The death rates from suffocation "in bed" show a tendency to decline recently. Again this might be due to more bottle-feeding, with the result that fewer mothers go to sleep and thereby suffocate their babies while breast-feeding them in bed. But there seems to have been during the decade a slight rise in the death rates from suffocation "in cot, cradle, etc.," and the two may be related. Higher wages have enabled more parents than formerly to buy cots if they wish to do so, and the babies may be placed in them in preference to beds containing other occupants.

Conclusion

The importance of precisely ascertaining the cause before attributing the death of a baby to accidental mechanical suffocation lies in the effect that such a diagnosis may have on the parents. Few would not bitterly reproach themselves for having contributed, however unwittingly, to the death of their baby. Yet many whose baby's death is so recorded, even though ignorant of the dangers of taking a baby into their bed, or laying it face down on a soft pillow, are in fact innocent of allowing their baby to die of suffocation. Especially is this true when babies are found dead in cots and cradles, as the sex ratios show.

Summary

The sex ratios of infant deaths from accidental mechanical suffocation in various circumstances suggest that the causes of death in cots and cradles differ considerably from the causes of death in bed and by food. Published evidence is cited indicating that in any case only a minority of these deaths are due to suffocation.

Recently, death rates for suffocation "in bed" have declined, while those for suffocation "in cot, cradle, etc.," and "by food" have risen.

No infant's death should be attributed to accidental mechanical suffocation unless there is clear positive evidence of it.

REFERENCES

Bowden, K. (1950). *Med. J. Aust.*, **1**, 65.
Brend, W. A. (1915). *An Inquiry into the Statistics of Deaths from Violence and Unnatural Causes in the United Kingdom*. Griffin, London.
Davison, W. H. (1945). *British Medical Journal*, **2**, 251.
Moncrieff, A. (1950). Ibid., **2**, 795.
Registrar-General (1951). *Statistical Review of England and Wales for the Two Years 1946–7*, Text, Vol. 1, Medical. London.
Werne, J. (1942). *Amer. J. Path.*, **18**, 759.
—— and Garrow, I. (1947). *Amer. J. publ. Hlth*, **37**, 675.

The October issue of *Industrial Health Monthly*, a publication of the Federal Security Agency (Public Health Service) from Washington, D.C., contains an article "Who's an Amputee?" which discusses the practical problems of artificial-limb fitting as seen in the U.S.A. Although there are about 900,000 civilians who have had an amputation, and about 40,000 new amputations are performed every year, only half of them are able to use artificial limbs. After the second world war the Veterans' Administration investigated the reasons for failure to use artificial limbs, and found two chief reasons. One was educational: the patient was not taught how to use his limb in the most comfortable and efficient way. The other was that after operation the stump became fixed. After amputation the patient was returned to bed and a soft pillow placed under the thigh stump so that it stuck up in the air, and in 10 days it was already stiff in this position. Then the patient went home to wait in a wheel-chair till his new leg was ready. When it arrived, with one hip fixed he had to tilt his spine and pelvis if he was to walk with it, and this quickly produced pains and complaints about the artificial leg.

EFFECT OF CIGARETTE-SMOKING ON BLOOD FLOW THROUGH THE HAND

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It is generally held that tobacco-smoking causes a decrease in the skin blood flow. Lampson stated in 1935, for example, that following the inhalation of the smoke from one cigarette the rate of peripheral blood flow was at least halved and remained depressed for about 60 minutes. If the smoke was not inhaled the vasoconstrictor response was almost as great but the decrease lasted only 15 minutes. He attributed these results to the pharmacological action of the tobacco. Roth, McDonald, and Sheard (1944) reviewed previous work, and in a further investigation on six normal subjects showed that after smoking two "standard" cigarettes the cutaneous temperature of the extremities of all the subjects decreased. They concluded that "the smoking of standard cigarets should be avoided in the presence of peripheral vascular disease."

A reflex vasoconstriction in the digits, however, follows deep inspiration (Bolton, Carmichael, and Stürup, 1936; Gilliatt, 1948), and Mulinos and Shulman (1940) were the first to point out that the primary factor in the causation of the decreased blood flow might be the deep breathing associated with the inhalation of the tobacco smoke, rather than the action of any substance contained in the tobacco.

It seemed to be worth while investigating whether the rate of smoking had any effect on the blood flow through the hand, and, if so, whether the flow was affected by what might be regarded as a normal rate of smoking. Evidence will be presented which suggests that when cigarette smoke is inhaled at a rapid rate there is a marked and continuous decrease in the blood flow. However, when smoking is carried out at approximately the normal rate there is only a transient decrease in hand blood flow at the moment of inhalation. In the former instance the decrease is mainly pharmacological, due to the action of the tobacco, but in the latter it is mainly physiological, due to the associated deep breath on inhaling.

Methods

The depth of respiration was recorded by means of two stethographs, one at the level of the fourth intercostal space and one at the level of the umbilicus, connected to a common volume recorder. Preliminary calibration of the system against collection and measurement of expired air showed a nearly linear relationship between the amplitude of the excursions and the tidal air.

Hand blood flows were recorded by means of a venous occlusion plethysmograph (Barcroft and Edholm, 1945) filled with water at 32° C., the principle being that described by Brodie and Russell (1905) and Hewlett and Van Zwaluwenburg (1909).

The subjects were young healthy males with normal cardiovascular systems. They smoked on the average 10–20 cigarettes a day. All were accustomed to hand plethysmography, and were told the details of the

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experimental procedure some time before observations were begun. This was to reduce to a minimum any psychological stimuli which can lead to marked variations in hand blood flow. They rested in the laboratory, the temperature of which was maintained at about 24–25° C., for half to one hour before the experiments began. During experiments the subject sat in a chair with the right hand in a plethysmograph. The observations were made in the afternoon or evening, and in all cases the subjects had smoked cigarettes at their accustomed intervals throughout the day.

A standard brand of British cigarette containing a mixture of Virginian and Empire tobacco was used throughout. In some of the experiments the subject inhaled at intervals of a minute. In other experiments the interval between inhalations was 20 seconds. In each experiment the actual smoking was preceded by a period of imitation smoking, in which the same technique was adopted except that the cigarette was unlit. The hand blood flow was recorded every 15 seconds.

The routine procedure was: 10 minutes control period, 10 minutes imitation smoking, 10 minutes control, 10 minutes smoking, 10 minutes control.

Results

When each subject was asked how often he normally inhaled during smoking the usual reply was, "Every 15 seconds," and this rate was therefore adopted in the preliminary series of experiments. However, generalized reactions, notably dizziness, nausea, and a feeling of faintness, commonly followed inhalation at this rate. Two subjects would probably have lost consciousness if the experiment had not been terminated. A series of observations was therefore made to determine the normal rate of inhalation. Fifty males were surreptitiously observed inhaling tobacco smoke in buses, restaurants, and public and private houses. The average rate of inhalation was determined for each subject, and the results are shown in Fig. 1. Sixteen of these were observed from start to finish of the cigarette, 24 for at least seven consecutive inhalations, and 10 for six consecutive inhalations. The average intervals between inhalations in the three groups were 67, 66, and 68 seconds respectively. The average of all observations was 66 seconds. The shortest individual average interval was 43 seconds, and yet when some of these people were asked what they thought was their normal rate of inhalation the usual reply was, "About once every 15 seconds." On the basis of these findings the inhalation rate adopted in the present experiments was once every minute, which was a convenient approximation to the mean of 66 seconds for the 50 subjects.

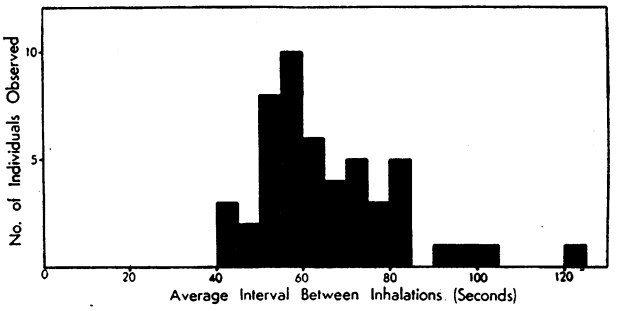

FIG. 1.—The average rate of inhalation of cigarette smoke in 50 male subjects (for details see text).

The average of the hand blood flows obtained in 12 subjects who inhaled at this rate is shown in the top half of Fig. 2. It will be seen that during the period of imitation smoking a sharp drop in hand blood flow occurred at intervals of a minute—that is, during the imitation inhalation—and that the flow 15 seconds later

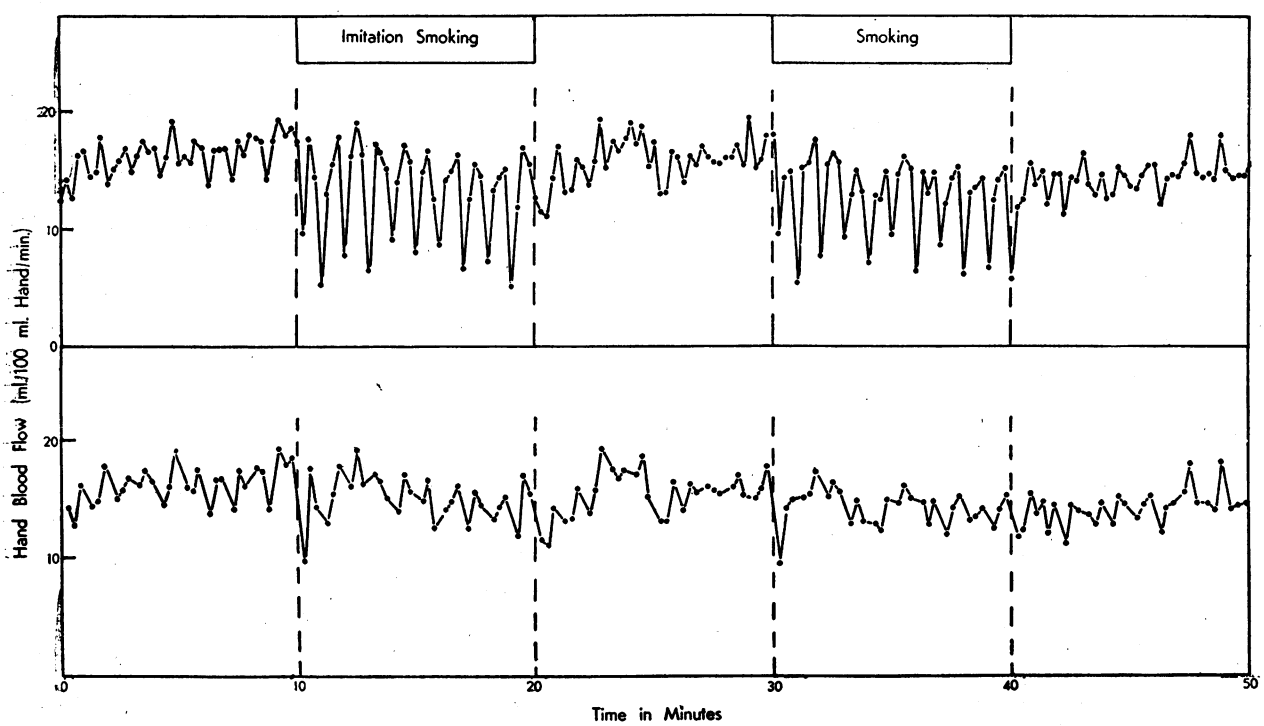

FIG. 2.—Upper half: A comparison of the effects of smoking and imitation smoking on the hand blood flow. Composite graph of the results obtained on 12 subjects. Inhalations every minute. At the time of inhalation there is a transient decrease in flow, both during smoking and imitation smoking. Lower half: The flows recorded at the moment of inhalation of the tobacco smoke and the corresponding flows during the imitation period have been removed to illustrate that the general level of hand blood flow remains substantially unchanged with either smoking or imitation smoking at this rate.

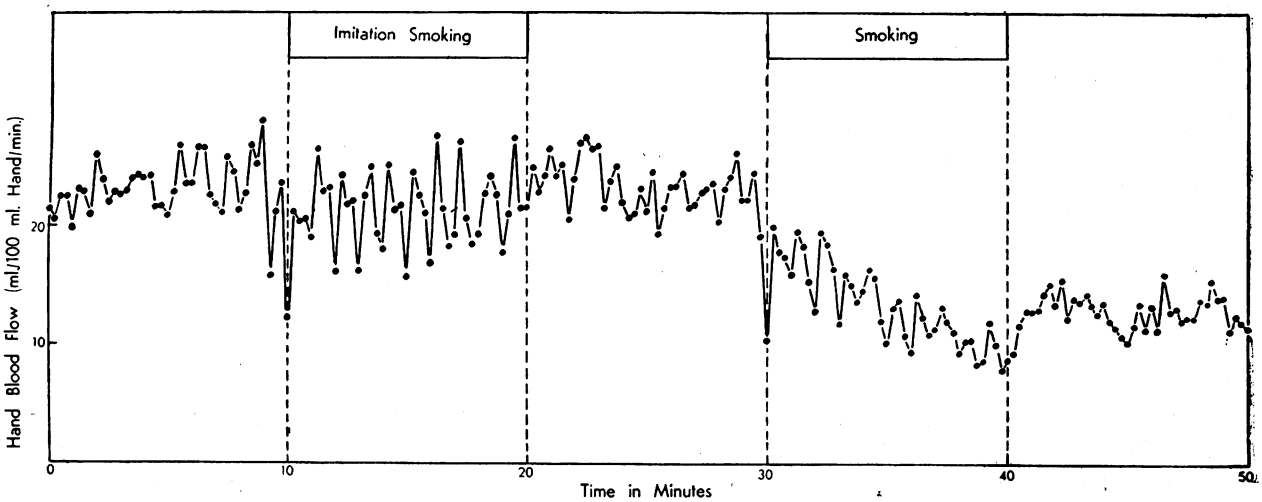

FIG. 3.—A comparison of the effects of smoking and imitation smoking on the hand blood flow. Composite graph of the results obtained on six subjects. Inhalations every 20 seconds. Note the gradual well-marked decrease in flow during the smoking period as compared with the imitation smoking period.

had returned to about the previous level. Very similar changes occurred during the smoking period, the blood flows decreasing to the same extent during inhalation, with an equally rapid recovery.

The stethographic records showed no significant difference between the depths of inhalation during these two periods. A comparison of the individual blood-flow records showed similar results, but the random fluctuations in hand blood flow were greater at all times.

In the bottom half of Fig. 2 the same results are shown, with the exception that the reading showing the sharp drop in flow during each real or imitation inhalation has been omitted. It can be seen that the general blood-flow level has not substantially altered throughout the whole 50-minute period of observation. The sharp drop in flow at the beginning of the smoking and imitation smoking period was due to placing a cigarette between the subjects' fingers, and is a good example of how transient vasoconstriction of the hand blood vessels may result from very minor stimuli.

The general hand blood-flow level varied from subject to subject, so that all degrees of vasodilatation are represented in the composite results.

Blood-pressure recordings were not made during these experiments, as it has been shown by Roddie (1951) that the rapid inflation of a blood-pressure cuff on one arm causes a transient decrease in hand blood flow on the opposite side. Further, there is no adaptation to this stimulus even with repeated application in trained subjects. It was felt that the additional vasoconstriction resulting from this stimulus would make the interpretation of the records difficult. A separate series of blood-pressure and pulse-rate recordings was made on six subjects before, during, and after inhaling cigarette smoke once a minute. The blood pressure was recorded by the auscultatory method. In no case was there a significant change in the blood pressure or pulse rate.

In order to determine the effects of smoking in excess of the normal rate the experiments were repeated on 6 of the 12 subjects, the routine being the same except that the imitation inhalations and the inhalations of the tobacco smoke were carried out at 20-second instead of at one-minute intervals. At this rate the subjects smoked about 1¼–1½ cigarettes in the 10 minutes. The composite results are shown in Fig. 3. There was in all cases a marked gradual decrease in hand blood flow during the period of smoking as compared with the period of imitation smoking, in spite of the fact that in this series the average depth of inhalation during the imitation was somewhat greater than during the smoking. Two of the subjects felt faint towards the

Table Showing the Effect of Inhaling Cigarette Smoke at 20-second Intervals on Hand Blood Flow, Blood Pressure, and Pulse Rate

Subject	Decrease in Hand Blood Flow	Average Blood Pressure (mm. Hg)						Average Pulse Rate (Beats per min.)		
		Basal		During Smoking		Increase				
		Sys.	Dias.	Sys.	Dias.	Sys.	Dias.	Before Smoking	During Smoking	Inc.
1	66%	126	78	128	81	2	3	65	76	11
2	60%	116	71	116	76	0	5	70	76	6
3	56%	133	80	147	100	14	20	64	64	0
4	29%	114	69	117	74	3	5	83	96	13
5	51%	120	78	122	80	2	2	76	86	10
6	62%	115	75	118	75	3	0	78	88	10
Average	54%	121	75	125	81	4	6	73	81	8

The percentage decrease in hand blood flow has been calculated from the average flow in the five-minute period preceding smoking and the average flow during the last five minutes of smoking.

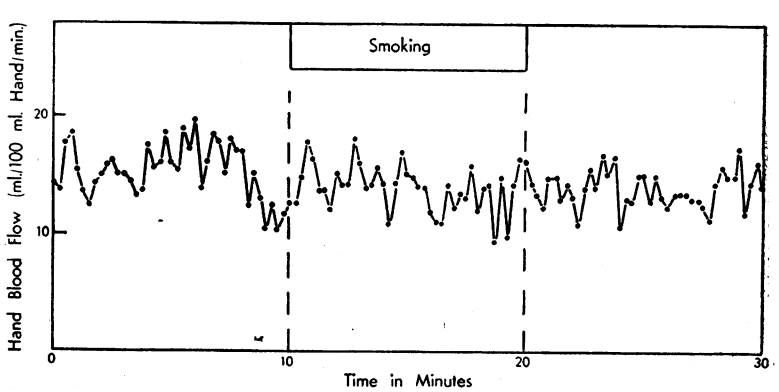

FIG. 4.—Composite result of six experiments which illustrate that the general level of hand blood flow remains unaltered as a result of cigarette-smoking without inhalation of the tobacco smoke.

end of the smoking period. Blood-pressure and pulse-rate recordings made on separate occasions on each of the six subjects during smoking at 20-second intervals showed variable results (see Table). The pulse rate generally increased by 10–12 beats a minute and there was a slight increase in blood pressure. The same two subjects as before felt faint and one of them showed a rise of 14 mm. Hg and 20 mm. Hg in systolic and diastolic pressure respectively, with no alteration in pulse rate. Repeat tests on five of these subjects smoking a common brand of American cigarette gave similar results.

Hand blood-flow measurements were also made during which the subjects kept the cigarette continually in the mouth for 10 minutes, puffing at frequent intervals without inhaling. Six of the original 12 subjects were used, and composite results are shown in Fig. 4. It is clear that no decrease in blood flow occurred with this type of smoking.

Discussion

The present results at first might appear to be at variance with many of those already reported, but the reason for the discrepancy seems to be the difference in the frequency of inhalation. In order to determine the effect of normal cigarette-smoking on the circulation, it is essential that the subject should inhale at his normal rate, and it appears from the literature that in the past insufficient attention has been given to this point. As the average person is incapable of estimating his normal rate, this must be decided by surreptitious observation. As a result of such observations an inhalation rate of once a minute was chosen for the present experiments. The inhalation of tobacco smoke at this rate leads to a sudden transient diminution in hand blood flow at the moment of inhalation, the flow returning to the previous level when the normal depth of breathing is resumed. Similar changes occur when inhalations of the same extent are carried out with an unlit cigarette, and thus the transient diminution appears to be due to the physiological effects of the deep breath rather than to the pharmacological action of the substances in the tobacco smoke. In addition, smoking at this rate causes little or no alteration in blood pressure or heart rate.

Inhalations every 20 seconds, however, lead to a well-marked decrease in flow during smoking. This cannot be explained by the greater number of deep inhalations, as there was no such decrease during the period of imitation smoking. It is therefore due to the action of substances in the tobacco smoke.

It is possible, therefore, that the discrepancy between our results and those of other workers may be explained by a difference in the rate of smoking adopted. Maddock and Coller (1933) stated that their subjects were given their customary form of tobacco to smoke at their normal rates, but occasionally a subject noted mild vertigo and nausea on smoking. Wright and Moffat's (1934) subjects, who were experienced smokers, also were urged to smoke at their normal rate, though they state that it is probable that there was an increase in both the rate and the depth of inhalation during the experiment. In some of their subjects severe symptoms developed during smoking, including "nausea with vomiting, marked vertigo, cold sweats, pallor, and even profound syncope."

None of our subjects experienced any unpleasant symptoms while inhaling at minute intervals, and, although the rate of smoking is not given by the workers mentioned, it seems likely from the symptoms described that it was more rapid than normal.

Roth, McDonald, and Sheard (1944), who found a very marked decrease in the skin temperature of the extremities with smoking, also state that their subjects inhaled with their accustomed depth and frequency, though they also do not say what this was. They state, however, that their subjects fasted for 15 hours and then smoked two cigarettes in succession until two-thirds of each had been smoked. The smoking period generally lasted 12–16 minutes. They found larger increases in pulse rate and blood pressure during smoking than we obtained on our subjects when inhaling with British or American cigarettes at 20-second intervals. When two of our subjects inhaled at this rate, however, following a 15-hour period without food and cigarettes, increases in pulse rate and blood pressure were observed similar to those described by Roth *et al.*

Summary

The frequency of inhalation of tobacco smoke has been determined in 50 male subjects. The average time between inhalations was 66 seconds.

Simultaneous measurements of hand blood flow and stethographic records of the respiratory movements have been made in moderate cigarette-smokers during smoking and imitation smoking.

Inhalation of the tobacco smoke at intervals of a minute (approximately the normal rate) leads to a transient decrease in flow at the moment of inhalation. A similar decrease occurs when inhalations of the same depth are carried out with the cigarette unlit.

The transient decrease in flow is therefore due to the physiological effect of the deep breath associated with the inhalation, and not to the pharmacological action of substances in the tobacco.

Apart from this decrease in flow on inhalation, the general level of hand blood flow is unaltered during and immediately after smoking one cigarette at this rate.

When cigarette smoke is inhaled every 20 seconds—that is, more rapidly than normal—the hand blood flow steadily decreases during the smoking period. There is no decrease in the general blood-flow level when inhalations of similar depth are made with the cigarette unlit.

When cigarettes are smoked at a rate in excess of the normal the resulting decrease in hand blood flow is therefore mainly pharmacological and not physiological in origin.

The significance of previous work on smoking where the frequency of inhalation has not been controlled or is not stated is therefore difficult to assess.

My thanks are due to Dr. C. de Largy for his collaboration in the earlier experiments. I would also thank my colleagues and students who acted as subjects.

References

Barcroft, H., and Edholm, O. G. (1945). *J. Physiol.*, **104**, 161.
Bolton, B., Carmichael, E. A., and Stürup, G. (1936). Ibid., **86**, 83.
Brodie, T. G., and Russell, A. E. (1905). Ibid., **32**, XLVIIP.
Gilliatt, R. W. (1948). Ibid., **107**, 76.
Hewlett, A. W., and Van Zwaluwenburg, J. G. (1909). *Heart*, **1**, 87.
Lampson, R. S. (1935). *J. Amer. med. Ass.*, **104**, 1963.
Maddock, W. G., and Coller, F. A. (1933). *Ann. Surg.*, **98**, 70.
Mulinos, M. G., and Shulman, I. (1940). *Amer. J. med. Sci.*, **199**, 708.
Roddie, I. C. (1951). *J. Physiol.*, **112**, 204.
Roth, G. M., McDonald, J. B., and Sheard, C. (1944). *J. Amer. med. Ass.*, **125**, 761.
Wright, I. S., and Moffat, D. (1934). Ibid., **103**, 318.