Physiological changes during electrical asphyxiation

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Levy, L. S. (1971). Brit. J. industr. Med., 28, 164-171. Physiological changes during electrical asphyxiation. In this study of death caused by electric shock the rabbit has been used as an experimental animal to elucidate some of the physiological changes that occur in the blood gas, acid-base, and cardiorespiratory systems during long, non-fibrillating electric shocks. The results obtained were compared with known and confirmed observations for obstructive asphyxiation produced by tracheal occlusion. The main finding was that in electrical asphyxiation the lack of respiratory movements during the shock coupled with the rapid development of a very severe metabolic acidosis caused very early cardiac standstill. This indicates the urgent need for the instigation of resuscitative procedures after such accidents.

Death due to electric shock may occur by one of three primary mechanisms - ventricular fibrillation, persistent respiratory arrest or asphyxiation. Ventricular fibrillation is believed to be the most commonly occurring mechanism (Bruttau, 1918; Ajello, 1929; Legge et al., 1922; Helpern and Strassmann, 1941) and has been particularly well documented and studied. It has been shown to be caused by currents of a particular magnitude and duration passing across the chest (Ferris, King, Spence, and Williams, 1936; Kouwenhoven et al., 1959; Kiselev, 1968).

Persistent respiratory arrest caused by interference with the respiratory centre is the mechanism on which is based the concept that artificial respiration should be applied to all victims of electric shock. However, as only a small percentage of electrical fatalities have suffered a pathway that includes the medulla, this mechanism may account for only a few of the total number of deaths (Lee, 1965).

The third mechanism is asphyxiation, caused by the current flowing across the chest and holding the muscles of respiration in tetanic contraction for the duration of its passage. The commonest pathway for this type of accident is either from arm to arm or arm to leg or legs. In either case, the shock needs to be above a certain threshold so that the victim is unable to release himself from the circuit; this is related to the 'let-go threshold' as defined by Dalziel, Ogden, and Abbott (1943). In the rabbit, an AC current in excess of 50 mA is required to cause tetanic contraction of the respiratory muscles for the duration of the shock (Angelis, Lee, and Zoledziowski, 1966) and in the dog, a current of about 50 mA will also produce this same effect (Greenberg, 1940). In man, accident cases in which electrical asphyxiation has occurred without the onset of ventricular fibrillation are well documented (Lee, 1961). This would suggest that similar events occur in man at currents below the ventricular fibrillation threshold, believed to be about 80 mA (Dalziel and Lee, 1968).

Method
The rabbit was used in this study as it reverts spon-
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The experimental work was performed in two groups. The first group was used for electrical asphyxiation, and the second with obstructive asphyxiation produced by tracheal occlusion. The latter group was used as a control because many physiological data are available in the literature (Herber, 1948; Grodins, Lein, and Adler, 1946) and also because the mechanism of death is essentially similar.

Both groups of animals were treated basically in the same way. A general anaesthetic was used throughout (urethane, 2 g/kg bodyweight in a 0·5 g/ml aqueous solution). In both groups of animals, a tracheotomy was performed and an endotracheal tube was tied into place. A respiratory transducer attached to the endotracheal tube allowed respiration to be monitored (Lee, Zoledziowski, and Temiyachol, 1967). A carotid artery catheter was used to obtain blood samples and to monitor blood pressure (Statham pressure transducer, P23 series). Right ventricular pressures were measured (Statham pressure transducer, P23 series) through a catheter introduced via the right jugular vein. The heart was also monitored with an electrocardiograph (Honeywell ECG amplifier, type 2581) except during shock periods. All the above variables were continuously monitored on two twin-beam oscilloscopes (Tequipment, type D43R) and permanently recorded on a multi-channel ultraviolet recorder (Honeywell, type 2500).

In the first group, electrical asphyxiation was produced by means of a fore-limb to fore-limb continuous 50 Hz shock at about 60 mA. The experimental set-up for an electrical asphyxiation is shown in Figure 1. In the second group of animals, obstructive asphyxiation was produced by clamping the endotracheal tube. Arterial blood samples were taken in both groups just before asphyxiation and at half-minute intervals from the onset of asphyxiation. These samples were analysed for oxygen tension (Radiometer type E.5044), carbon dioxide tension (Radiometer type E.530), pH (Radiometer E.5030), and lactic acid content (enzymic lactate U-V method; TC-D Art. No. 15972, published and assembled by C. F. Boehringer und Soehne). The base excess was calculated from a nomogram (Siggaard-Andersen, 1963) using observed pH and carbon dioxide tension values.

The experiment was terminated in each group when cardiac arrest occurred and/or any beats, if present, were non-expulsive.

Results

The results for the two groups of animals are shown in Figs. 2 to 4 and Tables 1 to 5. Table 1 and Fig. 2 show that the short and slight rise in blood pressure during the two series of experiments was similar, but that in electrical asphyxiation the subsequent fall occurred more quickly. The mean time taken to reach zero was 6·6 minutes in obstructive asphyxiation compared with 4·14 minutes in electrical asphyxiation.

Table 2 shows that the rate of fall of arterial oxygen tension is similar in the two groups of animals. When allowance is made for the difference in pH (see Table 4), which affects the oxygen saturation at a given oxygen tension (Kelman and Nunn, 1966), the rate at which the arterial blood became desaturated was slightly greater in the electrical asphyxiation group. Table 3 shows that there was also very little difference in the rate of carbon dioxide retention between the two groups.

The acid-base studies show that in electrical asphyxiation the deleterious effects were more rapid. The pH values for the two groups are shown in Table 4. Whereas the mean pH fell from 7·4 to 7·1 in 5 minutes during obstructive asphyxiation, the fall was from 7·4 to 6·85 in 4 minutes during electrical asphyxiation. Consequently the base excess figures, which are derived from the pH and CO₂ tension findings and are shown in Fig. 3, show a marked difference between the two groups. During obstructive asphyxiation there is a gradual fall in base excess which does not start until about the third minute, whereas during electrical asphyxiation the fall starts immediately and is much more rapid. The lactic acid estimations also showed a marked difference between the electrical and obstructive groups with the concentration of acid rising much faster during electric shock (Table 5 and Fig. 4). At 3·2 minutes there had been a fourfold increase in mean lactic acid content in the electrical group and less than a twofold increase in the obstructive group.

Mechanical respiratory activity ceased after a mean interval of 3·22 minutes from the onset of...
tracheal occlusion for the series of animals subjected to obstructive asphyxiation. The type of activity seemed constant, with increasingly deeper, though infrequent inspiratory gasps. During all the electrical asphyxiations from the beginning of the shock until the time the current was switched off, the chest muscles were held in complete tetanic contraction and no respiratory movements occurred. From the blood pressure and right ventricular recordings there was no evidence that the heart was directly affected by the electric shock.

In an earlier pilot study, it had been shown that in electrical asphyxiation the rabbit is unable to resume spontaneous respiration more than 2 or 3 minutes after the beginning of a shock, whereas, in obstructive asphyxiation, an animal was capable of making respiratory attempts up to about 4 minutes after the beginning of tracheal occlusion. This indicates that in electrical asphyxiation there might be less time in which to initiate successful resuscitative procedures. This is further supported by the times at which the circulation collapsed in the two groups of animals.

### Discussion

Obstructive asphyxiation has been studied in the dog by several workers (Herber, 1948; Grodins et al., 1946) and has been shown to follow a fairly constant pattern. This consists initially of a respiratory acidosis, associated with a decreasing pH and an increasing base excess. This effect, however, is superseded by an uncompensated metabolic acidosis characterized by a decreasing pH and a decreasing base excess. The primary cause of the metabolic acidosis was shown to be due to the accumulation

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**TABLE 1**

**BLOOD PRESSURE FOR OBSTRUCTIVE AND ELECTRICAL ASPHYXIATION (mmHg)**

<table>
<thead>
<tr>
<th>Minutes</th>
<th>Obstructive asphyxiation</th>
<th>Electrical asphyxiation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean S D</td>
<td>Range Max. Min. S D</td>
</tr>
<tr>
<td>0</td>
<td>102 89</td>
<td>130 96 60 50</td>
</tr>
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<td>170 120 80 55</td>
</tr>
<tr>
<td>11/2</td>
<td>138 100</td>
<td>170 120 70 50</td>
</tr>
<tr>
<td>2</td>
<td>139 93</td>
<td>200 120 55 45</td>
</tr>
<tr>
<td>21/2</td>
<td>126 86</td>
<td>200 120 60 40</td>
</tr>
<tr>
<td>3</td>
<td>114 77</td>
<td>180 120 50 37</td>
</tr>
<tr>
<td>31/2</td>
<td>101 70</td>
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<td>82 60</td>
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<td>120 85 8 5</td>
</tr>
<tr>
<td>51/2</td>
<td>31 21</td>
<td>95 70 0 0</td>
</tr>
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<td>6</td>
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</tr>
<tr>
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<td>34 22</td>
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<td>14 5 0 0</td>
</tr>
<tr>
<td>9</td>
<td>0 0</td>
<td>0 0 0 0</td>
</tr>
</tbody>
</table>

\( S = \text{Systolic pressure} \quad D = \text{diastolic pressure} \)

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**FIG. 2.** Systolic and diastolic blood pressure. ○—○ obstructive asphyxiation; ●—● electrical asphyxiation.
Physiological changes during electrical asphyxiation

TABLE 2
OXYGEN TENSION FOR OBSTRUCTIVE AND ELECTRICAL ASPHYXIATION (mmHg)

<table>
<thead>
<tr>
<th>Minutes</th>
<th>Obstructive asphyxiation</th>
<th>Electrical asphyxiation</th>
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</thead>
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<tr>
<td></td>
<td>Range</td>
<td>No. of readings</td>
</tr>
<tr>
<td></td>
<td>Max.</td>
<td>Min.</td>
</tr>
<tr>
<td>0</td>
<td>74</td>
<td>104</td>
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<tr>
<td>1⁄2</td>
<td>38</td>
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<td>2</td>
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<td>28</td>
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<td>18</td>
</tr>
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<td>4</td>
<td>11</td>
<td>16</td>
</tr>
<tr>
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<td>12</td>
</tr>
<tr>
<td>5</td>
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<td>6</td>
<td>7</td>
<td>11</td>
</tr>
<tr>
<td>6</td>
<td>6</td>
<td>9</td>
</tr>
<tr>
<td>6⁄4</td>
<td>6</td>
<td>10</td>
</tr>
<tr>
<td>7</td>
<td>6</td>
<td>9</td>
</tr>
<tr>
<td>7⁄4</td>
<td>6</td>
<td>9</td>
</tr>
<tr>
<td>8</td>
<td>0</td>
<td>0</td>
</tr>
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of lactic acid and other fixed acids formed by anaerobic glycolysis. These earlier workers were able to show that the rise of lactic acid in the blood was to two or three times its normal level. It would be expected that such a biochemical picture would be found in the present work on rabbits. The lactic acid is produced by the anaerobic glycolysis of pyruvic acid and therefore must be related to the amount of work done by the muscles. In electrical asphyxiation the muscles of the upper limbs and thorax are held in a state of tetanic contraction throughout the shock and, with the accompanying hypoxia, we assumed that the work done and, hence, the amount of lactic acid produced would be greater than in obstructive asphyxiation.

The results show that, although the rise and fall in blood pressure were similar in the two groups of animals, the mean time taken for the blood pressure

TABLE 3
CARBON DIOXIDE TENSION FOR OBSTRUCTIVE AND ELECTRICAL ASPHYXIATION (mmHg)

<table>
<thead>
<tr>
<th>Minutes</th>
<th>Obstructive asphyxiation</th>
<th>Electrical asphyxiation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Range</td>
<td>No. of readings</td>
</tr>
<tr>
<td></td>
<td>Max.</td>
<td>Min.</td>
</tr>
<tr>
<td>0</td>
<td>31</td>
<td>39</td>
</tr>
<tr>
<td>1⁄2</td>
<td>41</td>
<td>50</td>
</tr>
<tr>
<td>1</td>
<td>44</td>
<td>53</td>
</tr>
<tr>
<td>1⁄4</td>
<td>48</td>
<td>56</td>
</tr>
<tr>
<td>2</td>
<td>52</td>
<td>62</td>
</tr>
<tr>
<td>3⁄4</td>
<td>54</td>
<td>66</td>
</tr>
<tr>
<td>3</td>
<td>58</td>
<td>74</td>
</tr>
<tr>
<td>3⁄4</td>
<td>60</td>
<td>76</td>
</tr>
<tr>
<td>4</td>
<td>63</td>
<td>84</td>
</tr>
<tr>
<td>4⁄4</td>
<td>70</td>
<td>90</td>
</tr>
<tr>
<td>5</td>
<td>70</td>
<td>92</td>
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<tr>
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<td>75</td>
<td>80</td>
</tr>
<tr>
<td>7</td>
<td>79</td>
<td>82</td>
</tr>
<tr>
<td>7⁄4</td>
<td>81</td>
<td>82</td>
</tr>
</tbody>
</table>
to reach zero in electrical asphyxiation was 2½ minutes less than in obstructive asphyxiation. The reason for this difference may be found in one or more of three groups of recorded physiological data. These are the blood gas changes, the acid-base balance changes, and mechanical respiratory activity. It is, however, not possible to exclude the direct effect of the electric shock on the myocardium. This will be discussed later.

With regard to the blood gas changes, the rates of development of hypoxia and hypercapnia were very similar in the two experimental series. It might have been expected that during electrical asphyxiation, with the muscles of the thorax and fore-limbs in a state of tetanic contraction, the oxygen consumption, and hence the fall in oxygen tension, would be greater than that shown in obstructive asphyxiation. This, however, was found not to be the case, although when converted to oxygen saturations the results indicate that in electrical asphyxiation the blood becomes desaturated at a slightly faster rate. The rate of development of hypercapnia was again similar in the two series of experiments although, from the above reasoning, one would have expected the hypercapnia to develop at a faster rate during electrical asphyxiation. It is possible that apneustic diffusion respiration (Bartlett, Brubach, and Specht, 1959) occurs in electrical asphyxiation because, although the chest is held in tetanic contraction, the trachea is open to the atmosphere. However, three supplementary experiments have shown that clamping the trachea during the electrical asphyxiation of rabbits does not produce different results from the presented work in the rate of development of hypercapnia.

The acid-base changes in the two series are markedly different. The results of the pH changes show that during electrical asphyxiation, the mixed acidosis which occurs is extremely severe and occurs in a much shorter time than the less severe acidosis shown in obstructive asphyxiation. As the degree of respiratory acidosis, as exemplified by the carbon
TABLE 5
LACTIC ACID FOR OBSTRUCTIVE AND ELECTRICAL ASPHYXIATION (mg/100 ml.)

<table>
<thead>
<tr>
<th>Minutes</th>
<th>Obstructive asphyxiation</th>
<th>Electrical asphyxiation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>Range</td>
</tr>
<tr>
<td></td>
<td>Max.</td>
<td>Min.</td>
</tr>
<tr>
<td>0</td>
<td>40.4</td>
<td>56.0</td>
</tr>
<tr>
<td>1/2</td>
<td>45.8</td>
<td>76.0</td>
</tr>
<tr>
<td>1</td>
<td>60.6</td>
<td>92.5</td>
</tr>
<tr>
<td>2/3</td>
<td>84.9</td>
<td>135.4</td>
</tr>
</tbody>
</table>

TABLE 5
LACTIC ACID FOR OBSTRUCTIVE AND ELECTRICAL ASPHYXIATION (mg/100 ml.)

![Graph](https://via.placeholder.com/150)

**FIG. 4.** Lactic acid obstructive (○) and electrical (●) asphyxiation.
carried out by Swann and Brucer (1951) for obstruc-
tive asphyxiation in dogs. During electrical
asphyxiation no respiratory movements could occur
and, because the shock was continuous, we have no
real knowledge of the respiratory potentialities of
such an animal. However, from a few of our earlier
experiments, using shocks of from 30 seconds to
4 minutes’ duration, we found that spontaneous
respiratory activity stopped somewhere between
2 and 3 minutes after the start of the shock. The
important point to consider from this information
on respiratory activity is the help given to the
venous return, and hence the blood pressure and
circulation, by the mechanical act of respiration
during obstructive asphyxiation. Such circulatory
aid is not present at any time during electrical
asphyxiation.

This present study is concerned primarily with
the understanding of the changes that take place
during long, non-fibrillating electric shocks. There
may be a tendency to look upon the respiratory and
circulatory systems as separate entities but both
are completely interdependent. Without survival
experiments it is difficult to decide which of these
two systems is the weaker link, that is, which one
fails and is irrecoverable first during the shock.

Conclusions
It has been shown that the mechanism of death in
both electrical and obstructive asphyxiation is
essentially similar. In obstructive asphyxiation, the
primary biochemical changes associated with
hypoxia are an initial respiratory acidosis followed
by an uncompensated metabolic acidosis. In
electrical asphyxiation, the metabolic acidosis is
present virtually from the beginning of the shock
and rapidly becomes very pronounced. This has
been shown to be due mainly to the anaerobic
formation of lactic acid.

The main findings suggest that in electrical
asphyxiation circulatory collapse occurs sooner than
in obstructive asphyxiation, that this is due to the
decreased venous return resulting from absence of
respiratory movements, and that this effect is
probably enhanced by the effect of the fulminating
metabolic acidosis on the cardiac output. Both early
circulatory collapse and severe metabolic acidosis
could be caused by the direct effect of the electric
shock on the myocardium as well as the muscles
of the upper limbs and thorax. From the presented
results and the nature of cardiac muscle as compared
to voluntary muscle, it would seem more likely that
acid-base changes via the voluntary muscles were
affecting the myocardial function rather than that
myocardial changes were causing these changes
directly. The more rapid circulatory collapse
together with the earlier disappearance of spon-
taneous respiratory activity, the cause of which has
not been investigated, indicate that accidents in
which this mechanism is operating have an urgency
which is greater than in obstructive asphyxiation.

This investigation was supported by grants from the
Medical Research Council and the Central Electricity
Generating Board. I wish to extend my gratitude to
Dr. W. R. Lee for guidance and to Mr. C. L. Tomlin
for very able assistance.

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