DELAY IN RESTARTING RESPIRATION AFTER EXPERIMENTAL ELECTRIC SHOCK

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When respiration is arrested by a 50 c/s alternating current passed between the forelimbs of rabbits there may be a delay between the end of the shock and the spontaneous resumption of respiration. This paper examines the relation of such a delay with the duration of shock and the current magnitude independently and in terms of two physical concepts: (a) the product of shock duration and current magnitude, called here 'charge equivalent'; and (b) a quantity proportional to the energy input.

The influence of the shock duration apparently exceeded that of the current. Furthermore, delay was strongly associated with both 'charge equivalent' and energy input.

When temporary circulatory arrest due to ventricular fibrillation occurred, an additional mechanism appeared to operate. Although protracted, the delay showed similar associations with the shock duration and current magnitude.

An interesting observation was that the interval between spontaneous defibrillation and the resumption of respiration also showed a strong association with shock duration. The restarting of respiration appears to depend on the circulation. It may be that when the circulation restarts, after a period of ventricular fibrillation, blood-borne inhibitory substances, which accumulated during the period of circulatory arrest, could affect the respiratory centre.

In 1927 Urquhart found that when an alternating current of sufficient magnitude was passed directly through the medulla for a period of time 'a condition of profound paralysis or block became established in the nerve centres'. The presence of this block was manifested by 'the absence of the respiratory, vagal and conjunctival reflexes normally functioning through these centres'. After a certain interval following a break of the current, the paralysis or block passed off and the reflexes returned 'provided efficient artificial respiration is meanwhile applied and provided there has been no charring of the nerve structures'.

In a further study, Urquhart and Noble (1929) extended these observations to show that an alternating current applied directly along the spinal cord of a decerebrate cat caused a similar condition of temporary block in the spinal cord.

In the study of this 'a-c block' on respiration a current pathway from the back of the nose to the atlanto-occipital membrane was used. However, a

more common pathway in accidental electric shock is from one upper limb to another limb. In an experimental study by two of us (Lee and Zoledziowski, 1964) in which 50 c/s alternating currents were passed between the forelimbs, it was observed that there was sometimes a delay between the end of the shock and the spontaneous resumption of respiration. This paper examines the relation between this delay and the shock duration and current magnitude.

Method

The electrical circuit and the physiological recording systems have previously been described (Lee and Zoledziowski, 1964); only one modification was made in the later experiments. Difficulty had been experienced in recording blood pressure during the passage of the shock, due to the muscular contractions of the shock kinking the carotid artery where the cannula was inserted. To overcome this, a No. 2 polythene (1 mm. internal diameter) tube was

passed down the carotid artery so as to lie in the innominate artery or aorta (the position was checked at post-mortem examination).

Most of the results were obtained from different animals, although a few animals were used for more than one result. In an earlier study of delay in restarting respiration in cases without ventricular fibrillation consistent results had been found with first and subsequent shocks (Lee, 1963).

Findings

The search for the most appropriate formula to express the relation between delay in restarting respiration (d) and the variables, shock duration (t) and current (i), presents a complex problem. The solution of this problem should be susceptible of interpretation in terms of electrical concepts and physiological response. Therefore it was decided to examine only the relation between the delay and two simple physical concepts: the product of shock duration and r.m.s. value of the current ($i \times t$, in this paper called the 'charge equivalent') and a quantity which is proportional to the energy input ($i \times t^2$). The independent effects of shock duration and current were also examined.

When ventricular fibrillation occurred another variable was added which might affect the duration of the delay. For this reason, the experiments in which fibrillation occurred are considered separately.

No Ventricular Fibrillation.—On 12 occasions there was no evidence to suggest that fibrillation had occurred; details of these experiments are given in Table 1.

Delay was found to be related to both shock duration and current. The regression equations obtained and the corresponding correlation coefficients are:

$$d = 0.01 i + 1.43 t - 15.46 (\pm 2.35); R = 0.98. \quad (1)$$

$$d = 0.0014 it - 1.3361 (\pm 3.66); r = 0.98 \quad \dots (2)$$

$$d = 0.0001 i^2 t - 2.36 (\pm 4.52); r = 0.94 \quad \dots (3)$$

TABLE 1
NO VENTRICULAR FIBRILLATION

Current (mA; r.m.s.)	Shock Duration (sec.)	Delay (sec.)
220	10	0
520	10	1
535	10	2
200	10	3
2,000	2	9
950	9	11
950	10	13
950	10	13
950	12	16
2,150	5	16
1,000	15	18
950	40	52

where d = delay (sec.), i = current (mA), and t = shock duration (sec.).

All these relationships yield highly significant correlation coefficients ($P < 0.001$) and provide strong support for the assumption that the regression between delay and the other variates is linear. There is, however, no conclusive evidence to show which of them is the most representative. For simplicity, only the relationship between delay and the 'charge equivalent' (equation 2) is illustrated in Figure 1.

FIG. 1.—No ventricular fibrillation. Delay in restarting respiration by current $\times$ shock duration ($r = 0.98$; $P < 0.001$).

The independent effects of shock duration and of current on delay were studied from equation 1. When the current is held constant there is a strong association between shock duration and delay (see Figure 2). The partial correlation coefficient of 0.97 is highly significant ($P < 0.001$). On the other hand, with shock duration held constant, the association between current and delay shown in Fig. 3 is less marked with a partial correlation coefficient of 0.52.

The graphs, Figs. 2 and 3, are derived from calculations illustrating the relationships existing between individual results. These are set against ordinates and abscissae of the appropriate units with arbitrary zero points. The same applies to Figure 5.

Therefore, although this study does not explain the delay in restarting respiration, it indicates that the influence of shock duration is greater than that of the current.

FIG. 2.—No ventricular fibrillation. Delay in restarting respiration by shock duration, current held constant. Note: $d_{(i)}$ and $i_{(i)}$ were calculated from the simple regressions of d and i on t . ($d - d_{(i)}$) and ($i - i_{(i)}$) represent the variations which would occur in d and i were t held constant.

FIG. 3.—No ventricular fibrillation. Delay in restarting respiration by current, shock duration held constant. Note: $d_{(t)}$ and $t_{(t)}$ were calculated from the simple regressions of d and t on i . ($d - d_{(t)}$) and ($t - t_{(t)}$) represent the variations which would occur in d and t were i held constant.

Ventricular Fibrillation.—Table 2 gives details of the 22 shocks in which ventricular fibrillation

TABLE 2
VENTRICULAR FIBRILLATION

Current (mA; r.m.s.)	Shock Duration (sec.)	Delay (sec.)	Time from	
			End of Shock to Defibrilla- tion (sec.)	Start of Fibril- lation to End of Shock (sec.)
100	15	4	4	Unknown
120	35	10	0	34
360	18	22	20	Unknown
370	60	60	12	56
420	9	13	10	Unknown
530	15	20	0	Unknown
600	5	10	10	Unknown
600	5	12	37	Unknown
700	5	0	5	Unknown
900	5	3	3	Unknown
825	30	76	2	28
830	30	61	2	26
700	13	13	1	9
700	20	15	2	18
1,500	10	22	8	Unknown
2,200	5	20	0	Unknown
900	10	33	20	Unknown
925	20	42	7	12
950	4	11	1	Unknown
1,080	5	18	7	Unknown
1,000	25	40	Unknown	Unknown
1,050	5	18	Unknown	Unknown

occurred and in which there was a spontaneous reversion to normal heart rhythm. Ettinger (1933) distinguished in rabbits between ventricular fibrillation which spontaneously reverted only rarely and ventricular flutter which often spontaneously reverts to normal rhythm. For the present study the distinction is immaterial as the result of either is circulatory arrest, and the term ventricular fibrillation will be used throughout.

In this group also, the delay was found to be associated ($P < 0.001$) with both shock duration and current. A regression equation and correlation coefficient for the 'charge equivalent' passed are:

$$d = 0.0022 it + 1.2 (\pm 9.05); r = 0.89$$

The regression coefficient differs significantly ($t = 2.8$; $P < 0.01$) from that found for the non-fibrillating animals. The difference in slope of the regression lines is shown in Figure 4. It is apparent that the animals which had ventricular fibrillation had a protracted delay compared with those which did not develop ventricular fibrillation.

The independent effects of shock duration and current were studied as before. There is a strong association between delay in restarting respiration and shock duration; the partial correlation coefficient of 0.87 is highly significant ($P < 0.001$). The association between current and delay is less strong; the partial correlation coefficient is 0.47 ($P < 0.05$). Thus, as in the experiments in which there was no fibrillation, it appears that shock duration plays a more important role than the current in causing a delay in restarting respiration after the end of the shock.

In 20 experiments respiration restarted after

FIG. 4.—Ventricular fibrillation. Delay in restarting respiration by current $\times$ shock duration ($r = 0.89$; $P < 0.001$).
(i) Regression line when ventricular fibrillation occurred.
(ii) Regression line when ventricular fibrillation did not occur.

spontaneous defibrillation had occurred. Figure 5 shows the association between shock duration and the interval elapsing between defibrillation and the spontaneous resumption of respiration when the current is held constant. There is a significant ($P < 0.01$) partial correlation coefficient of 0.81. The

FIG. 5.—Time between defibrillation and spontaneous resumption of respiration (y) by shock duration, current held constant. Note: $y_{(i)}$ and $t_{(i)}$ were calculated from the simple regressions of y and t on i . ($y - y_{(i)}$) and ($t - t_{(i)}$) represent the variations which would occur in y and t were i held constant.

association between the current and the interval from defibrillation to resumption of respiration is not so close; the partial correlation coefficient of 0.53 is possibly significant ($P < 0.05$).

There is no *a priori* reason to suppose that the duration of fibrillation after the shock would be related either to current or to shock duration, and this was found to be the case, the partial correlation coefficients being -0.23 and -0.16 respectively.

Discussion

It must be stressed that these results are only valid within the limits shown in Table 1, that is, current values from 0.2 to 2.15 amperes and duration of shock from 2 to 40 seconds. In fact, as shown in our previous paper, in the absence of fibrillation delay in restarting respiration was not observed with shock currents of less than 200 mA no matter how long they were continued. This is comparable with the observations on 'a-c block' of isolated sciatic nerve by Urquhart and Noble (1929), who found that a definite threshold of current was required before block was induced.

Examination of the present results throws some light on the physiological disturbance underlying the delay in onset of respiration after its arrest by an electric current. That the responsible mechanism is physiological rather than electrical is strongly suggested by the finding that there is a closer relationship between delay and shock duration than between delay and current, whether expressed as charge equivalent or energy input.

Any physiological explanation must be in accord with the fact that the apnoeic period persists in spite of the joint stimuli to respiration provided by arterial oxygen desaturation, carbon dioxide retention and 'acidosis' inevitably accompanying the inhibition of respiration, and the muscular contractions associated with the passage of the current. These stimuli will be greater the longer the duration of the shock, whereas paradoxically in this respect we have observed that the longer the shock, the longer the resultant apnoea. Possible mechanisms are as follows:

(a) Muscle fatigue will not serve as an adequate explanation for the delay in restarting respiration because it has been shown that in this preparation all the respiratory muscles are in tetanic contraction when currents greater than 60 mA are passed (Lee and Zoladzowski, 1964). By the 'all or none' law of physiological response the muscle fibres will be in no greater state of fatigue merely by increasing the magnitude of the stimulus, yet no delay has been observed with currents less than 200 mA.

(b) A direct inhibitory effect on either the respiratory centre or its efferent nerves (Urquhart

FIG. 6.—Schematic representation of the experimental record in a case of ventricular fibrillation.
 i = shock current of duration t
 d = delay from end of shock to spontaneous resumption of respiration
 y = time from defibrillation to spontaneous resumption of respiration

and Noble, 1929) could be contributory. This presupposed an effective current density in the area, and the fact that apnoea ensues only after currents greater than 200 mA (Lee and Zoledziowski, 1964) suggests that there is a 'threshold effect'.

However, neither of these explanations is adequate in itself to account for our findings in the animals that developed ventricular fibrillation. In particular, there is the observation that as the shock duration increased (for currents of the same strength) the interval (y in Fig. 6) between spontaneous defibrillation and resumption of respiration also increased and that the statistical association between these two increases was significant at the 1% level. In other words, respiration restarted a certain time after the circulation had restarted, and this time increased with shock duration but was less strongly influenced by the current strength. The restarting of respiration appears to depend on the circulation.

The shock duration was found to be closely associated with duration of fibrillation as follows. The difficulties in recording blood pressure accurately during the shock have already been noted. However, on seven occasions a good blood pressure tracing was obtained during the shock, and it was noted that when fibrillation did occur it did so within eight seconds of the start of the shock. Despite the small numbers there was a strong association between the shock duration and the duration of fibrillation (Fig. 7) ($r = 0.93$; $P < 0.05$). In view of the consistently early start of the fibrillation this result would be expected. But it follows that when ventricular fibrillation occurred a shock of long duration was associated with a long period of circulatory arrest. Combining this observation with that in the preceding paragraph, on the association between shock duration and the interval y (in Fig. 6) between

defibrillation and the resumption of respiration, it appears that as the period of circulatory arrest increased so also did the interval y .

One explanation is that some inhibitory substance accumulates in the respiratory centre during the shock, and the longer the shock the more of the substance accumulates. When the circulation starts again this substance is removed and the more that has accumulated the longer will its removal take. But on this hypothesis the 600 mA for 5 seconds shock which resulted in a delay of 12 seconds (Table 2) is inexplicable because breathing restarted before the circulation.

Alternatively, it is possible that an inhibitory

FIG. 7.—Duration of fibrillation by shock duration.

substance accumulates in the body during the shock, and again, the longer the shock the more of the substance accumulates. Immediately the circulation starts again the inhibitory substance is carried to the respiratory centre which can only start to function if the continuing circulation now removes the substance. Mackay (1947) showed that narcotic levels of carbon dioxide caused a transient depression of respiration in cats, but in those experiments there was no gross anoxia which undoubtedly occurred in the present investigation. The probability that high concentrations of carbon dioxide, possibly in association with the accompanying profound acidosis, might produce respiratory depression was suggested by Lambertsen (1961). Our records did not show hyperventilation when respiration restarted, but there is no reason to suppose that a severely anoxic respiratory centre exposed to high concentrations of these inhibitory substances will behave in the same manner as a normal or mildly anoxic centre exposed to relatively smaller changes in pH and carbon dioxide.

It is postulated that at the time of defibrillation the anoxic respiratory centre is flooded with high concentrations of acidic metabolites, including CO₂, which depress its activity. It is not until the concentration of these has been reduced by redistribution in the rest of the body fluids and by diffusion of carbon dioxide out of the alveoli that the level of

carbon dioxide in the blood falls sufficiently to stimulate the respiratory centre so that breathing starts again.

Conclusion

The cause of the 'a-c block' first described by Urquhart nearly 40 years ago is still unexplained. It is possible that this phenomenon is related to the delay that occurs before respiration is spontaneously resumed after an A.C. electric shock passed between the forelimbs. When ventricular fibrillation occurs it seems that an additional mechanism operates, and inhibitory substances, which have accumulated during the period of circulatory arrest and are carried by the blood, might affect the respiratory centre.

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