ARTIFICIAL RESPIRATION

THE NEED FOR A GREATLY INCREASED RATE IN ASPHYXIA

BY

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The rate in all methods and teachings of artificial respiration varies between 12 and 18 times a minute. This rate has persisted to the present time, unaltered and unchallenged for all conditions and requirements. The purpose of this article is simply to demonstrate the obvious error or oversight in the application of *all methods* of artificial respiration to cases of asphyxia without at the same time considering the greatly increased respiratory requirements existing in asphyxia and making some attempt to meet these requirements. (The word "asphyxia" is used throughout as being synonymous with "suffocation" and "cyanosis.")

All cases for which artificial respiration is required may be considered to fall into two main groups:—Group 1: cases *without asphyxia*—e.g., under anaesthesia, from electric shock, etc.; Group 2: cases *with asphyxia*—e.g., *suffocation* from drowning, from lack of air (buried in bomb debris), from smoke, gases, etc. Any Group 1 case, if untreated, quickly becomes a Group 2 case.

Artificial respiration done at the usual rate of 15 times a minute is adequate and excellent for Group 1 cases, as the respiratory requirements are approximately the same as in natural respiration at rest; *but the same rate is quite inadequate to deal satisfactorily with Group 2 cases of asphyxia*, in which the respiratory requirements are very much greater. The body not only provides a natural respiration at rest—namely, a tidal breath every four seconds—but also provides it for all normal degrees of exercise and asphyxia whenever they arise in daily life. This natural respiration in asphyxia makes use not only of tidal but also of full complementary breathing at whatever increased rate may be required to deal with existing needs.

The truth of these statements can very easily be demonstrated, by simply holding the breath *as long as possible* and then, on the resumption of breathing, noting the volume and rate of the very necessary respirations. It will be found that slow tidal breathing is most inadequate, *if not frankly impossible*—and this from a very mild degree of asphyxia. Now consider the deeply asphyxiated person. He must certainly have *far greater respiratory requirements*, for he has passed more milestones along the road to death; he has lost consciousness, his respiratory centre has ceased to function—only his heart clings stubbornly, though precariously, to life. He is metaphorically shouting aloud for air—and is in actual fact dying for want of it. Obviously his respiratory requirements cannot be adequately

the volume or the rate. It may be assumed that each method of artificial respiration at present in use provides the greatest exchange of air possible by that method, so that only by increasing the rate can any greatly increased respiration be obtained. Fortunately, the rate can be raised in all methods with little effort, with no loss in efficiency, and with *great increase* of the total respiratory exchanges.

It is convenient and correct to consider the increasing depths of asphyxia as being of three degrees: Degree 1, up to loss of consciousness; Degree 2, up to cessation of respiration; Degree 3, up to final heart failure. It should be realized that it is only in Degree 1 that actual experiments on the state of asphyxia can be carried out, for the deeper degrees of asphyxia occur only accidentally and not under circumstances suitable for experimenting; while it is not practicable that any such asphyxia should be produced "artificially by force" for experimental purposes. It is, however, quite practicable to produce varying depths of Degree 1 asphyxia at will, and without danger, by simply holding the breath for varying lengths of time. It is from experiments in such self-administered asphyxia that we are obliged to gain our knowledge of the manner in which asphyxia may be relieved by varying amounts and rates of respiration.

Experiments in Asphyxia

Each experiment is done in the sitting position, and is started by taking a full, free complementary breath (without forcing inspiration to the utmost) and holding the breath for various stated periods. At least five minutes of natural respiration at rest is enforced before each experiment.

In these recorded experiments a subject has been chosen with sufficient determination to hold his breath for long periods, and of sufficient integrity to report the times at which "the pulse commences to be heard" on holding the breath, and, on resuming respiration, the times at which "the pulse ceases to be heard" and the "acute discomfort ceases." This latter somewhat arbitrary time is that at which one no longer has to "fight for more air," though deep rapid breathing may continue for some minutes.

The tidal respiration used in Series 2 has been judged by the subject to the best of his ability. It is quite feasible for the subject (by conscious control) to breathe an approximately similar amount to the tidal at any time or rate required. That this amount should be exactly the tidal is, of course, unlikely, but any respiratory experiment on the conscious subject is open to error of the "human element," and in these, even though the results were affected thereby very considerably, the same fundamental conclusions could and would be drawn.

Series 1 experiments (see Table) shows results of varying degrees of asphyxia on the natural *uncontrolled respiration*. As the asphyxia becomes deeper, though the respiratory exchanges are greater the recovery is slower. Asphyxia from only two minutes' stoppage of respiration (Experiment 3) causes the volume of each respiration to be increased at least twice, and the rate to be increased four times. Thus the total volume of air exchanged naturally after only

Experiments in Asphyxia

Experiment No.	Time Breath held (Seconds)	Ear Pulse heard (Seconds)	Type of Respiration used	Rate of Respiration	Ear Pulse ceased (Seconds)	Time taken to be Free of Discomfort (Seconds)	Remarks
Series 1	1 ..	60	Natural	6 in 10 secs.		8	Tidal plus complementary breathing used naturally without control. Volume of each respiration increased to full in Exp. 1, with marked increase in rate also. Rate further increased to as fast as possible in Exp. 4
	2 ..	90	"	9 " 10 "		10	
	3 ..	120	"	12 " 12 "	8	14	
	4 ..	150	"	30 " 20 "	15	20	
Series 2	5 ..	120	Controlled tidal	1 in 4 secs.	Constant	Discomfort increased	Very difficult; asphyxia growing worse all the time. By great effort experiment continued for 40 secs., when will-power dulled and natural respiration reasserted itself. Relief definite but very slow. Relief much improved over Exp. 6. Practically as good relief as in Exp. 3
	6 ..	120	"	1 " 2 "	45	c. 60	
	7 ..	120	"	1 " 1 sec.	15	c. 30	
	8 ..	120	Forced rapid tidal	2 " 1 "	10	15	
9 ..	60		Controlled tidal	1 in 4 secs.		c. 60	Relief definite but very slow

met by a tidal breath once in four seconds. Yet that is the manner in which it is being administered to-day to the deeply asphyxiated.

On the truth of these simple and obvious facts, which have never before been applied practically to this vital need, I base my claim for the necessity for providing increased respiration in asphyxia. This increase can be obtained only by raising

a moderate Degree 1 asphyxia is *at least eight times* the total volume exchanged naturally at rest for the same unit of time. (The total volume exchanged by any artificial method at present is seldom much greater than the total volume exchanged naturally at rest.)

Series 2 experiments show the effect on a constant degree of asphyxia obtained by altering only the rate—the volume remaining tidal throughout. Exp. 5 shows that the rate of 1 in 4 seconds provides insufficient respiratory exchange to relieve this particular

degree of asphyxia, and the experiment had to be broken off because of increasing asphyxia. Exp. 6 shows that by increasing the rate to 1 in 2 seconds the relief is definite but very slow. Exp. 7 shows that by increasing the rate to 1 in 1 second the relief is much improved. Exp. 8 shows that by further increasing the rate to 2 in 1 second (in the same manner as a dog pants) the relief is almost as good as by natural breathing in Exp. 3. (This rate of 2 in 1 second is not practicable artificially, but it shows that there is no effective limit to the practical increase in rate.) Exp. 9 shows that tidal breathing 1 in 4 seconds may relieve a very mild asphyxia, but fails to relieve a deeper asphyxia (compare Exp. 5).

Conclusions from Experiments in Degree 1 Asphyxia

1. The deeper the asphyxia the greater the respiratory requirement.
2. In any asphyxia that needs artificial respiration the respiratory requirement must be *greatly in excess of eight times* the normal resting requirement.
3. In the deeper degrees of asphyxia a greater respiratory exchange than in the lesser degrees is required to give an appreciable relief.
4. The total volume of air exchanged in a given time largely determines the extent of relief from asphyxia.
5. The faster the rate of tidal respiration the greater the relief from asphyxia.

Discussion

The above conclusions are undoubtedly true in Degree 1 asphyxia. The respiratory requirements of Degree 3 are essentially the same in quality as in Degree 1, and vary only in quantity. Degree 3 asphyxia should therefore respond to increased respiratory exchange in much the same manner as does Degree 1—*provided the circulation is adequate to take up and distribute the extra oxygen made available.*

The circulation, which remains fairly constant in Degree 1, becomes gradually more and more enfeebled and inadequate throughout Degree 3. It must therefore be fully realized that in Degree 3 asphyxia *the state of the circulation may play as great a part in determining the extent of relief from asphyxia as the respiratory exchanges do.* They are very closely inter-related. The circulation is now directly dependent on the respiration. The only possible way to improve the circulation is to ensure that a fully adequate supply of oxygen is available in the lungs, to be transported to the needy cardiac muscle. In brief, in a hypothetical case, if the circulation is still fairly adequate (the radial pulse being discernible) the recovery obviously must be much quickened by using a greatly increased rate. If, however, owing to long asphyxia, the heart is failing fast and the circulation is hopelessly inadequate, so that a tidal breath once in four seconds already provides more oxygen than can be taken up by the greatly depleted pulmonary circulation, there is nothing to be gained by increasing the respirations, as the patient would seem to be doomed, whatever may be done. It is, however, in the border-line case, in which the circulation is able to take up all the oxygen available at the usual rate and still be wanting more, that the greatest benefit will result from an increased rate. For in this case of deep asphyxia with failing circulation the tidal breath once in four seconds is inadequate to clear up the deep existing cyanosis and to maintain in addition the heart and body requirements (as shown by Exp. 5). Such cases have no doubt provided most of the recorded failures. By increasing the rate, and providing about four times as much air, not only do we give the patient a "much better chance," but it is not unreasonable to assume that some such cases, previously "on the down grade" at the usual rate, would revive with the increased rate. It must further be borne in mind that the blood in such a case is almost completely devoid of oxygen, and therefore can take up far more of it, volume for volume, than the blood in Degree 1, which can take up eight times the normal amount very readily. Thus, even though the flow of blood through the lungs may be very poor indeed, it is quite possible that all the oxygen available from tidal breaths every four seconds may be fully taken up by this deeply cyanotic blood, which then is left "hungering" for more oxygen. Therefore, it is essential to provide *all the respiratory exchanges possible* if every case is to have its best chance of recovery. It may, at least, confidently be stated that an increased rate can do no harm in any case, and must in many instances do much good.

It has been shown (Exp. 8) that the effectiveness of increasing the rate of tidal breathing continues beyond any practicable limit—provided the circulation is adequate; and as any hopelessly inadequate circulation cannot be recognized on examination, the only safe rule to adopt in all cases is to employ as fast a rate as may be efficiently possible.

It is not essential to discuss the relative parts played in asphyxia by the diminishing oxygen supply and the increasing carbon dioxide, or the varying effects of asphyxia on the different nerve cells or its effect on the cardiac muscle. All such considerations are of little importance from the practical point of view, because *all manifestations of asphyxia, of whatever nature or degree, are the direct result of respiratory failure, and can only be improved by providing adequate respiratory exchanges.*

The Rate Suggested

In every method the present rate is slow. Four seconds is a long time when applied to one respiratory cycle. Indeed, in most methods it has been necessary to include a pause of one to two seconds in each cycle in order to keep the rate down to the so-called normal. In Schäfer's the interval is at the end of inspiration; in Eve's it is at the end of expiration. There has been considerable discussion as to the proper place in the cycle for this interval and the proper timing. My only comment is to draw attention to the undoubted fact that in natural respiration for even *mild* degrees of asphyxia there is *no time for a pause at all at any period*, inspiration and expiration being as quick as possible, and following each other as rapidly as possible. In all artificial methods the same manner of respiration should be provided.

In Schäfer's method it is difficult to occupy fully even three seconds with the actual movements necessary for one cycle. It is, in fact, much easier, and just as effective, to do all the movements in two seconds instead of three. The second so saved could be utilized, along with the second previously set aside for an interval, by providing the two seconds necessary for another cycle. Thus the rate may be doubled, without any undue effort at all, by merely substituting an easier rate of 30 times a minute. Now, in the interest of the dying patient some real physical effort should be made, and under most conditions this rate of 30 times a minute can be doubled. It is not difficult to do Schäfer's method at a rate of 60 times a minute without any loss of volume per respiration. Under few circumstances, with an adult operator, should it be necessary to let the rate drop very much below 60.

Silvester's method, because of the nature of the movements necessary, takes more time to perform than Schäfer's, and a rate of about 45 times a minute would seem to be the efficient maximum. My limited experience of Eve's method does not permit me to suggest the most suitable rate, but the same rule holds true—"as fast as possible consistent with no sacrifice in volume"—and I have little doubt that the efficient rate of rocking could be at least doubled, and probably much further increased.

Considering the number of cases of asphyxia occurring on land and sea under war conditions, the early adoption of this principle, and its teaching to all appropriate personnel, cannot be too strongly urged.

Conclusion

The respiratory requirements are so great in all cases of asphyxia which require artificial respiration that, in such cases, the adoption in all methods of as fast a rate as possible, consistent with efficiency, must result in the quicker resuscitation of the patient, thereby enhancing the chances of life.

Holler (*Die Chemie*, 1944, 57, 14; *Dtsch. Gesell. inn. Med.*, Oct., 1943) classifies the causes of liver damage on a clinical basis as follows: (1) Endocrine disturbances: hyperthyroidism, diabetes mellitus, pregnancy. (2) Chemical poisons: phosphorus, arsenic, hypnotics, etc. (3) Food toxins as in "meat poisoning." (4) Spirochaetal diseases: Weil's disease, marsh fever, secondary syphilis. (5) Drugs: atophan, salvarsan, evipan, *Filix mas*. (6) Infections: jaundice in pneumonia, sepsis, rheumatism, diphtheria, scarlet fever, epidemic meningitis, etc. (7) Infections with monocytic or monolymphocytic reaction (Pfeiffer's glandular fever, epidemic hepatitis). Holler regards as specially characteristic of epidemic hepatitis the appearance of numerous monocytes in the blood; this he interprets as "a mobilization" of the reticular endothelial system.