M.E. concentration at the end of ninetysix hours. The renal clearance of AIP increases progressively as the exposure period increases except for a few occasions when the urinary excretion drops.
The renal clearance of AIP appears to be dependent upon the degree of necrosis. Thus, the progressive necrosis may be responsible for progressive renal clearance of AIP under the influence of M.E. During necrosis the tissue debris and the lysosomes are swept in to the tubules' lumina and hence the lysosomal enzymes like AIP are seen in the urine. Besides, the contribution of damaged renal tissue, the serum bearing elevated AIP may be contributing the renal clearance of AIP. The serum AIP elevations may be due to the other tissues undergoing necrotic changes under the influence of mining effluents. The decreases in the renal clearance of AIP may be due to the varying degree of necrosis of tissues which vary the serum AIP levels. The serum tends to eliminate AIP only when it rises above the tolerance level and if the AIP rise is not alarmingly high or less than the threshold level, the amount of AIP passing to the urine would naturally drop. The serum may be eliminating AIP to avoid induction of degenerative changes in the serum proteins under the influence of AIP. In short, the occurrence of AIP in the urine is a natural process under the development of pathological conditions in the animal body and may be considered as a protective mechanism and an indication of tissue damage, especially of a renal tissue damage and the amount of AIP in urine i.e. AIP activity in urine may be considered as a measure of renal pathological state.

III) NONSPECIFIC ESTERASE:

The M.E. induce concentration dependent renal clearance of nonspecific esterase at the end of twelve, twentyfour andforty eight hours and the 100% M.E. promotes highest renal clearance at the end of twelve hours, but in general, all the concentrations of M.E. promote increases in the urinary elimination of nonspecific esterase, though there are at some occasions rise and falls in the renal clearances of esterase.

Like other lysosomal enzymes, the appearances of esterase in the urine can be interpreted on the grounds of necrosis. The degree of necrosis decides
the quantity of esterase to be released in the urine but a perfect correlation in
the degree of necrosis in one organ and the renal clearance of esterase is not
always obtainable. In the present studies it is evident that the lysosomes from
the cytoplasm may be coming to the lumina of the tubules along with the
cytoplasmic debris and then may get passed on to the urine. Thus, the
esterase may be coming to the urine from the injured nephrons. Besides
kidney, the organs like alimentary tract and the liver are likely to get exposed to
the mining effluents which may induce necrosis in them depending upon the
concentration of M.E and the heavy metals deposited in the organs. Under
such necrotic conditions the lysosomal enzyme esterase may be released into
the serum and serum esterase may get elevated. This elevation of serum
esterase can induce more renal clearance of esterase. The fluctuations or
reductions in the renal eliminations of esterase may therefore depend upon the
necrotic states of the organs, the elevations of serum esterase and on the
passing down of the esterase against the concentration gradient in to the
extracellular spaces (Merill et. al., 1956). It is also known that the release of
enzymes into the blood and extracellular space correlates with the degree of
necrosis or the size of the area of infarction (Metkiewski et. al., 1968, Zelman
et. al., 1959). Therefore, the rise or fall in the urine esterase is likely to depend
upon the area of renal infraction, the level of serum esterase and the amount of
esterase being passed to the extracellular space.

IV) PROTEINS:

The mining effluents appear to induce protein-urea into the ducks as
indicated by the occurrence of protein into the urine. It appears that the
occurrence of proteins in the urine does not perfectly depend upon the
concentrations of M.E. as well as upon the exposure period. The urinary
elimination of proteins fluctuate considerably under the influence of mining
effluents. Normally the proteins are not excreted through urine and the ducks
do not excrete proteins through the urine as observed in the controls. The low concentrations of M.E. induce relatively higher elimination of proteins at the end of six hours, but when the necrosis extends to many tubules at the end of one twenty hours, they induce pronounced elimination of proteins, but the higher concentrations tend to eliminate more proteins in the early exposure periods as they promote acute necrosis very early and at the later hours, comparatively less proteins are eliminated.

Palmela et. al., (1966) have shown that the kidney is highly unselective in the excretion of protein molecules in acute ischemic renal failure. Karlwilhelm et. al., (1977) have shown that protein-urea exists in nephrotoxic nephritis. Beregi et. al., (1962) have shown that 5-HT induce proteinemia and have shown proteins in Bowman’s capsule and basement membrane. Skjorten and Fredrik (1964) have reported fibrin precipitation in glomeruli in cases of bilateral renal necrosis. Mandel and Popper (1971) reported protein-urea in ischemic necrosis. Maiorca et. al., (1967) reported protein-urea with nephrotic syndrome and other chronic kidney diseases with or without uremia. Kashgurian and (1977) have mentioned that tubular disorders induce protein-urea.

The protein-urea induction may be due to the failure to reabsorb protein, normally filtered by glomeruli, possibly complicated by a leak of plasma proteins directly into the tubular lumen and failure of their reabsorption (Palmela et. al., 1966). In the present investigation the cytoplasmic debris is found in the lumina of tubules and the tubules also bear damaged brush and luminal border. Under such conditions the tubules would fail to reabsorb the proteins from the debris, as a result the proteins may enter into the urine. The quantity of protein occurrence in the urine may then fluctuate with the degree of necrosis and partial reabsorption of proteins by some tubules. Further Carohe et. al., (1979) have observed that mammalian renal tubules play an important role in the metabolism of proteins and peptides. Only the proximal tubules
possess the mechanism of degradation or transport of these substances and reabsorption of metabolic products. Since these tubules undergo necrosis under the influence of M.E., such protein metabolism may be getting disturbed resulting in the excretion of proteins through urine. Besides, the M.E. induced variations in the urine outputs which may partly influence the renal elimination of proteins. The fluctuations in the renal clearances of proteins may also be due to the variations in the serum protein levels, variations in the degree of necrosis of other organs like alimentary canal and liver and also due to the variations in absorptions of the proteins by the intact tubules which have escaped damage.

Prasad Rao et al., (1989) have also reported changes in the kidney proteins but unfortunately there is no single report on the elimination of proteins through urine by birds under the influence of heavy metals or mining effluents. Desai (1983) has reported renal clearance of proteins by fowls under the conditions of renal necrosis. Thus, the appearance of proteins in the urine may be considered due to the renal impairment or renal necrosis by the M.E.

V\text{N\text{U}}\text{REA :}

The renal clearance of urea is profoundly influenced by the mining effluents and the renal clearance of urea shows M.E. concentration dependency at the end of twelve hours and such a relationship is not introduced by M.E. at any other exposure period under study. But interestingly the mining effluents (M.E.) induce a pathological condition called as polyurea at all the time intervals under study. The M.E. induces a progressive polyurea up to certain extent as at some time intervals the renal clearances drop.

About 40% of the filtered urea is absorbed in the proximal convoluted tubule and when ADH levels are high, water is absorbed from the collecting tubules resulting in the rise in urea concentration. Under the influence of M.E. the kidney undergoes necrosis and depending upon the concentration of M.E.
and the exposure period the necrosis progresses. The proximal tubules are
seen to get injured. Therefore, the necrotic proximal tubules may not be able
to perform the absorption of urea thereby increasing the urea concentrations in
the tubular fluid which may be passed to the collecting tubules. The collecting
tubules may be attempting the absorption of water and electrolytes under the
influence of ADH which may further increase the urea concentration leading to
the excessive release of urea in the urine to develop a condition called
polyurea. The drops in the renal clearance of urea may be partly due to the
improved absorption of urea by the intact proximal tubules and partly due to the
passing of urea in to the extracellular space and blood. The alterations in
serum urea levels are also likely to influence the renal clearance of urea.

Brulles et. al., (1969) have reported that increase in GFR increases
urea clearance. Park et. al., (1969) have shown the reduction in urea excretion
in relation to fall in GFR. Niesel et. al., (1970) have shown the inter
dependence of urea, NaCl and KCl excretion. In the present study the renal
clearance of urea is high though the rate of urine flow and GFR are reduced
under the influence of mining effluents. This is in contrast to the observations
of Brulles et. al., (1969) and Park et. al., (1969). Therefore, the reasons for the
polyurea may be the necrosis of proximal tubules and absorption of more water
by the collecting tubules, as suggested earlier.

UB) URIC ACID:

The mining effluents (M.E.) induce wide fluctuations in the renal
 clearance of uric acid. The 5.0% M.E. promotes highest renal clearance of uric acid at the end of ninetysix hours while, 10.0% M.E. promotes lowest renal clearance of uric acid at the end of fortyeight hours. Generally, the higher concentrations of M.E. induce increase in the renal clearance of uric acid but in few-instances the uric acid clearances fall below the control levels. The increase in the renal clearance of the uric acid may be due to the increase in
the kidney uric acid concentrations caused by the probable stepping up of the renal uric acid synthesis. At the same time increase in the synthesis of uric acid by the liver due to increased purine metabolism coupled with rise in serum uric acid level may induce increase in the renal clearance of uric acid under the influence of high M.E. concentration. The fluctuations in the uric acid synthesis and the uric acid levels of the kidney may promote fluctuations in the renal clearance of uric acid.

VI) CREATININE:

The mining effluents induce increase in the renal clearance of creatinine. M.E. induces concentration dependent enhancement in the renal clearance of creatinine at the end of six and twelve hours, but, generally the M.E. concentration induce increase in the renal clearance of creatinine as the exposure period increases but a perfect correlation is not obtained. M.E. induces wide fluctuations in the renal clearances of creatinine.

The enhancement of renal clearance of creatinine may be due to the increase in the renal creatinine level and also due to the increased serum creatinine level. The fluctuations in the renal creatinine clearances may depend upon the fluctuations in the serum creatinine level as well as kidney creatinine levels. Indirectly the renal creatinine level would also depend upon the alterations in the degree of muscular dystrophy brought about by the M.E. and also on the failures of phosphorylations of creatine. As the creatinine represents the waste products of creatine metabolism, the enhancement of creatine metabolism may elevate the renal clearance of creatinine and the fluctuations in creatine metabolism may fluctuate the renal clearance. But it needs further investigation to find out how M.E. induces alterations in the creatine metabolism. But it is clear that M.E. are inducing creatinuria in the ducks. The induction of creatinuria is a pathological symptom, indicating the animals are under stress. Rennick (1967) has shown that creatinine is
secreted by the renal tubules and some of it is absorbed by the tubules. But the damaged tubules would fail to reabsorb the creatinine. As a result the creatinine clearance in urine may significantly increase. Therefore, the renal clearance may be stepped up.

VII) CALCIUM:

The 100% M.E. induces highest renal clearance of calcium at the end of twenty four hours while relatively low but higher than control level, renal clearance is induced by 1.0% M.E. at the end of six hours. However, all the concentrations of M.E. promote enhanced renal clearance of calcium. The M.E. induce wide fluctuations in the renal clearance of calcium.

The enhancement in the renal clearance of calcium may be due to the renal necrosis. The M.E. induces renal necrosis and the degree of necrosis partly depends upon the concentration of M.E. and the exposure period. During necrosis the tissue is damaged, the cytoplasmic debris is exuded in the tubules' lumina and the ultra structural studies have revealed the breakdown of mitochondria, proliferations of Golgi bodies and E.R. into the tubules' lumina. Therefore, the calcium present in the cytoplasmic debris, calcium bound to mitochondria and other organelles may be swept into the tubules from where it may be passed down to the urine thereby increasing the urine calcium level.

Besides this the elevations of serum calcium levels may also induce increase in the renal clearance of calcium. The fluctuations in the renal clearance of calcium may be due to the fluctuations in the serum calcium levels and changes in the degree of necrosis as well as due to the variations in the calcium absorptions by the intact renal tubules which have escaped damage.

VIII) CHLORIDES:

The mining effluents (M.E.) appear to induce enhancement of renal clearance of chlorides as well as wide fluctuations in the urinary
concentrations. The higher concentrations of M.E. appear to induce more renal clearances of chlorides though a perfect correlation between the high concentrations of M.E. and elevated renal clearances are difficult to attain. But at the end of ninetysix hours and partially at the end of one twenty hours a nearly perfect correlation is reached. The 100% M.E. induce the highest clearance of chlorides.

The elevated renal clearances of chlorides could be due to the elevated renal concentrations of chlorides and partly also due to the elevations in chloride contents of the serum. The fluctuations in the chloride contents may be due to the fluctuations in the renal and serum chloride levels. Besides this, the variations in the degree of necrosis would also contribute to the elevations and fluctuations in the urinary chloride contents. During necrosis the cytoplasmic debris is swept into the lumina and the chloride contents of the debris may get passed on to the urine thereby increasing the urinary chloride levels. Besides, the intact renal tubules especially collecting tubules may try to reabsorb some of the chlorides and these reabsorptions of chlorides may partly contribute to the fluctuations in renal clearances of chloride. However, the reabsorption of chlorides by the intact collecting tubules & renal apparatus under elevated serum chlorides and renal chloride levels would be very negligible and even may not take place but under the circumstances, drop in chloride levels of renal tissues and serum levels being not so high, the collecting tubules may absorb more chlorides which may partially change the renal clearance of chlorides. But largely the changes in the renal clearances of chlorides would depend upon the degree of necrosis, accumulation of calcium ions which are known to induce chloride accumulations in renal tissues and elevations or depletions of serum chloride levels.
IX) ELECTROLYTES:

A) Na⁺

The mining effluents profoundly influence the renal clearance of Na⁺ and M.E. induce elevated renal clearance at all the exposure periods under study. The low concentrations of M.E. induce slightly elevated renal clearance at the end of six and twelve hours while the highest renal clearance of Na⁺ is promoted by 100% M.E. at the end of seventy two hours. The M.E. also induce wide fluctuations in the renal clearance of Na⁺. Carone et al., (1979) have observed that the renal disease in animals and in man or in renal disorders with comparable or greater reduction in GFR, Na⁺ retention does not occur and fractional excretion of Na⁺ in urine is increased in proportion to the reduction in GFR. In the present studies the reduction in GFR and reduced urine outputs are observed under the influence of mining effluents. Therefore, reduction in GFR and urine output under the influence of M.E. may be inducing failures of Na⁺ retentions, thereby promoting more renal clearance of Na⁺. Besides this, the damage of the proximal and distal tubules may decrease the absorption of Na⁺, which may lead to an increase in the Na⁺ excretion. Thus, the tubular necrosis may be responsible for high Na⁺ clearance (Kashgurian et al., 1977). Laurence and Friedler (1966) reported that angiotensin induced vasodilation resulted in marked increase in Na⁺ excretion and decrease in Na⁺ reabsorption. It would be of immense interest to find out if M.E. induce the angiotensin mediated Na⁺ excretion. During necrosis the juxta glomerular apparatus is also getting injured, as a result the angiotensin production may be decreasing which possibly may induce increase in the renal clearance of Na⁺. In addition to this, the acute increase in the serum Na⁺ levels may also contribute to the elevation of the renal clearance of Na⁺. The fluctuations in the renal clearances of Na⁺ may be due to the fluctuations in the serum Na⁺.
levels, variations in the degree of necrosis at different time intervals and also due to the variations in the GFR as well as urine output.

B) K⁺:
The mining effluents (M.E.) induce rise in the renal clearance of K⁺. The renal clearance of K⁺ is concentration dependent at the end of fortyeight hours and the 100% M.E. promote relatively very high renal clearance of K⁺. But in general, though the renal clearance of K⁺ is higher than the control, the K⁺ clearance fluctuates widely under the influence of M.E. Giebisch and Gerhead (1965) reported that K⁺ secretion of the distal tubules is influenced by four factors: the extracellular K⁺ concentration, the electrical gradient across the luminal tubular membrane, the strength of the active transport process reabsorbing K⁺ and the K⁺ permeability of the luminal cell membrane. The renal distal tubules undergo necrosis under the influence of mining effluents and the necrotic tubules lose the luminal borders which then may fail to reabsorb the K⁺. Therefore, the renal clearance of K⁺ may be getting enhanced. Thus, this may be the cause of higher renal clearance of K⁺. Higher the degree of necrosis, higher may be the renal clearance of K⁺. The fluctuations in the renal K⁺ clearance may be due to the variations in the degree of necrosis and the number of tubules involved in the necrosis. Besides this the elevations in the serum K⁺ levels may also promote the increase in the K⁺ clearance through urine and the rises and falls in the serum K⁺ levels may induce alterations in the renal clearances of K⁺. Besides this, the decrease in active transport as a result of ATP depletion due to necrosis and damaged mitochondria, would result in excessive renal clearance of K⁺.

X) CHANGES IN URINE FLOW AND GFR:
The mining effluents induce alterations in the urine flow and GFR. The different concentrations of M.E. induce reduction in urine flow and these
reductions are more over proportionate to the M.E. concentrations and exposure period. The rate of urine flow partly depends upon the state of dehydration and partly on the arterial pressure / pressure in glomerular capillaries (Sturkie, 1976). In the present studies, the mining effluents induce damage to the glomeruli, proximal, distal and collecting tubules. The necrosis of glomeruli would induce decrease in glomerular capillary blood pressure which may reduce the rate of urine flow. Similarly, the renal tubular necrosis may develop inability in the tubules to reabsorb water, especially in the collecting tubules, as a result there would be more loss of water and this loss of water and failure to reabsorb water may eventually induce dehydration. Therefore, after necrosis, the rates of urine flows may drop due to the decreased glomerular filtration and dehydration. Besides this, the macula densa also regulate glomerular capillary pressure through afferent arterioles (Sullivan and Grantham, 1982). In the present studies the macula-densa also appears to be damaged, hence it may not be able to regulate the glomerular capillary blood pressure. The fluctuations in the urine flow may be due to the variations in the degree of necrosis at different time intervals for various concentrations of mining effluents.

The mining effluents (M.E.) induce reductions in the Glomerular Filtration Rates (GFR) in proportion to the concentrations of M.E. The M.E. also induces fluctuations in the GFR at some time intervals. The decrease in GFR under the influence of M.E. may be due to the decrease in urine flow and dehydration (Sturkie, 1976; Skadhauge and Schmidt - Nielson, 1967). GFR reduction in quail has been attributed to a reduction in the number of filtering reptilian nephrons coupled with drop in urine flow (Sturkie, 1976). In the present investigation the M.E. induces necrosis of both the reptilian and mammalian tubules along with the glomeruli. Therefore, the number of intact filtering nephrons of both the reptilian and mammalian types get reduced which may reduce the GFR. Shoemaker (1967) proposed that GFR in bird reduces
with decrease in urine flow. And this could be the reason for drop in GFR of ducks under the influence of M.E. as M.E. reduces urine flow.

Macula densa cells of the juxta glomerular apparatus sense the rate of fluid flow through the adjacent distal tubules and signal the afferent arteriole to constrict (Sullivan and Grantham, 1982). In the present investigation M.E. induces injury to the macula densa of the juxta glomerular apparatus and the damaged macula densa may fail to sense the rate of fluid flow through the adjacent tubules and hence may fail to signal the afferent arteriole. This may result in the alterations in GFR. Therefore some fluctuations in GFR may be due to the variations in the number of glomeruli and macula densa getting involved in the necrosis. Besides this angiotensin may also be responsible for alterations in GFR up to certain extent as angiotensin is known to be associated with GFR (Sturkie, 1976). But it needs further investigation to find out if M.E. alters the angiotensin levels.

RETENTION PATTERNS:

A] KIDNEY:

The mining effluents induce retentions of cadmium, chromium, copper, iron, lead, magnesium, manganese, nickel and zinc in the kidney. The ultrastructural studies have also supported the retentions of heavy metals. All the metals are retained maximally at specific time intervals and mostly the heavy metal retentions depend upon the M.E. concentrations and the exposure periods. At the end of six hours cadmium, chromium, iron, and magnesium are retained maximally under the influence of M.E. While at the end of twelve hours cadmium, chromium, copper, iron, magnesium, manganese and zinc are retained significantly high. Twenty four hours exposure promotes maximum retentions of cadmium, iron (for 0.1% M.E. only), magnesium and manganese
IV) ESTERASE (NON SPECIFIC):

The alterations in the serum nonspecific esterase are compiled in Table No. 33 and are graphically expressed in Graph No. 33. The non specific esterase activity is expressed as μmoles per hundred millilitre of serum. The control birds exhibited nonspecific esterase activity equivalent to 560.0 ± 4.10 μmoles per hundred millilitre of serum. From the table and the graph it is obvious that the serum nonspecific esterase activity increased in a mining effluent (M.E.) concentration dependent manner for all concentrations except 5% M.E., at the end of six hours. Similarly, the M.E. concentration dependent elevation of esterase (non specific) activity was induced at the end of ninety-six and one hundred and twenty hours by the concentrations ranging from 0.1 to 50.0 percent. Besides, the M.E. concentrations like as 0.1 and 5.0 percent induced elevation of nonspecific esterase activity in relation to the exposure period, up to the end of ninety-six hours.

The ducks exposed to 0.01% mining effluents (M.E.) did not exhibit any significant alteration in the non specific esterase activity up to the end of twenty nine days. But by end of thirty days the serum nonspecific esterase activity increased a little and continued to increase further up to the end of fortyfive days. Thus, at the end of thirty and fortyfive days the nonspecific esterase activity was equivalent to 630.0 ± 2.135 and 750.0 ± 2.607 μmoles per 100 ml respectively.

Under the influence of 0.1% M.E. the serum nonspecific esterase activity increased significantly at the end of six hours and then exhibited exposure time dependent elevation up to the end of ninety six hours. By the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one hundred and twenty hours the serum nonspecific esterase activity was equivalent to 820.0 ± 1.414, 855.0 ± 3.370, 900.0 ± 6.752, 950.0 ± 2.059, 1020.0 ± 6.887, 1100.0 ± 8.795 and 1090.0 ± 3.878 μmoles per 100 ml.
On exposing the ducks to 1.0% M.E. the nonspecific esterase activity increased sharply at the end of six hours and then continued to increase up to the end of fortyeight hours and then dropped a little at the end of seventytwo hours. Then, once again the esterase (non specific) activity increased at the end of ninetysix hours and continued to increase up to the end of one hundred and twenty hours. Thus, at the end of six, twelve, twentyfour, foryeight, seventytwo, ninetysix and one hundred and twenty hours, the non specific esterase activity was equivalent to 950.0 ± 3.49, 980.0 ± 2.60, 1020.0 ± 3.20, 1230.0 ± 10.0, 1050.0 ± 1.85, 1150.0 ± 2.315 and 1190.0 ± 4.019 μmoles per 100 ml respectively.

The 5% M.E. induced progressive elevation in the esterase activity from the end of six hours to the end of ninetysix hours indicating exposure time dependency of the hike in the enzyme activity. By the end of six, twelve, twentyfour, foryeight, seventytwo, ninetysix and one hundred and twenty hours the serum non specific esterase activity was equivalent to 940.0 ± 4.534, 960 ± 6.73, 1040 ± 1.414, 1050 ± 4.242, 1170.0 ± 2.449, 1380.0 ± 2.416 and 1280.0 ± 4.147 μmoles per 100 ml respectively.

The ducks treated with 10.0% M.E. exhibited almost two folds increase in the esterase activity at all the time intervals under study except twentyfour hours. The non specific esterase activity showed progressive increase from the end of foryeight hours to one hundred and twenty hours. At the end of one hundred and twenty hours the esterase activity was maximum for this concentration of M.E. By the end of six, twelve, twentyfour, foryeight, seventytwo, ninetysix and one hundred and twenty hours, the serum esterase (non specific) activity was equivalent to 1190.0 ± 3.20, 1150.0 ± 4.127, 980.0 ± 4.56, 1190.0 ± 3.033, 1330.0 ± 4.534, 1390.0 ± 2.416 and 1470.0 ± 3.929 μmoles per 100 ml respectively.

On exposure to 50.0% M.E. the ducks showed acute increase in the non specific esterase activity at the end of six hours and then the enzyme activity
steadily decreased up to the end of twentyfour hours. The maximum increase in the esterase activity was observed at the end of one hundred and twenty hours for this M.E. concentration. By the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one hundred and twenty hours, the serum esterase (non specific) was equivalent to 1360.0 ± 3.20, 1290.0 ± 4.36, 1190.0 ± 3.162, 1540.0 ± 5.16, 1320.0 ± 4.04, 1640.0 ± 4.069 and 1670.0 ± 1.414 μmoles per 100 ml respectively.

The 100% M.E. induced the highest increase in the esterase activity at the end of twelve hours and then subsequently the esterase activity went on decreasing up to the end of ninety six hours. By the end of six, twelve, twenty four, fortyeight, seventytwo, ninetysix and one twenty hours the serum non specific esterase activity was equivalent to 1540.0 ± 4.223, 2040.0 ± 3.929, 1720.0 ± 3.611, 1680.0 ± 3.464, 1420.0 ± 2.856, 1070.0 ± 4.223 and 1300.0 ± 3.26 μmoles per 100 ml respectively.
TABLE NO. 33: ALTERATIONS IN THE SERUM ESTERASE (NON SPECIFIC) ACTIVITY UNDER THE INFLUENCE OF MINING EFLUENTS.

<table>
<thead>
<tr>
<th></th>
<th>6 Hours</th>
<th>12 Hours</th>
<th>24 Hours</th>
<th>48 Hours</th>
<th>72 Hours</th>
<th>96 Hours</th>
<th>120 Hours</th>
<th>720 Hours</th>
<th>1080 Hours</th>
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<tbody>
<tr>
<td>Control</td>
<td>560.0 ± 4.10</td>
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<tr>
<td>0.01% M.E.</td>
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<td></td>
<td>630 ± 2.135</td>
<td>750 ± 2.607</td>
</tr>
<tr>
<td>0.10% M.E.</td>
<td>820 ± 1.414</td>
<td>855 ± 3.370</td>
<td>900 ± 6.752</td>
<td>950 ± 2.059</td>
<td>1020 ± 6.887</td>
<td>1100 ± 8.795</td>
<td>1090 ± 3.878</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td>1% M.E.</td>
<td>950 ± 3.49</td>
<td>980 ± 2.60</td>
<td>1020 ± 3.20</td>
<td>1230 ± 10.0</td>
<td>1050 ± 1.85</td>
<td>1150 ± 2.315</td>
<td>1190 ± 4.019</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td>5% M.E.</td>
<td>940 ± 4.534</td>
<td>960 ± 6.73</td>
<td>1040 ± 1.414</td>
<td>1050 ± 4.242</td>
<td>1170 ± 2.449</td>
<td>1380 ± 2.416</td>
<td>1280 ± 4.147</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td>10% M.E.</td>
<td>1190 ± 3.20</td>
<td>1150 ± 4.127</td>
<td>980 ± 4.56</td>
<td>1190 ± 3.033</td>
<td>1330 ± 4.534</td>
<td>1390 ± 2.416</td>
<td>1470 ± 3.929</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td>50% M.E.</td>
<td>1360 ± 3.20</td>
<td>1290 ± 4.36</td>
<td>1190 ± 3.162</td>
<td>1540 ± 5.16</td>
<td>1320 ± 4.04</td>
<td>1640 ± 4.069</td>
<td>1670 ± 1.414</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td>100% M.E.</td>
<td>1540 ± 4.223</td>
<td>2040 ± 3.929</td>
<td>1720 ± 3.611</td>
<td>1680 ± 3.464</td>
<td>1420 ± 2.856</td>
<td>1070 ± 4.223</td>
<td>1300 ± 3.26</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
</tbody>
</table>

Note: Unit: μmoles/100 ml.
N.D.: Not Determined.
All alterations are statistically significant - P < 0.01.
GRAPH NO. 33: SERUM - ESTERASE.
Units: μmoles/100ml.

- Control
- 6 hours
- 12 hours
- 24 hours
- 48 hours
- 72 hours
- 96 hours
- 120 hours
- 720 hours
- 1080 hours
V) ALTERATIONS IN SERUM UREA:

The alterations in the serum urea concentrations are compiled in Table No.34 and are graphically expressed in Graph No. 34. The serum urea concentrations are expressed as milligrams per 100 ml of serum. The control birds showed the urea concentration equivalent to 6.3 ± 1.0 mg per 100 ml. From the table and the graph it appears that all the concentrations of mining effluents (M.E.) induced rise in the serum urea level. At the end of ninety six hours the M.E. concentration dependent rise in the serum urea level was observed. Similarly, the M.E. concentration dependent elevation in serum urea concentration was noticed at the end of six hours except for 50% M.E. It was observed that the higher concentrations of M.E. induced higher elevation in serum urea at many occasions. It is also seen from the graph that 50% M.E. promoted exposure period dependent rise in the serum urea level and a similar correlation was caused by 100% M.E. up to the end of ninetysix hours.

When the ducks were exposed to 0.01% M.E., there was no significant change in the serum urea level up to the end of twenty nine days but at the end of thirty days the serum urea level elevated significantly and continued to increase further up to the end of forty five days. Thus, at the end of thirty and forty five days the serum urea concentrations was equivalent to 8.20 ± 0.93 and 11.30 ± 1.27 mg per 100 ml.

The ducks subjected to 0.1% M.E. showed exposure time dependent increase in the serum urea concentrations up to the end of seventy two hours. By the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one hundred and twenty hours the serum urea level was equivalent to 7.0 ± 0.629, 7.50 ± 0.791, 8.90 ± 0.932, 9.50 ± 1.24, 10.0 ± 1.30, 7.90 ± 0.91 and 8.30 ± 0.87 mg per 100 ml respectively.

Under the influence of 1.0% M.E. the ducks exhibited fluctuations in the serum urea level. The serum urea level fluctuated between 7.90 ± 0.89, and 10.20 ± 1.07 mg per 100 ml. By the end of six, twelve, twentyfour, fortyeight,
seventy two, ninetysix and one hundred and twenty hours, the serum urea level was equivalent to 8.90 ± 0.73, 8.50 ± 0.60, 7.90 ± 0.89, 9.0 ± 0.937, 9.30 ± 1.32, 10.20 ± 1.07 and 8.90 ± 1.74 mg per 100 ml respectively.

On exposure to 5.0% M.E. the serum urea concentrations of the ducks fluctuated and the fluctuations ranged between 9.20 ± 1.60 and 12.20 ± 0.70 mg per 100 ml. The serum urea concentrations were equivalent to 9.20 ± 1.6, 10.30 ± 2.10, 9.70 ± 1.356, 11.30 ± 1.40, 10.90 ± 1.0, 12.20 ± 0.70 and 11.90 ± 2.0 mg per 100 ml at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one hundred and twenty hours respectively.

The 10.0% M.E. induced exposure period dependent rise in serum urea level from the end of six hours to the end of twentyfour hours as well as from the end of seventy two hours to the end of one twenty hours. The serum urea level was equivalent to 9.70 ± 0.90, 10.90 ± 0.70, 11.70 ± 1.80, 11.00 ± 1.10, 12.70 ± 1.60, 13.00 ± 1.01 and 13.70 ± 0.80 mg per 100 ml at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one hundred and twenty hours respectively.

The 50.0% M.E. promoted exposure period dependent rise in serum urea concentrations. The acute rise in serum urea level was observed at the end of one twenty hours. By the end of six, twelve, twentyfour, fortyeight, seventytwo, ninety six and one hundred and twenty hours the serum urea level was equivalent to 8.90 ± 1.78, 10.30 ± 1.62, 11.30 ± 1.20, 13.70 ± 0.80, 14.30 ± 2.10, 15.70 ± 1.40 and 15.90 ± 1.10 mg per 100 ml respectively.

The ducks treated with 100% M.E. showed exposure period dependent elevations in the serum urea level up to the end of ninetysix hours only. The highest increase in the serum urea level was noticed at the end of ninetysix hours only. Thus, by the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours, the serum urea concentration was equivalent to 10.70 ± 1.16, 11.00 ± 1.01, 12.70 ± 1.62, 13.40 ± 1.35, 14.70 ± 1.70, 16.90 ± 1.85 and 15.40 ± 1.40 mg per 100 ml respectively.
TABLE NO. 34: ALTERATION IN THE SERUM UREA UNDER THE INFLUENCE OF MINING EFFLUENTS.

<table>
<thead>
<tr>
<th></th>
<th>6 Hours</th>
<th>12 Hours</th>
<th>24 Hours</th>
<th>48 Hours</th>
<th>72 Hours</th>
<th>96 Hours</th>
<th>120 Hours</th>
<th>720 Hours</th>
<th>1080 Hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>6.30 ±1.0</td>
<td></td>
<td></td>
<td></td>
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<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>0.01% M.E.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>8.20 ±0.93</td>
<td>11.30 ±1.27</td>
<td></td>
</tr>
<tr>
<td>0.1% M.E.</td>
<td>7.0 ±0.629</td>
<td>7.50 ±0.791</td>
<td>8.90 ±0.932</td>
<td>9.50 ±1.24</td>
<td>10.0 ±1.3</td>
<td>7.90 ±0.91</td>
<td>8.30 ±0.87</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td>1% M.E.</td>
<td>8.90 ±0.73</td>
<td>8.50 ±0.6</td>
<td>7.90 ±0.89</td>
<td>9.0 ±0.937</td>
<td>9.30 ±1.32</td>
<td>10.20 ±1.07</td>
<td>8.90 ±1.74</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td>5% M.E.</td>
<td>9.20 ±1.6</td>
<td>10.30 ±2.1</td>
<td>9.70 ±1.356</td>
<td>11.30 ±1.4</td>
<td>10.90 ±1.0</td>
<td>12.20 ±0.70</td>
<td>11.90 ±2.0</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td>10% M.E.</td>
<td>9.70 ±0.9</td>
<td>10.90 ±1.2</td>
<td>11.70 ±1.1</td>
<td>11.0 ±1.6</td>
<td>12.70 ±1.1</td>
<td>13.0 ±1.01</td>
<td>13.70 ±0.8</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td>50% M.E.</td>
<td>8.90 ±1.78</td>
<td>10.30 ±1.62</td>
<td>11.30 ±1.2</td>
<td>13.70 ±0.8</td>
<td>14.30 ±2.1</td>
<td>15.70 ±1.4</td>
<td>15.90 ±1.1</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td>100% M.E.</td>
<td>10.70 ±1.16</td>
<td>11.0 ±1.01</td>
<td>12.70 ±1.62</td>
<td>13.40 ±1.35</td>
<td>14.70 ±1.7</td>
<td>16.90 ±1.85</td>
<td>15.40 ±1.4</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
</tbody>
</table>

Note: Unit: Urea in mg/dl.
N.D.: Not Determined.

All alterations are statistically significant - P < 0.01.
GRAPH NO. 34: CHANGES IN THE SERUM UREA LEVEL IN DUCKS UNDER THE INFLUENCE OF MINING EFFLUENTS.
Units: mg %

[Graph showing changes in serum urea levels in ducks over time with different concentrations of effluents.]
VI) ALTERATIONS IN URIC ACID:

The alterations in the serum uric acid levels of the ducks under the influence of mining effluents (M.E.) are compiled in Table No. 35 and are graphically presented in Graph No. 35. The serum uric acid levels are expressed as milligrams per hundred millilitre of serum. The control animals showed the serum uric acid concentration equivalent to 9.0± 0.37 mg per 100 ml.

From the graph and the table it is obvious that the mining effluents (M.E.) induced wide and significant fluctuations in the serum uric acid levels. The highest increase in the serum uric acid level was induced by 100% M.E. at the end of ninety six hours while the highest reduction in the uric acid level was promoted by 5.0% M.E. at the end of seventy two hours.

When the ducks were exposed to 0.01% M.E there was no significant change in the serum uric acid concentration up to the end of twentynine days but at the end of thirty days the serum uric acid level reduced significantly and then subsequently increased at the end of forty five days. Thus, the serum uric acid level was equivalent to 6.90 ± 0.80 and 10.90 ± 0.53 mg per 100 ml at the end of thirty and forty five days respectively.

The exposure of ducks to 0.1% M.E. induced acute reduction in the serum uric acid level at the end of six hours and subsequently the uric acid level steadily increased up to the end of twentyfour hours. But at the end of fourtyeight hours the serum uric acid level decreased once again and then showed a steady increase up to the end of one hundred and twenty hours. Thus, at the end of six, twelve, twentyfour, fourtyeight, seventy two, ninetysix and one twenty hours the serum uric acid level was equivalent to 3.30 ± 0.24, 5.10 ± 0.39, 5.60 ± 0.43, 3.70 ± 0.11, 4.30 ± 0.17, 4.90 ± 0.25 and 5.50 ± 0.342 mg per 100 ml respectively.

Under the influence of 1.0% M.E. the ducks exhibited significant fluctuations in the uric acid level and the serum uric acid level fluctuated
between 2.90 ± 0.377 and 8.0 ± 0.271 mg per 100 ml. By the end of six, twelve, twentyfour, foryeight, seventytwo, ninetysix and one hundred and twenty hours the serum uric acid level was equivalent of 4.80 ± 0.17, 7.40 ± 0.43, 5.50 ± 0.215, 4.50 ± 0.34, 2.90 ± 0.377, 5.40 ± 0.13, and 8.0 ± 0.271 mg per 100 ml respectively.

The ducks subjected to 5.0% M.E. exhibited the highest reduction in the uric level at the end of seventy two hours. The serum uric acid level fluctuated between 2.20 ± 0.282 and 5.30 ± 0.13 mg per 100 ml. By the end of six, twelve, twenty four, foryeight, seventy two, ninetysix and one twenty hours the serum uric acid level was equivalent to 4.0 ± 0.13, 2.80 ± 0.24, 5.30 ± 0.13 4.30 ± 0.37, 2.20 ± 0.282, 4.50 ± 0.24 and 3.00 ± 0.135 mg per 100 ml respectively.

The exposure of ducks to 10.0% M.E. promoted considerable fluctuations in the serum uric acid level. The serum uric acid level fluctuated between 4.20 ± 0.30 and 10.10 ± 0.17 mg per 100 ml. The serum uric acid level was equivalent to 4.20 ± 0.30, 4.80 ± 0.53, 5.40 ± 0.303, 4.50 ± 0.146, 10.10 ± 0.17, 5.80 ± 0.44 and 6.50 ± 0.70 mg per 100 ml at the end of six, twelve, twentyfour, foryeight, seventytwo, ninetysix, and one twenty hours respectively.

The 50.0% M.E. induced reduction in the serum uric acid level at the end of six hours and subsequently the serum uric acid level increased steadily up to the end of seventy two hours. At the end of twenty hours the serum uric acid level elevated sharply. Thus, the serum uric acid concentration was equivalent to 5.90 ± 0.80, 7.30 ± 0.426, 8.20 ± 1.78, 9.10 ± 1.85, 10. 172 ± 1.41, 8.70 ± 1.01 and 12.732 ± 2.0 mg per 100 ml at the end of six, twelve, twentyfour, foryeight, seventytwo, ninetysix and one twenty hours respectively.

On exposing the ducks to 100.0% M.E., the serum uric acid level fluctuated between 8.40 ± 0.462 and 14.60 ± 1.32 mg per 100 ml. And the 100% M.E. promoted the highest increase in serum uric acid level at the end of ninety six hours. By the end of six, twelve, twentyfour, foryeight, seventytwo,
ninety-six and one twenty hours, the serum uric acid level was equivalent to 9.70 ± 0.271, 8.40 ± 0.462, 10.70 ± 1.56, 13.20 ± 1.16, 12.60 ± 1.62, 14.60 ± 1.32 and 9.80 ± 1.01 mg per 100 ml respectively.
TABLE NO. 35: ALTERATIONS IN THE SERUM URIC ACID LEVEL UNDER THE INFLUENCE OF MINING EFFLUENTS.

<table>
<thead>
<tr>
<th></th>
<th>6 Hours</th>
<th>12 Hours</th>
<th>24 Hours</th>
<th>48 Hours</th>
<th>72 Hours</th>
<th>96 Hours</th>
<th>120 Hours</th>
<th>720 Hours</th>
<th>1080 Hours</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Control</strong></td>
<td>9.0</td>
<td>±0.37</td>
<td></td>
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<tr>
<td><strong>0.01% M.E.</strong></td>
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<tr>
<td></td>
<td>3.30</td>
<td>±0.24</td>
<td>5.10</td>
<td>±0.39</td>
<td>5.60</td>
<td>±0.43</td>
<td>3.70</td>
<td>±0.11</td>
<td>4.30</td>
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<tr>
<td><strong>0.1% M.E.</strong></td>
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<tr>
<td></td>
<td>4.80</td>
<td>±0.17</td>
<td>7.40</td>
<td>±0.43</td>
<td>5.50</td>
<td>±0.215</td>
<td>4.50</td>
<td>±0.34</td>
<td>2.90</td>
</tr>
<tr>
<td><strong>1% M.E.</strong></td>
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<tr>
<td></td>
<td>4.0</td>
<td>±0.13</td>
<td>2.80</td>
<td>±0.24</td>
<td>5.30</td>
<td>±0.13</td>
<td>4.30</td>
<td>±0.37</td>
<td>2.20</td>
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<td><strong>5% M.E.</strong></td>
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<tr>
<td></td>
<td>4.20</td>
<td>±0.3</td>
<td>4.80</td>
<td>±0.53</td>
<td>5.40</td>
<td>±0.303</td>
<td>4.50</td>
<td>±0.146</td>
<td>10.10</td>
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<tr>
<td><strong>10% M.E.</strong></td>
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<tr>
<td></td>
<td>5.90</td>
<td>±0.8</td>
<td>7.30</td>
<td>±0.426</td>
<td>8.20</td>
<td>±1.78</td>
<td>9.10</td>
<td>±1.85</td>
<td>10.172</td>
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<tr>
<td><strong>50% M.E.</strong></td>
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<tr>
<td></td>
<td>9.70</td>
<td>±0.271</td>
<td>8.40</td>
<td>±0.462</td>
<td>10.70</td>
<td>±1.56</td>
<td>13.20</td>
<td>±1.16</td>
<td>12.60</td>
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<td><strong>100% M.E.</strong></td>
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</tbody>
</table>

**Note:** Unit: Uric acid in mg/dL.

N.D.: Not Determined.

All alterations are statistically significant - P < 0.01.
GRAPH NO. 35: ALTERATIONS IN THE SERUM URIC ACID LEVEL.
Units: mg/dL.

<table>
<thead>
<tr>
<th>Hours</th>
<th>0.01% M.E.</th>
<th>0.1% M.E.</th>
<th>1% M.E.</th>
<th>5% M.E.</th>
<th>10% M.E.</th>
<th>50% M.E.</th>
<th>100% M.E.</th>
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</thead>
<tbody>
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</tr>
</tbody>
</table>
VII) ALTERATIONS IN THE SERUM CREATININE:

The alterations in the serum creatinine levels are tabulated in Table No.36 and are graphically expressed in Graph No. 36. The serum creatinine concentrations are expressed as milligrams per 100 ml (per decilitre). The control birds exhibited the serum creatinine concentration equivalent to 0.2 ± 0.02 mg per decilitre.

From the table and the graph it appears that under the influence of mining effluents (M.E.) the ducks showed M.E. concentration dependent increase in the serum creatinine level at the end of six hours. Similarly, the exposure of ducks to M.E for twelve hours promoted increase in the serum creatinine level as the M.E. concentrations increased except for 10% M.E.

When the ducks were treated with 0.01% M.E. there was no significant change in the serum creatinine levels up to the end of twenty nine days but at the end of thirty days the serum creatinine concentrations increased and continued to increase further up to the end of forty five days. Thus, at the end of thirty and forty five days the serum creatinine level was equivalent to 0.50 ± 0.098 and 0.90 ± 0.215 mg per decilitre respectively.

Under the influence of 0.1% M.E. the serum creatinine level fluctuated between 0.5 ± 0.141 and 1.375 ± 0.147 mg per decilitre. The maximum increase in the serum creatinine level was observed under the influence of 0.1% M.E. at the end of one hundred and twenty hours.

By the end of six, twelve, twenty four, forty eight, seventy two, ninety six and one hundred and twenty hours the serum creatinine concentration was equivalent to 0.50 ± 0.141, 0.68 ± 0.312, 0.48 ± 0.132, 0.34 ± 0.101, 1.06 ± 0.185, 1.24 ± 0.101 and 1.375 ± 0.147 mg per decilitre respectively.

On exposure to 1.0% M.E. the ducks exhibited fluctuations in the serum creatinine levels and the serum creatinine level ranged between 0.46 ± 0.135 and 1.84 ± 0.101 mg per decilitre. By the end of six, twelve, twenty four, forty eight, seventy two, ninety six and one twenty hours the serum creatinine
level was equivalent to $0.60 \pm 0.141$, $1.06 \pm 0.162$, $0.46 \pm 0.135$, $0.86 \pm 0.185$, $1.42 \pm 0.172$, $1.04 \pm 0.101$ and $1.84 \pm 0.101$ mg per decilitre respectively.

The ducks subjected to 5.0% M.E. showed increase in serum creatinine level from the end of six hours to the end of twentyfour hours and subsequently the serum creatinine level fluctuated. The maximum serum creatinine level for this M.E. concentration was observed at the end of seventytwo hours. By the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours the serum creatinine level was equivalent to $0.96 \pm 0.215$, $1.36 \pm 0.233$, $2.08 \pm 0.256$, $1.76 \pm 0.377$, $2.52 \pm 0.193$, $2.02 \pm 0.074$ and $1.48 \pm 0.231$ mg per decilitre respectively.

The 10.0% M.E. induced fluctuations in the serum creatinine level and the creatinine level ranged between $0.84 \pm 0.397$ and $2.42 \pm 0.172$ mg per decilitre. This dose of M.E. induced maximum increase in serum creatinine level at the end of ninetysix hours. By the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix, and one twenty hours the serum creatinine level was equivalent to $1.48 \pm 0.285$, $1.24 \pm 0.307$, $0.84 \pm 0.397$, $1.46 \pm 0.241$, $1.74 \pm 0.185$, $2.42 \pm 0.172$ and $1.45 \pm 0.165$ mg per decilitre respectively.

Under the influence of 50% M.E. the serum creatinine levels increased progressively from the end of six hours to the end of ninety six hours indicating exposure period dependency of creatinine elevation. By the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix, and one twenty hours the serum creatinine level was equivalent to $1.74 \pm 0.705$, $1.94 \pm 0.32$, $2.56 \pm 0.445$, $3.02 \pm 0.29$, $3.42 \pm 0.074$, $3.60 \pm 0.141$ and $3.02 \pm 0.172$ mg per decilitre respectively.

The ducks treated with 100% M.E. exhibited acute rise in the serum creatinine level and the creatinine level fluctuated between $2.23 \pm 0.617$ and $3.72 \pm 0.634$ mg per decilitre. The highest increase in the serum creatinine level was observed at the end of seventy two hours and this rise was over twelve fold. The serum creatinine level was equivalent to $2.86 \pm 0.24$, $3.46 \pm$
0.382, 2.60 ± 0.648, 2.23 ± 0.617, 3.72 ± 0.634, 3.06 ± 0.730 and 2.25 ± 0.456 mg per decilitre at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one hundred and twenty hours respectively.
**TABLE NO. 36: ALTERATIONS IN THE SERUM CREATININE UNDER THE INFLUENCE TO MINING EFFLUENT.**

<table>
<thead>
<tr>
<th></th>
<th>6 Hours</th>
<th>12 Hours</th>
<th>24 Hours</th>
<th>48 Hours</th>
<th>72 Hours</th>
<th>96 Hours</th>
<th>120 Hours</th>
<th>720 Hours</th>
<th>1080 Hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>0.20±0.02</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>0.01% M.E.</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>0.50±0.098</td>
<td>0.90±0.215</td>
<td></td>
</tr>
<tr>
<td>0.1% M.E.</td>
<td>0.50±0.141</td>
<td>0.68±0.312</td>
<td>0.48±0.132</td>
<td>0.34±0.101</td>
<td>1.06±0.185</td>
<td>1.24±0.101</td>
<td>1.375±0.147</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td>1% M.E.</td>
<td>0.60±0.141</td>
<td>1.06±0.162</td>
<td>0.46±0.135</td>
<td>0.86±0.185</td>
<td>1.40±0.172</td>
<td>1.04±0.101</td>
<td>1.84±0.101</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td>5% M.E.</td>
<td>0.96±0.215</td>
<td>1.36±0.233</td>
<td>2.08±0.256</td>
<td>1.76±0.377</td>
<td>2.52±0.193</td>
<td>2.02±0.074</td>
<td>1.48±0.231</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td>10% M.E.</td>
<td>1.48±0.285</td>
<td>1.24±0.307</td>
<td>0.84±0.397</td>
<td>1.46±0.241</td>
<td>1.74±0.185</td>
<td>2.42±0.172</td>
<td>1.45±0.165</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td>50% M.E.</td>
<td>1.74±0.705</td>
<td>1.94±0.32</td>
<td>2.56±0.445</td>
<td>3.02±0.29</td>
<td>3.42±0.074</td>
<td>3.60±0.141</td>
<td>3.02±0.172</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td>100% M.E.</td>
<td>2.86±0.24</td>
<td>3.46±0.382</td>
<td>2.60±0.648</td>
<td>2.23±0.617</td>
<td>3.72±0.634</td>
<td>3.06±0.730</td>
<td>2.25±0.456</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
</tbody>
</table>

**Note:** Unit: Creatinine in mg/dL.  
N.D.: Not Determined.  
All alterations are statistically significant - P < 0.01.
GRAPH NO. 36: ALTERATIONS IN THE SERUM CREATININE LEVEL.
Units: mg/dL.
ALTERATIONS IN SERUM CALCIUM LEVEL:

The changes in the serum calcium level under the influence of mining effluents (M.E.) are given in Table No. 37 and are graphically presented in Graph No. 37. The serum calcium level is expressed as milligrams (mg) per decilitre (100 ml). The control animals had serum calcium level equivalent to $4.30 \pm 0.10$ mg per decilitre. From the table and graph it appears that all the concentrations of M.E. except 10.0% promoted concentration dependent elevations in the serum calcium level at the end of six and twelve hours. The 10.0% M.E. induced highest increase in the serum calcium level at the end of one hundred and twenty hours while 0.1% M.E. induced reduction in the serum calcium level at the end of six and twelve hours.

The ducks treated with 0.01% M.E. did not exhibit any significant change in the serum calcium level up to the end of twenty nine days but at the end of thirty days the serum calcium level elevated and continued to elevate up to the end of forty five days. Thus, at the end of thirty and fortyfive days the serum calcium level was equivalent to $8.37 \pm 0.172$, $13.72 \pm 0.312$ mg per decilitre respectively.

When the ducks were subjected to 0.1% M.E., the serum calcium concentrations dropped marginally at the end of six and twelve hours and then elevated gradually from the end of twentyfour hours to the end of one twenty hours, except for seventy two hours. By the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one hundred and twenty hours the calcium concentrations were equivalent to $4.02 \pm 0.116$, $4.02 \pm 0.116$, $5.94 \pm 0.205$, $7.04 \pm 0.185$, $6.64 \pm 0.249$, $9.70 \pm 0.167$ and $13.64 \pm 0.257$ mg per decilitre respectively.

The exposure of ducks to 1.0% M.E. promoted fluctuations in the serum calcium levels and the calcium level fluctuated between $6.06 \pm 0.386$ and $12.26 \pm 0.185$ mg per decilitre. Thus, this dose of M.E. in general caused elevation in the serum calcium concentration. By the end of six, twelve, twentyfour, forty
eight, seventytwo, ninetysix and one twenty hours, the serum calcium level was equivalent to 6.80 ± 0.404, 6.06 ± 0.386, 7.50 ± 0.228, 8.06 ± 0.344, 6.66 ± 0.185, 12.26 ± 0.185 and 11.56 ± 0.508 mg per decilitre respectively.

The 5.0% M.E. induced elevation in the serum calcium level and the calcium concentrations varied between 6.84 ± 0.30 and 11.0 ± 0.228 mg per decilitre. The serum calcium level was equivalent to 9.0 ± 0.363, 9.88 ± 0.172, 10.28 ± 0.466, 8.14 ± 0.313, 11.0 ± 0.228, 8.98 ± 0.172 and 6.84 ± 0.30 mg per decilitre at the end of six, twelve, twenty four, fortyeight, seventytwo, ninetysix and one twenty hours respectively.

Under the influence of 10.0% M.E. the ducks showed exposure period dependent rise in the serum calcium level. Thus, longer the exposures of ducks to 10% M.E. the higher the concentrations of calcium were observed. This dose induced the highest elevation in serum calcium level at the end of one twenty hours and the serum calcium level was equivalent to 5.50 ± 0.20, 6.62 ± 0.172, 7.68 ± 0.116, 13.30 ± 0.260, 17.60 ± 0.244, 22.325 ± 0.402 and 26.20 ± 0.316 mg per decilitre at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one hundred and twenty hours respectively.

The 50.0% M.E. caused sharp rise in the serum calcium level and the serum calcium level fluctuated between 10.06 ± 0.257 and 18.56 ± 0.195 mg per decilitre. The serum calcium concentration was equivalent to 15.42 ± 0.365, 10.06 ± 0.257, 10.12 ± 0.376, 11.34 ± 0.392, 13.34 ± 0.257, 18.56 ± 0.195 and 14.72 ± 0.132 mg per decilitre at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one hundred and twenty hours respectively.

The exposure of ducks to 100% M.E. promoted, in general, elevation of serum calcium concentration, especially at the end of six and twentyfour hours the calcium level increased acutely. The serum calcium level varied between 8.68 ± 0.27 and 23.18 ± 1.783 mg per decilitre. The serum calcium level was equivalent to 21.94 ± 0.349, 18.50 ± 0.303, 23.18 ± 1.783, 10.26 ± 0.50, 9.48 ±
0.278, 8.68 ± 0.27 and 15.84 ± 0.135 mg per decilitre at the end of six, twelve, twenty four, fortyeight, seventy two, ninetysix and one hundred and twenty hours respectively.
### TABLE NO. 37: ALTERATIONS IN THE SERUM CALCIUM LEVEL UNDER THE INFLUENCE OF MINING EFFLUENTS.

<table>
<thead>
<tr>
<th></th>
<th>6 Hours</th>
<th>12 Hours</th>
<th>24 Hours</th>
<th>48 Hours</th>
<th>72 Hours</th>
<th>96 Hours</th>
<th>120 Hours</th>
<th>720 Hours</th>
<th>1080 Hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>4.30±0.1</td>
<td></td>
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<tr>
<td>0.01% M.E.</td>
<td></td>
<td>4.02±0.116</td>
<td>4.02±0.116</td>
<td>5.94±0.205</td>
<td>7.04±0.185</td>
<td>6.64±0.249</td>
<td>9.70±0.167</td>
<td>13.64±0.257</td>
<td>8.37±0.172</td>
</tr>
<tr>
<td>0.1% M.E.</td>
<td></td>
<td>6.80±0.404</td>
<td>6.06±0.386</td>
<td>7.50±0.228</td>
<td>8.06±0.344</td>
<td>6.66±0.185</td>
<td>12.26±0.185</td>
<td>11.56±0.508</td>
<td>N.D.</td>
</tr>
<tr>
<td>1% M.E.</td>
<td></td>
<td>9.0±0.363</td>
<td>9.88±0.172</td>
<td>10.28±0.466</td>
<td>8.14±0.313</td>
<td>11.0±0.228</td>
<td>8.98±0.172</td>
<td>6.84±0.30</td>
<td>N.D.</td>
</tr>
<tr>
<td>5% M.E.</td>
<td></td>
<td>5.50±0.20</td>
<td>6.62±0.172</td>
<td>7.68±0.116</td>
<td>13.30±0.260</td>
<td>17.60±0.244</td>
<td>22.325±0.402</td>
<td>26.20±0.316</td>
<td>N.D.</td>
</tr>
<tr>
<td>10% M.E.</td>
<td></td>
<td>15.42±0.365</td>
<td>10.06±0.257</td>
<td>10.12±0.376</td>
<td>11.34±0.392</td>
<td>13.34±0.257</td>
<td>18.56±0.195</td>
<td>14.72±0.132</td>
<td>N.D.</td>
</tr>
<tr>
<td>50% M.E.</td>
<td></td>
<td>21.94±0.349</td>
<td>18.50±0.303</td>
<td>23.18±1.783</td>
<td>10.26±0.50</td>
<td>9.48±0.278</td>
<td>8.68±0.27</td>
<td>15.84±0.135</td>
<td>N.D.</td>
</tr>
<tr>
<td>100% M.E.</td>
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</tbody>
</table>

**Note:** Unit: Calcium in mg/dL.  
N.D.: Not Determined.  
All alterations are statistically significant - P < 0.01.
GRAPH NO. 37: ALTERATIONS IN THE SERUM CALCIUM LEVEL.
Units: mg/dL.
IX) ALTERATIONS IN THE SERUM CHLORIDE CONCENTRATIONS:

The alterations in the serum chloride concentrations under the influence of mining effluents (M.E.) are compiled in Table No. 38 and are graphically expressed in Graph No. 38. The chloride concentrations of the serum are expressed as milliequivalance (mEq) per litre. The control animals showed the serum chloride concentrations equivalent to $89.0 \pm 2.70$ mEq/litre.

From the table and the graph it appears that mining effluents profoundly influenced the serum chloride concentrations, especially the higher concentrations of M.E induced acute increase in the serum chloride levels. The serum chloride levels did not alter in general in a dose (M.E. concentrations) and exposure time dependent manner. The $50\%$ M.E. induced highest rise in the serum chloride level at the end of six hours while the acute reduction in the chloride concentrations was promoted by $0.1\%$ M.E. at the end of forty eight hours.

The ducks exposed to $0.01\%$ M.E. did not show any significant change in the serum chloride level up to the end of twenty nine days but at the end of thirty days the chloride concentrations increased significantly and continued to increase further up to the end of forty five days. By the end of thirty and forty five days, the serum chloride concentrations were equivalent to $110.80 \pm 5.10$ and $184.60 \pm 1.624$ mEq per litre respectively.

Under the influence of $0.1\%$ M.E. the serum chloride level dropped acutely at the end of forty eight hours and increased significantly to a higher level at the end of ninetysix hours. The serum chloride level was equivalent to $126.92 \pm 2.10$, $121.0 \pm 1.41$, $118.0 \pm 1.632$, $52.0 \pm 1.41$, $77.0 \pm 1.341$, $127.68 \pm 0.80$ and $120.58 \pm 1.36$ mEq per litre at the end of six, twelve, twentyfour, fortyeight, seventy two, ninetysix and one hundred and twenty hours respectively.

On exposure to $1.0\%$ M.E. the ducks exhibited fluctuations in the serum chloride concentrations and the serum chloride concentrations varied between
105.0 ± 0.77 and 176.40 ± 4.31 mEq per litre. By the end of one hundred and twenty hours exposure the serum chloride levels elevated acutely. The serum chloride level was equivalent to 115.40 ± 2.059, 105.0 ± 0.77, 165.0 ± 1.6, 108.0 ± 3.521, 137.0 ± 4.4, 128.60 ± 1.496 and 176.40 ± 4.31 mEq per litre at the end of six, twelve, twenty four, fortyeight, seventytwo, ninetysix and one hundred and twenty hours respectively.

The ducks subjected to 5.0% M.E. exhibited elevation of serum chloride contents but the serum chloride concentrations varied between 120.0 ± 4.8 and 190.0 ± 3.12 mEq/litre. By the end of six, twelve, twentyfour, fortyeight, seventy two, ninetysix and one twenty hours the serum chloride concentrations were equivalent to 125.0 ± 1.85, 120.0 ± 4.8, 189.0 ± 1.01, 185.0 ± 4.40, 190.0 ± 3.12, 153.0 ± 1.78 and 131.0 ± 1.41 mEq/litre respectively.

When the ducks were introduced to 10.0% M.E. the serum chloride levels elevated sharply and the maximum chloride contents were seen at the end of fortyeight hours while at the end of one hundred and twenty hours the chloride concentrations lowered a little and remained above that observed in the control. The serum chloride concentration was equivalent to 176.0 ± 2.20, 178.0 ± 1.16, 170.0 ± 3.92, 193.0 ± 2.90, 180.0 ± 1.72, 110.0 ± 1.23 and 100.0 ± 1.30 mEq/litre at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one hundred and twenty hours respectively.

The 50% M.E. promoted the highest increase in the serum chloride level at the end of six hours and for almost all time intervals except fortyeight hours, the serum chloride levels were four to five fold higher than that observed in the controls. By the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours, the serum chloride concentration was equivalent to 485.0 ± 2.607, 452.0 ± 3.95, 443.20, ± 2.39, 137.20 ± 2.40, 391.0 ± 7.50, 443.40 ± 2.87 and 444.60 ± 2.57 mEq/litre respectively.

The exposure of ducks to 100% M.E. caused over four fold increase in the serum chloride concentration up to the end of fortyeight hours but at the
end of seventy two hours, the serum chloride level dropped below that found in the control animals. Then, once again the chloride concentrations increased progressively for the subsequent exposure periods. By the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one hundred and twenty hours, the serum chloride concentrations were equivalent to $443.40 \pm 3.10$, $439.40 \pm 4.49$, $445.0 \pm 1.09$, $443.80 \pm 3.18$, $78.80 \pm 1.72$, $110.0 \pm 3.10$ and $127.20 \pm 2.10$ mEq/litre respectively.
<table>
<thead>
<tr>
<th></th>
<th>6 Hours</th>
<th>12 Hours</th>
<th>24 Hours</th>
<th>48 Hours</th>
<th>72 Hours</th>
<th>96 Hours</th>
<th>120 Hours</th>
<th>720 Hours</th>
<th>1080 Hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>89.0</td>
<td>±2.7</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>0.01% M.E.</td>
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<tr>
<td></td>
<td>No significant change</td>
<td></td>
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<tr>
<td>0.1% M.E.</td>
<td>126.92</td>
<td>±2.10</td>
<td>121.0</td>
<td>±1.41</td>
<td>118.0</td>
<td>±1.632</td>
<td>52.0</td>
<td>±1.41</td>
<td>77.0</td>
</tr>
<tr>
<td>1% M.E.</td>
<td>115.4</td>
<td>±2.059</td>
<td>105.0</td>
<td>±0.77</td>
<td>165.0</td>
<td>±1.6</td>
<td>108.0</td>
<td>±3.521</td>
<td>137.0</td>
</tr>
<tr>
<td>5% M.E.</td>
<td>125.0</td>
<td>±1.85</td>
<td>120.0</td>
<td>±4.80</td>
<td>189.0</td>
<td>±1.01</td>
<td>185.0</td>
<td>±4.40</td>
<td>190.0</td>
</tr>
<tr>
<td>10% M.E.</td>
<td>176.0</td>
<td>±2.20</td>
<td>178.0</td>
<td>±1.16</td>
<td>170.0</td>
<td>±3.92</td>
<td>193.0</td>
<td>±2.90</td>
<td>180.0</td>
</tr>
<tr>
<td>50% M.E.</td>
<td>485.0</td>
<td>±2.607</td>
<td>452.0</td>
<td>±3.95</td>
<td>443.20</td>
<td>±2.39</td>
<td>137.20</td>
<td>±2.40</td>
<td>391.0</td>
</tr>
<tr>
<td>100% M.E.</td>
<td>443.40</td>
<td>±3.10</td>
<td>439.40</td>
<td>±4.49</td>
<td>445.0</td>
<td>±1.09</td>
<td>443.80</td>
<td>±3.18</td>
<td>78.80</td>
</tr>
</tbody>
</table>

Note: Unit: Chloride in mEq/Litre.
N.D.: Not Determined.
All alterations are statistically significant - P < 0.01.
GRAPH NO. 38: ALTERATIONS IN SERUM CHLORIDE LEVEL.
Units: mEq/L.
X) ALTERATIONS IN THE SERUM ELECTROLYTES:

a) Na⁺:

The alterations in the serum sodium concentrations under the influence of mining effluents (M.E.) are given in Table No. 39 and are graphically presented in Graph No. 39. The Na⁺ concentrations of the serum are expressed as milliequivalence per litre. The control animals showed serum Na⁺ concentration equal to 120.0 ± 3.0 mEq/litre.

From the graph and the table it appears that M.E. induced elevation in the serum Na⁺ concentrations at all the time intervals under study and under all the M.E. concentrations. The elevations in the serum Na⁺ contents were neither M.E. concentration dependent nor exposure period dependent.

When the ducks were exposed to 0.01% M.E. the serum Na⁺ concentrations did not vary significantly up to the end of twenty nine days but at the end of thirty days the Na⁺ concentration elevated and continued to increase further up to the end of forty five days. Then, the serum Na⁺ concentration was equivalent to 160.0 ± 2.10 and 172.80 ± 1.62 mEq/litre at the end of thirty and forty five days respectively.

The exposure of ducks to 0.1% M.E. promoted increase in the serum Na⁺ concentration at the end of six hours and the Na⁺ level continued to increase progressively up to the end of twenty four hour. Subsequently, the serum Na⁺ concentrations fluctuated. By the end of six, twelve, twenty four, forty eight seventy two, ninetysix and one hundred and twenty hours, the serum Na⁺ concentration was equivalent to 141.0 ± 1.78, 150.80 ± 1.16, 160.60 ± 2.10, 142.20 ± 2.78, 160.60 ± 1.624, 172.0 ± 2.80 and 146.40 ± 2.65 mEq/litre respectively.

Under the influence of 1.0% M.E. the serum Na⁺ concentrations elevated and fluctuated between 144.20 ± 1.72 and 161.0 ± 1.854 mEq/litre. By the end of ninety six hours the serum Na⁺ concentration increased by about
thirty three percent. The serum Na\(^+\) concentration was equivalent to 144.20 ± 1.72, 156.40 ± 1.35, 147.0 ± 2.20, 158.60 ± 1.01, 148.80 ± 1.72, 161.60 ± 1.85 and 148.60 ± 1.01 mEq/litre at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one hundred and twenty hours respectively.

When the ducks were introduced to 5.0% M.E. the serum Na\(^+\) concentration varied in the range of 148.40 ± 1.20 to 179.0 ± 3.40 mEq/litre. By the end of one hundred and twenty hours, the serum Na\(^+\) level stepped up by fortynine percent. By the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one hundred and twenty hours the serum Na\(^+\) concentration was equivalent to 158.60 ± 1.01, 155.40 ± 3.0, 164.20 ± 3.1, 174.20 ± 3.4, 164.40 ± 3.8, 148.40 ± 1.20 and 179.0 ± 3.40 mEq/litre respectively.

The exposure of ducks to 10.0% M.E. induced rise in serum Na\(^+\) level at the end of six hours and it continued to increase gradually up to the end of twenty four hours but subsequently the Na\(^+\) concentrations fluctuated considerably. The Na\(^+\) concentration increased maximally by 42.30 percent at the end of ninetysix hours. At the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix, and one twenty hours, the serum Na\(^+\) concentration was equivalent to 148.0 ± 1.4, 151.40 ± 1.74, 158.60 ± 2.41, 149.0 ± 1.4, 162.40 ± 3.44, 170.80 ± 2.56 and 162.40 ± 3.26 mEq/litre respectively.

The ducks subjected to 50.0% M.E. exhibited significant variations in the serum Na\(^+\) concentrations but at all the time intervals the Na\(^+\) level remained elevated. The Na\(^+\) concentration elevated to maximally 65.66 percent at the end of ninetysix hours. The serum Na\(^+\) concentration was equivalent to 159.80 ± 1.40, 170.40 ± 2.0, 161.0 ± 1.85, 186.60 ± 4.80, 194.0 ± 3.0, 198.80 ± 2.31 and 181.20 ± 4.40 mEq/litre at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one hundred and twenty hours respectively.
<table>
<thead>
<tr>
<th></th>
<th>6 Hours</th>
<th>12 Hours</th>
<th>24 Hours</th>
<th>48 Hours</th>
<th>72 Hours</th>
<th>96 Hours</th>
<th>120 Hours</th>
<th>720 Hours</th>
<th>1080 Hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>120.0 ±3.0</td>
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<tr>
<td>0.01% M.E.</td>
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<tr>
<td>0.1% M.E.</td>
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<tr>
<td>1% M.E.</td>
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<tr>
<td>5% M.E.</td>
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<tr>
<td>10% M.E.</td>
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<td>50% M.E.</td>
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<tr>
<td>100% M.E.</td>
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</tbody>
</table>

Note: Unit: Sodium in mEq/L.  
N.D.: Not Determined.  
All alterations are statistically significant - P < 0.01.
GRAPH NO. 39: ALTERATIONS IN SERUM SODIUM LEVEL.

Unit: mEq/L.
b) $K^+$:

The changes in the serum $K^+$ concentrations under the influence of mining effluents (M.E.) are compiled in Table No. 40 and are graphically given in Graph No. 40. The serum $K^+$ concentration is expressed as milliequivalence (mEq) per litre. The controls exhibited $K^+$ concentration equivalent to $4.0 \pm 0.17$ mEq/litre.

From the table and the graph it appears that the various M.E. concentrations induced elevation in the serum $K^+$ level. The serum $K^+$ concentrations elevated in a M.E. concentration dependent manner at the end of six, twentyfour and seventytwo hours. The highest rise in $K^+$ concentration was promoted by 100% M.E. at the end of seventytwo hours and this increase was over six fold of the control value.

When the ducks were exposed to 0.01% M.E. the serum $K^+$ concentrations did not show any significant change up to the end of twentynine days but at the end of thirty days the serum $K^+$ concentration increased and continued to increase up to the end of forty five days. Thus, at the end of thirty and forty five days the serum $K^+$ concentration was equivalent to $7.70 \pm 0.14$ and $10.82 \pm 0.11$ mEq/litre respectively.

The ducks introduced to 0.1% M.E. showed fluctuations in the serum $K^+$ levels. The $K^+$ concentrations fluctuated between $4.84 \pm 0.101$ and $8.52 \pm 0.132$ mEq/litre. The $K^+$ concentration increased over two fold at the end of six and fortyeight hours. The serum $K^+$ concentration was equivalent to $8.52 \pm 0.132$, $7.70 \pm 0.14$, $6.62 \pm 0.16$, $8.02 \pm 0.116$, $7.16 \pm 0.24$, $5.96 \pm 0.13$ and $4.84 \pm 0.101$ mEq/litre at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours respectively.

Under the influence of 1.0% M.E. the serum $K^+$ concentration increased over two fold at the end of six hours and continued to decrease gradually up to the end of twentyfour hours. Then, once again, the serum $K^+$ concentration increased significantly at the end of fortyeight hours and subsequently went on
decreasing up to the end of one hundred and twenty hours. By the end of six, twelve, twentyfour, fouryteight, seventytwo, ninetysix and one hundred and twenty hours, the K+ concentration was equivalent to 10.58 ± 0.172, 9.04 ± 0.135, 8.64 ± 0.149, 9.64 ± 0.249, 8.0 ± 0.17, 5.62 ± 0.10 and 4.86 ± 0.135 mEq/litre respectively.

The exposure of ducks to 5.0% M.E. promoted over three fold increase in serum K+ concentration at the end of six hours and subsequently the K+ concentrations went on declining up to the end of fortyeight hours. Then, once again the K+ concentration elevated over two folds of the control value at the end of seventy two hours and decreased gradually at the end of ninetysix and one hundred and twenty hours. The serum K+ concentration was equivalent to 12.16 ± 0.12, 11.42 ± 0.172, 10.46 ± 0.162, 9.24 ± 0.135, 10.78 ± 0.116, 8.68 ± 0.25 and 6.62 ± 0.23 mEq/litre at the end of six, twelve, twentyfour, fouryteight, seventytwo, ninetysix and one hundred and twenty hours respectively.

On exposing the ducks to 10% M.E. the serum K+ level elevated 3.83 folds of the control value at the end of six hours and subsequently decreased gradually up to the end of twenty four hours. Then, again, the K+ concentration increased a little at the end of fortyeight hours but went on declining steadily up to the end of one hundred and twenty hours. By the end of six, twelve, twenty four, fouryteight, seventytwo, ninetysix and one twenty hours the serum K+ concentration was equivalent to 15.32 ± 0.271, 13.22 ± 0.256, 12.04 ± 0.18, 12.82 ± 0.24, 10.80 ± 0.282, 8.60 ± 0.20 , and 5.64 ± 0.215 mEq/litre respectively.

The 50.0% M.E. induced significant variations in the serum K+ levels of the ducks. By the end of twelve hours, the serum K+ concentration increased over five folds of the control value but the minimum of two folds and little more, increase in K+ concentration was maintained at the end of one hundred and twenty hours. The serum K+ concentration was equivalent to 19.16 ± 0.377, 20.72 ± 0.256, 17.10 ± 0.374, 14.56 ± 0.272, 17.10 ± 0.228, 11.04 ± 0.372 and
8.84 ± 0.12 mEq/litre at the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one twenty hours respectively.

The exposure of ducks to 100.0% M.E. caused very sharp rise in the serum K⁺ levels. By the end of seventy two hours the ducks exhibited highest rise in the serum K⁺ concentration. The serum K⁺ concentrations ranged between 16.72 ± 0.426 and 27.78 ± 0.16 mEq/litre. At the end of six, twelve, twentyfour, fortyeight, seventytwo, ninetysix and one hundred and twenty hours, the serum K⁺ concentration was equivalent to 22.78 ± 0.435, 19.80 ± 0.268, 22.22 ± 1.18, 24.32 ± 0.39, 27.78 ± 0.16, 21.52 ± 0.342 and 16.72 ± 0.426 mEq/litre respectively.
### TABLE NO. 40: ALTERATIONS IN THE SERUM POTASSIUM CONCENTRATION IN THE DUCKS UNDER THE INFLUENCE OF MINING EFFLUENTS.

<table>
<thead>
<tr>
<th></th>
<th>6 Hours</th>
<th>12 Hours</th>
<th>24 Hours</th>
<th>48 Hours</th>
<th>72 Hours</th>
<th>96 Hours</th>
<th>120 Hours</th>
<th>720 Hours</th>
<th>1080 Hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>4.0</td>
<td>± 0.17</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>0.01% M.E.</td>
<td></td>
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</tr>
<tr>
<td>0.1% M.E.</td>
<td>8.52 ±0.132</td>
<td>7.70 ±0.14</td>
<td>6.62 ±0.16</td>
<td>8.02 ±0.116</td>
<td>7.16 ±0.24</td>
<td>5.96 ±0.13</td>
<td>4.84 ±0.101</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td>1% M.E.</td>
<td>10.58 ±0.172</td>
<td>9.04 ±0.135</td>
<td>8.64 ±0.149</td>
<td>9.64 ±0.249</td>
<td>8.02 ±0.17</td>
<td>5.62 ±0.10</td>
<td>4.86 ±0.135</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td>5% M.E.</td>
<td>12.16 ±0.12</td>
<td>11.42 ±0.172</td>
<td>10.46 ±0.162</td>
<td>9.24 ±0.135</td>
<td>10.78 ±0.116</td>
<td>8.68 ±0.25</td>
<td>6.62 ±0.23</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td>10% M.E.</td>
<td>15.32 ±0.271</td>
<td>13.22 ±0.18</td>
<td>12.04 ±0.18</td>
<td>12.82 ±0.24</td>
<td>10.80 ±0.282</td>
<td>8.60 ±0.20</td>
<td>5.64 ±0.215</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td>50% M.E.</td>
<td>19.16 ±0.377</td>
<td>20.72 ±0.256</td>
<td>17.10 ±0.374</td>
<td>14.56 ±0.272</td>
<td>17.10 ±0.228</td>
<td>11.04 ±0.372</td>
<td>8.84 ±0.12</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
<tr>
<td>100% M.E.</td>
<td>22.78 ±0.435</td>
<td>19.80 ±0.268</td>
<td>22.22 ±1.18</td>
<td>24.32 ±0.39</td>
<td>27.78 ±0.342</td>
<td>21.52 ±0.342</td>
<td>16.72 ±0.426</td>
<td>N.D.</td>
<td>N.D.</td>
</tr>
</tbody>
</table>

**Note:** Unit: Serum potassium in mEq / Litre.

**N.D.:** Not Determined.

All alterations are statistically significant - *P < 0.01.*
GRAPH NO. 40: ALTERATIONS IN SERUM POTASSIUM LEVEL.
Units: mEq/L.