have not taken into account the beta and other agglutinable types of *Bact. coli*. Even so, we are perplexed by the discrepancy between our findings and those of others, and record the figures for what they are worth.

The incidence of this particular strain of *Bact. coli* in our cases is much lower than we had expected, and we thought that our 30 individual colonies slide-agglutinated would have given us a higher percentage of positive results than the three or four colonies reported as having been examined by some other workers. A false positive slide agglutination is more likely than a false negative, and were our findings to be explained on technical grounds we would have expected an even higher incidence than was obtained. Similarly, from a study of the published work we expected our control series of cases—as comparable as we could get them, except that there was no gastro-intestinal disorder—to have a much lower incidence than was found.

Taylor (1951) and others have emphasized that their greatest isolation of *Bact. coli* O group 111 was obtained in epidemics, whereas our cases, with the exception of the few arising *de novo* in another ward of the hospital and subsequently transferred to the gastro-enteritis unit, cannot be regarded as epidemic cases. The L.C.C. cases are probably mostly sporadic; and here again, to put it no stronger, our figures show that there is no correlation between the total number of cases notified and the percentage incidence of *Bact. coli* O group 111 in our hospital cases.

We are unable to offer any explanation why our findings are so different from those of others, unless such is to be found in some difference between the epidemic and sporadic disease, or an enhancement of virulence of a causal organism leading to an epidemic. Even so, we are disappointed that an organism impliedly associated with the pathogenesis of epidemic gastro-enteritis has in our experience been so infrequently found in sporadic cases of what appears to be the same disease. We are perturbed, also, by the incidence of the same potential pathogen in the rectal swabs of healthy babies of the same age group, an incidence for all practical purposes identical over a period of six months with that found in our cases of sporadic infantile gastro-enteritis.

There has been a tendency recently to regard these agglutinable strains of *Bact. coli* as the *cause* of gastro-enteritis, and, while we do not suggest that there is no relationship between the organism and the disease, our results do not support the idea that the particular organism we have investigated is causal.

Summary

Figures are presented of the isolation of *Bact. coli* O group 111 (D433) from 264 cases of sporadic infantile gastro-enteritis observed over a period of two years.

In a shorter control series this organism was isolated as often as in the cases of the disease.

No correlation was found between the total number of cases notified to the L.C.C. and the percentage of cases in which the organism was found on admission to the Hospital for Sick Children.

We are grateful to Dr. Joan Taylor for advice, to Sir Allen Daley for the L.C.C. notifications, and to the physicians of the Hospital for Sick Children for access to their cases.

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SO-CALLED ACCIDENTAL MECHANICAL SUFFOCATION OF INFANTS

BY

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During 1940–9 in England and Wales the average annual number of deaths ascribed to the accidental mechanical suffocation of infants aged under 1 year was 620. There has for some time been considerable doubt whether the deaths so registered are in fact due to the cause alleged. Brend (1915) believed that many supposedly suffocated infants died from natural causes, and gave some statistics that lent support to his suggestion. Davison (1945), coroner for the City of Birmingham, ordered post-mortem examinations to be carried out by skilled pathologists on every case in which asphyxia was considered to be a possible cause of death in an infant; he found that out of 318 cases only 24 died from asphyxia caused mechanically. In the U.S.A., Werne and Garrow (1947) investigated the deaths of 167 infants where accidental mechanical suffocation was alleged to be the cause. In 43 the gross findings were adequate to establish a natural cause of death; of the remaining 124 cases, microscopical studies showed natural causes of death in many. In Australia, Bowden (1950) recorded the post-mortem findings in 40 cases of sudden death in infants, nearly all in bed. Morbid changes were found in all of them.

Though the pathological evidence indicates that some infant deaths ascribed to accidental mechanical suffocation are much more probably due to disease, it does not enable us to estimate what proportion. Suffocation may leave no signs discernible in an infant after death, and it is possible that an infant suddenly smitten with pneumonia, for example, might succumb to asphyxia from pressure of its sleeping mother's breast or from a pillow in its cot, while a healthy infant would have no difficulty in surviving either hazard. But certainly a considerable proportion of the cases described showed lesions sufficient to account for death without any need to invoke mechanical suffocation as a cause when there was no firm evidence of it.

The numbers of deaths in England and Wales recorded by the Registrar-General in the periods 1921–30, 1931–9, and 1940–9, distinguished by sex of infants and circumstances of death, are shown in Table I. Because of changes in the method of classification, the actual numbers of deaths in the first two periods are not strictly comparable with those in the third.

Table I.—*Deaths from Accidental Mechanical Suffocation in Infants in Three Periods, 1921–49, England and Wales*

Years	In Bed		In Cot, Cradle, etc.		By Food		Other		Total	
	M	F	M	F	M	F	M	F	M	F
1921–30	1,810	1,622	171	112	214	146	203	185	2,398	2,065
1931–9	1,031	791	311	192	313	248	45	32	1,700	1,263
1940–9	1,165	892	1,014	638	1,258	907	185	144	3,622	2,581

Sex Ratios

A feature of any cause of death is the distribution of the deaths between males and females—that is, the sex ratio of the deaths. This distribution is as characteristic of a particular cause of death as, for example, the age

distribution of the people who die from that cause. Like the age distribution it varies from year to year or decade to decade in accordance with certain definable attributes of the population exposed to the risk of death from that cause, and it is also subject to chance variation because of the influence of many small and incalculable factors acting in a random manner. If the number of deaths from which the sex ratio is calculated is small, chance variation tends to obscure the operation of the main factors that determine the size of the sex ratio. As the sample becomes larger the effect of certain important factors becomes apparent. The two main factors concerned here are the sex distribution of the population exposed to risk and the differing susceptibility, characteristic of each sex, to death from a particular cause. When comparing the sex ratios of deaths from two or more causes in the same population, we find that the main effective reason for the difference between them is the fact that the deaths are caused by different agents.

Sex ratios are expressed here in the form of male deaths per 100 total deaths. In Table II these ratios are given for deaths in three defined categories of deaths from accidental mechanical suffocation recorded by the Registrar-General.

TABLE II.—*Percentage Male Deaths from Accidental Mechanical Suffocation in Infants in Three Periods, 1921–49, England and Wales*

Years	In Bed	In Cot, Cradle, etc.	By Food	Other	Total
1921–30 ..	52·74	60·42	59·44	52·32	53·73
1931–9 ..	56·59	61·83	55·79	58·44	57·37
1940–9 ..	56·64	61·38	58·11	56·23	58·39

It will be seen that, in the three periods considered, deaths " in cot, cradle, etc." showed a higher percentage of males than did deaths " in bed " or " by food." In the period 1921–30 the difference between the sex ratio of cot deaths and that of bed deaths is 7.68 and is statistically significant (S.E. = 3.09 ; difference/S.E. = 2.5). In 1931–9 the comparable difference is 5.24 (S.E. = 2.49 ; difference/S.E. = 2.1), which is statistically significant, and in 1940–9 it is 4.74 (S.E. = 1.63 ; difference/S.E. = 2.9), also statistically significant. As to the difference between cot and food deaths, in 1921–30 it is 0.98 (S.E. = 3.89 ; difference/S.E. = 0.3) ; in 1931–9 it is 6.04 (S.E. = 3.02 ; difference/S.E. = 2.0) ; and in 1940–9 it is 3.27 (S.E. = 1.60 ; difference/S.E. = 2.0). Thus in two of the periods the differences between cot and food deaths are significant at the 5% level, while in 1921–30 the difference does not attain significance. The probable reason for its not doing so is that comparatively few deaths in cot and by food were recorded in that period. The standard error of the sex ratios is therefore rather high ; for the sex ratio of the food deaths it is 2.59.

The conclusion suggested by these differences is that many of the cot deaths are caused differently from deaths in bed or by food. Yet all are alleged to be due to the same cause—accidental mechanical suffocation.

The Cot Deaths

Since pathological investigations have indicated that many babies supposedly suffocated probably died from natural causes, we should expect to find that the sex ratio of total deaths from accidental mechanical suffocation is considerably higher than that of live births and approaches rather the sex ratio of total infant deaths. The relevant figures are set out in Table III.

TABLE III.—*Sex Ratios (Percentage Males) of Live Births, Total Infant Deaths, and Deaths from Accidental Mechanical Suffocation in Three Periods, 1921–49, England and Wales*

Years	Live Births		Infant Deaths		Deaths from Accidental Mechanical Suffocation	
	Sex Ratio	S.E.	Sex Ratio	S.E.	Sex Ratio	S.E.
1921–30	51·10	0·019	57·61	0·069	53·73	0·75
1931–9	51·27	0·021	57·71	0·087	57·37	0·91
1940–9	51·48	0·019	57·68	0·087	58·39	0·63

In the two later periods our expectations are fulfilled. But for 1921–30 the sex ratio of deaths from suffocation is surprisingly low. In this decade there were proportionately more deaths " in bed " than in the other two periods, and it is the sex ratio of these deaths that chiefly determines the size of the sex ratio for all deaths from accidental mechanical suffocation. Deaths in bed then comprised 76.9% of all deaths from suffocation, while in 1931–9 they comprised 61.5%, and, in 1940–9, 33.2%. Probably many of the deaths genuinely due to mechanical suffocation are to be found among those classified as having occurred in bed, the suffocation being caused by another occupant of the bed. Since the sex ratio of such deaths is likely to be considerably lower than the sex ratio of deaths from infectious diseases, the inclusion of a substantial number of them in the category of death from accidental mechanical suffocation, together with the deaths from infectious diseases that are also undoubtedly included there, will lower the sex ratio of the composite group. It is impossible to say how many of the deaths were in fact due to suffocation, but there may well have been more then than later, for antenatal clinics and other sources of education were then sparser.

But the main problem is why deaths from suffocation in cots should have a higher sex ratio than those in beds or by food. It seemed worth while considering whether the sex ratio of cot deaths was higher than the sex ratios of the others because the population at risk had a different sex ratio. This could happen if the cot deaths were of infants who died soon after birth and the other deaths were of infants later in the first year of life, when deaths from other causes would have reduced the excess of males over females under 1 year of age in the population. To test the extreme case, adjusted figures were obtained in the following way : Cot deaths were first assumed to have occurred in a population whose sex composition was that of the live births in each period : the numbers of them were then reduced to the numbers there would have been if they had occurred at the same rate in a population whose sex composition was that of infants at the end of one year's losses from infant deaths. A new sex ratio was computed from the adjusted figures. The calculations carried out are expressed symbolically as follows :

S = Number of suffocation deaths in cot
B = Number of live births
D = Number of all infant deaths
Subscripts M and F = male and female

$$S'_M = S_M \times \frac{B_M - D_M}{B_M}$$

$$S'_F = S_F \times \frac{B_F - D_F}{B_F}$$

$$\text{Adjusted sex ratio} = \frac{S'_M}{S'_M + S'_F} \times 100$$

By this means the adjusted sex ratios were found to be 59.94 in 1921–30, 61.45 in 1931–9, and 61.10 in 1940–9.

Clearly the reduction effected on this assumption is negligible.

The disparity between cot deaths and the others is probably a true one. A likely explanation of it is that the cot deaths are nearly all due to natural causes—and causes that show a high sex ratio—while the bed and food deaths include relatively many more that are genuinely caused by suffocation. Accidents are as likely to befall male as female infants in the circumstances considered here, though it is quite possible that there is some sex distinction in the ability to survive them. But this distinction would not be notably higher in the cot than in the bed.

Diseases Causing Sudden Death

The sex ratios of deaths from certain conditions apt to attack infants with great rapidity, and mentioned as having been found at necropsy in the pathological investigations, were calculated for the periods in question here, and are listed in Table IV.

TABLE IV.—*Sex Ratios of Infant Deaths from Certain Causes in Three Periods, 1921–49, England and Wales*

Cause of Death	Sex Ratio		
	1921–30	1931–9	1940–9
Acute nephritis	59·85	61·09	64·33
Diseases of ear and mastoid antrum	58·86	60·18	60·29
Enteritis and diarrhoea	59·33	59·70	59·47
Congenital malformation of heart	57·89	60·18	58·27
Influenza	59·10	58·72	58·29
Bronchopneumonia	58·03	58·14	57·28
Lobar pneumonia	58·92	58·49	56·37
Bronchitis	57·65	57·29	58·42
Meningitis (non-meningococcal)	58·11	58·19	57·45

As can be seen by comparison with Table III, nearly all these sex ratios are above the sex ratios of total infant deaths in the three periods. The sex ratios of deaths from the first three causes listed approximate closely to the sex ratios of cot deaths from accidental mechanical suffocation. To conclude that acute nephritis, diseases of the ear and mastoid, and diarrhoea and enteritis are the chief causes of death in cases of so-called suffocation in cots would be rash, but certainly diseases of the ear and mastoid are often found in cases of suspected or supposed suffocation. Werne and Garrow (1947), in their 167 cases already referred to, considered that the gross findings at necropsy were adequate to establish a natural cause of death in 43; of these, 20 had mastoiditis and otitis media, and another had bronchopneumonia and otitis media. It is not clear how many of the other cases showed microscopical evidence of natural causes of death, but "complete microscopical studies as a rule showed acute inflammation of the respiratory tracts" in association with other lesions. In view of the fact that deaths from acute nephritis show a notably high sex ratio it is of interest to note that Werne (1942), discussing the pathology of 50 infants who died unexpectedly, stated that many of them showed glomerular lesions.

Bowden (1950) found in his 40 cases 7 cases of otitis media, all associated with pneumonia or bronchitis, 2 cases of enteritis, and 2 cases of subacute nephritis. Some kind of respiratory infection was commonly found.

Out of the 318 cases discussed by Davison (1945), 77 had otitis media with bronchopneumonia or respiratory disease and 8 otitis media with mastoiditis. There were also 2 cases of enteritis, 1 of congenital heart disease, and 1 of meningitis. The largest group of his cases was 152 with bronchopneumonia, capillary bronchitis, and bronchiolitis, without complications. In 24 cases death was attributed to asphyxia by mechanical means, and in 11 to asphyxia by inspiration of vomit.

It yet remains rather surprising that the sex ratio of the cot deaths from suffocation should lie consistently between 60 and 62. It would hardly be likely to do so if the great majority of these deaths were due to uncomplicated respiratory infections, though still less likely is it that many of them were due to mechanical suffocation. Probably otitis media and mastoiditis comprise the commonest causes of death here, and Werne's findings suggest that acute nephritis is present more often than it is diagnosed.

Changes in Death Rates

The Registrar-General (1951) has pointed out that the annual death rates from accidental mechanical suffocation in infants tended to increase from 1931 to 1947 (the last year that he was reviewing). This increase, in so far as it includes rates for the years 1931–9, must be accepted with caution, for from 1940 onwards the method of classification has changed, deaths from suffocation on which coroners' "open verdicts" were given being assigned to this category instead of to a separate one. Whether there was a real increase in the death rates during the decade 1940–9 as compared with the period 1931–9 is therefore open to slight doubt, but the last column of Table V shows that the death rate for 1940–9 (0.87 per thousand live births) was apparently much higher than that for the previous nine years (0.54).

TABLE V.—*Deaths and Death Rates from Accidental Mechanical Suffocation in Infants in Certain Periods, 1921–49, England and Wales*

	No. of Deaths					Rate per Thousand Live Births				
	Bed	Cot	Food	Other	Total	Bed	Cot	Food	Other	Total
1921–30	3,432	283	360	388	4,463	0·48	0·04	0·05	0·05	0·63
1931–9	1,822	503	561	77	2,963	0·33	0·09	0·10	0·01	0·54
1940..	192	99	137	18	446	0·32	0·16	0·23	0·03	0·73
1941..	273	100	131	32	536	0·47	0·17	0·23	0·06	0·93
1942..	218	136	141	10	505	0·33	0·21	0·22	0·02	0·78
1943..	166	168	120	33	487	0·24	0·25	0·18	0·05	0·71
1944..	240	170	180	51	641	0·32	0·23	0·24	0·07	0·85
1945..	219	155	183	43	600	0·32	0·23	0·27	0·06	0·88
1946..	212	198	270	45	725	0·26	0·24	0·33	0·05	0·88
1947..	242	231	385	53	911	0·27	0·26	0·44	0·06	1·03
1948..	170	189	312	25	696	0·22	0·24	0·40	0·03	0·90
1949..	125	206	306	19	656	0·17	0·28	0·42	0·03	0·90
1940–9	2,057	1,652	2,165	329	6,203	0·29	0·23	0·30	0·05	0·87

During 1940–9 the rates for the years individually rise very slightly, though irregularly, and it is too soon to attach much importance to such trend as is seen. But there does seem to be a distinct post-war rise in the death rate from suffocation "by food" after a small decline during the war. Whether there has been a post-war decrease in breast-feeding is problematical, but Moncrieff (1950) has drawn attention to a decline in the practice in recent years. If breast-feeding has declined in the last few years—and it is a matter only for speculation—the cause of the rising death rates might be sought there, for babies have been suffocated by being left on their own to suck from a bottle; they rapidly ingest the milk, regurgitate some of it, and inhale the vomit. It is a dangerous practice—possibly common—and worth while preventing so far as is possible by instruction to mothers. Coroners may be able to elucidate the true cause of these deaths and by publicity help to prevent them.

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.

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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

BY

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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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