Physiological changes during electrical asphyxiation¹

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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 resuscitatory 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 (Burttau, 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-

¹This paper is based on work presented in an M.Sc. thesis.

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taneously to sinus rhythm from ventricular fibrillation (Ferris *et al.*, 1936). This means that accidental ventricular fibrillation could be discounted as a cause of death. The experimental work was performed in two groups. The first dealt with 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 first performed and an endotracheal tube was tied into place. A respiratory transducer attached to the endotracheal tube allowed respiration to be monitored (Lee, Zoladzowski, 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 (Telequipment, 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.S.30), 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 3½ 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

FIG. 1. Electrical asphyxiation – experimental design.

TABLE 1
BLOOD PRESSURE FOR OBSTRUCTIVE AND ELECTRICAL ASPHYXIATION (mmHg)

Minutes	Obstructive asphyxiation						Electrical asphyxiation									
	Mean S D		Range				No. of readings S D	Mean S D		Range				No. of readings S D		
			Max. S D		Min. S D					Range						
										Max.		Min.				
	S	D	S	D	S	D	S	D	S	D	S	D	S	D		
0	102	89	130	96	60	50	11	9	102	87	120	115	45	35	13	13
$\frac{1}{2}$	145	104	180	120	70	50	11	9	138	101	170	120	80	55	13	13
1	140	100	170	120	80	55	11	9	116	81	150	120	65	35	13	13
$1\frac{1}{2}$	138	100	170	120	70	50	11	9	94	59	130	90	52	13	13	13
2	139	93	200	120	55	45	11	9	76	47	100	80	45	13	13	13
$2\frac{1}{2}$	126	86	200	120	60	40	11	9	60	28	90	55	30	10	13	13
3	114	77	180	120	50	37	11	9	47	20	70	40	12	5	14	14
$3\frac{1}{2}$	101	70	170	120	35	25	11	9	27	13	80	50	0	0	14	14
4	82	60	145	120	30	27	11	9	19	8	70	30	0	0	10	10
$4\frac{1}{2}$	66	46	130	100	27	12	11	9	3	1	18	8	0	0	7	7
5	51	33	120	85	8	5	11	9	0	0	0	0	0	0	0	0
$5\frac{1}{2}$	31	21	95	70	0	0	11	9								
6	33	23	80	60	0	0		7	6							
$6\frac{1}{2}$	34	22	70	50	10	8		5	4							
7	23	14	60	30	0	5		5	4							
$7\frac{1}{2}$	14	6	45	20	0	0		4	4							
8	10	5	20	10	0	0		2	2							
$8\frac{1}{2}$	14	5	14	5	—	—		1	1							
9	0	0	0	0	0	0		0	0							

S = Systolic pressure D = diastolic pressure

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 resuscitatory 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

FIG. 2. Systolic and diastolic blood pressure. ○—○ obstructive asphyxiation; ●---● electrical asphyxiation.

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

Minutes	Obstructive asphyxiation				Electrical asphyxiation			
	Mean	Range		No. of readings	Mean	Range		No. of readings
		Max.	Min.			Max.	Min.	
0	74	104	53	11	80	110	50	14
$\frac{1}{2}$	38	42	32	11	43	61	20	14
1	30	36	22	11	34	45	18	14
$1\frac{1}{2}$	25	33	15	11	28	37	16	14
2	20	28	11	11	26	36	12	14
$2\frac{1}{2}$	18	26	10	11	24	34	11	14
3	15	21	8	11	23	32	9	13
$3\frac{1}{2}$	12	18	7	11	21	29	7	12
4	11	16	2	11	20	28	7	10
$4\frac{1}{2}$	10	12	5	11	21	24	16	5
5	9	11	3	10	14		14	1
$5\frac{1}{2}$	7	11	3	8				
6	6	9	2	6				
$6\frac{1}{2}$	6	10	2	5				
7	6	9	2	3				
$7\frac{1}{2}$	6	9	2	2				
8	0	0	0	0				

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)

Minutes	Obstructive asphyxiation				Electrical asphyxiation			
	Mean	Range		No. of readings	Mean	Range		No. of readings
		Max.	Min.			Max.	Min.	
0	31	39	27	11	23	33	14	14
$\frac{1}{2}$	41	50	35	11	37	53	20	14
1	44	53	43	11	42	50	77	14
$1\frac{1}{2}$	48	56	40	11	45	77	31	14
2	52	64	41	11	55	100	35	14
$2\frac{1}{2}$	54	66	45	11	59	110	35	14
3	58	74	44	11	61	115	30	14
$3\frac{1}{2}$	60	76	45	11	66	119	40	12
4	63	84	51	11	73	121	45	10
$4\frac{1}{2}$	70	90	50	11	73	92	62	5
5	70	92	59	10	70		70	1
$5\frac{1}{2}$	68	82	61	7				
6	74	83	68	7				
$6\frac{1}{2}$	75	80	74	5				
7	79	82	78	3				
$7\frac{1}{2}$	81	82	80	2				

TABLE 4
pH FOR OBSTRUCTIVE AND ELECTRICAL ASPHYXIACTION

Minutes	Obstructive asphyxiation				Electrical asphyxiation			
	Mean	Range		No. of readings	Mean	Range		No. of readings
		Max.	Min.			Max.	Min.	
0	7.398	7.47	7.28	11	7.429	7.53	7.32	14
$\frac{1}{2}$	7.345	7.44	7.20	11	7.285	7.42	7.21	14
1	7.39	7.40	7.17	11	7.157	7.25	7.11	14
$1\frac{1}{2}$	7.296	7.38	7.16	11	7.056	7.16	6.98	14
2	7.265	7.36	7.14	11	6.994	7.095	6.89	14
$2\frac{1}{2}$	7.247	7.36	7.11	11	6.946	7.08	6.845	14
3	7.25	7.34	7.09	11	6.906	7.075	6.82	14
$3\frac{1}{2}$	7.186	7.31	7.04	11	6.872	6.98	6.75	12
4	7.156	7.27	7.03	11	6.847	6.97	6.71	10
$4\frac{1}{2}$	7.124	7.24	6.99	11	6.745	6.79	6.70	4
5	7.103	7.19	6.97	10	6.71	6.71		1
$5\frac{1}{2}$	7.112	7.18	7.04	7				
6	7.09	7.13	7.04	6				
$6\frac{1}{2}$	7.052	7.13	7.02	5				
7	7.07	7.10	7.04	3				
$7\frac{1}{2}$	7.04	7.07	7.01	2				

to reach zero in electrical asphyxiation was $2\frac{1}{2}$ 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.

FIG. 3. Base excess for obstructive (○—○) and electrical (●—●) asphyxiation.

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.)

Minutes	Obstructive asphyxiation				Electrical asphyxiation			
	Mean	Range		No. of readings	Mean	Range		No. of readings
		Max.	Min.			Max.	Min.	
0	40.4	56.0	23.0	8	33.3	63.3	13.6	11
$\frac{1}{2}$								
1								
1½	45.8	76.0	30.4	7	84.9	113.5	57.8	10
2								
2½					109.4	159.4	68.8	7
3								
3½	60.6	92.5	38.5	7	114.7	188.6	75.7	6
4								
4½								
5								
5½	84.9	135.4	59.2	6				

dioxide tension, was the same in both groups of animals, the difference in the mean pH changes must, therefore, be caused by the metabolic acidosis. When expressed as base excess the combined pH and carbon dioxide tension changes show the difference very clearly, the fall in base excess during electrical asphyxiation being far steeper and to a much greater depth than that shown in obstructive asphyxiation. The measured amounts of lactic acid demonstrate the primary cause of the metabolic acidosis and show clearly the degree in both groups of animals to which the lactic acid in the circulating blood rose.

In both groups of animals, therefore, the rate of development of respiratory acidosis was virtually

the same, but, in electrical asphyxiation, the degree and rate of development of the metabolic acidosis was far greater than in obstructive asphyxiation. This difference in the acid-base balance probably contributes to the earlier circulatory failure in electrical asphyxiation, as it has been shown that a metabolic acidosis will decrease the cardiac output (Carson *et al.*, 1965). Although recorded right ventricular and carotid artery blood pressure measurements did not demonstrate any direct mechanistic effect of the electric shock on the myocardium, it is not possible to rule out this possibility. If the myocardium was in some way affected by the shock, such an effect would in some way depress or alter the myocardial muscle contractility and so reduce the cardiac output: this in itself would produce a metabolic acidosis and earlier cardiac standstill. Such a direct effect could, therefore, account for the physiological and biochemical events seen in electrical asphyxiation. The heart is, however, normally well protected against external stimuli. This is accomplished by means of the peculiar absolute refractory period (the R-T interval of the ECG) during which the myocardium cannot be stimulated into contraction. A more subtle alteration in contractility could occur, but this would be difficult to detect during electrical asphyxiation.

In the section on results, it was stated that during obstructive asphyxiation respiratory movements ceased after a mean time of 3.22 minutes. It is not known whether at this time positive-pressure insufflation of oxygen would have resuscitated these animals, and from our results it is not possible to speculate upon this. To find the point at which the respiratory system was irreversibly damaged would have required a detailed survival study of the kind

FIG. 4. Lactic acid obstructive (○—○) and electrical (●—●) asphyxiation.

carried out by Swann and Brucer (1951) for obstructive 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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Received for publication March 2, 1970.