Outside Greater London, where fog was also widespread, the corresponding increase in deaths in the Great Towns of England and Wales was from 8,443 to 9,141, an increase of only 8%.

Morbidity

Although this paper is concerned with the effects of the London fog on mortality, two tables have been added to show certain concomitant morbidity trends in other statistics routinely published in the Registrar-General's Weekly Returns. Weekly notifications of pneumonia are shown in Table XII and National Insurance claims for sickness benefit in Table XIII. During weeks ended January 10 and 17

TABLE XI.—Deaths Registered in Five Groups (see Table X) of Great Towns in the Outer Ring of Greater London. Weeks Ended December 24 and 31, 1955, and January 7 and 14, 1956

	Weeks Ended Dec. 24 and 31	Weeks Ended Jan. 7 and 14	% Increase
West	492	647	32
South	418	517	24
South-East	183	185	1
North-East	394	533	35
North	346	432	25

TABLE XII.—Notifications of Pneumonia in London Administrative County and in Remainder of England and Wales from Week Ended December 24, 1955, to January 28, 1956

Week ended:	Dec. 24	Dec. 31	Jan. 7	Jan. 14	Jan. 21	Jan. 28
London	69	60	81	83	77	60
Remainder of Eng- land and Wales	465	592	769	814	719	776

TABLE XIII.—Ministry of Pensions and National Insurance New Claims for Benefit on Account of Incapacity in Successive Two-weekly Periods

Weeks Ended	London Ad. C. and Middlesex	Remainder of London and South-Eastern Region	Remainder of England and Wales
Dec. 13 and 20	32,800	22,900	185,600
.. 27 and Jan. 3	34,600	23,500	213,800
Jan. 10 and 17	59,400	39,800	302,300
.. 24 and 31	50,100	34,500	281,100
Increase between Dec. 27 and Jan. 3 and Jan. 10 and 17	72%	69%	41%

claims in London and Middlesex were 24,800 more than in the previous two weeks (which included Christmas), a relative increase of 72%. In the rest of the south-eastern region the corresponding increase was 69%, but in the remainder of England and Wales it was 41%.

Discussion

Three recently reported fog incidents have led to an increase in mortality in the London area: in November, 1948 (Logan, 1949); in December, 1952 (Logan, 1953; Ministry of Health, 1954; Committee on Air Pollution, 1953, 1954); and now January, 1956. After the fog of December, 1952, a scrutiny was made, in the General Register Office, of London mortality records during the previous 115 years to see whether there had been any previous major increase in mortality associated with periods of severe fog. Five previous incidents were discovered—in December, 1873, January, 1880, February, 1882, December, 1891, and December, 1892. In the face of the apparent infrequency of such incidents throughout much of the nineteenth and the first half of the twentieth century, the fact that three incidents have occurred during the past eight winters is disquieting. It suggests the possibility either that the atmospheric pollution associated with London fogs has recently become more toxic, particularly to the very young, the infirm, and the elderly, or that there has been an increase in the number of

persons who are specially vulnerable to its effects. The 4,000 deaths in December, 1952, made a deep impression on the public imagination. These further 1,000 deaths last January are a stern reminder that this major public health problem has not yet been solved.

Summary

Dense fog during January 4 to 6, 1956, caused almost 1,000 additional deaths in Greater London.

The distribution of these deaths by age, cause, and geographical area is described.

This is the third major fog mortality incident in London since 1948.

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EFFECT OF DURATION OF ANAESTHESIA ON APNOEA NEONATORUM AFTER CAESAREAN SECTION

BY

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In cases of caesarean section, many authorities (Clifford, 1940; Gillies, 1950; Lull and Hingson, 1948; Ten Teachers, 1955; *Queen Charlotte's Textbook of Obstetrics*, 1952; Wylie, 1953) have emphasized the importance of delivering the child as soon after the induction of anaesthesia as possible, thereby minimizing the amount of anaesthetic agents passing to the foetus and there interfering with the normal onset of respiration. We had gained the impression in clinical practice that the often unseemly haste was not justified. Therefore infants delivered by caesarean section were investigated with special emphasis upon the relationship between the duration of the anaesthesia to which they might have been exposed and the delay in the onset of respiration.

Method

One hundred and two infants, the product of 100 consecutive caesarean sections performed on mothers anaesthetized by one or other of us over a period of six months, were studied. Indications for the sections were varied, and are listed in Tables I and II.

TABLE I.—Indications for Caesarean Sections

Indication	No. of Cases	Code for Table II
Incoordinate uterine action ..	11	A
Pre-eclamptic toxæmia ..	11	B
Disproportion or obstructed labour ..	35	C
Placenta prævia ..	8	D
Elective ..	18	E
Eclampsia ..	2	F
Oblique lie ..	1	G
Diabetic elective ..	3	H
Foetal distress ..	6	I
Prolapsed cord ..	5	J
Strong labour in previous rupture of uterus ..	1	K
Carcinoma of cervix ..	1	L

TABLE II.—Data of Investigation

Baby No.	Anaesthetic	Exposure to Anaesthetic (Min.)	Delay in Respiration (Min.)	Indication for Section (See Table I)	Baby No.	Anaesthetic	Exposure to Anaesthetic (Min.)	Delay in Respiration (Min.)	Indication for Section (See Table I)
1	GOE	23	4	C	52	GOEF	17	0	C
2	GOE	15	5	C	53	GOEF	18	0	C
3	GOE	10	3½	C	54	GOEF	14	4	C
4	GOE	10	1	C	55	GOE	13½	0	C
5	PGOE	18	0	E	56	GOE	12½	0	C
6	GOE	18	6	F	57	GOE	15½	0	C
7	GOE	15	9	C	58	GOEF	22	0	A
8	GOE	11	3	B	59	GOE	15	15	E
9	PGOEF	14	0	C	60	GOE	19	14	C
10	GOEF	15	0	E	61	GOE	13	14	C
11	PGOEF	10	15	A	62	GOE	14½	4	C
12	PGOEF	16	4	C	63	GOEF	12	SBc	C
13	PGOEF	20	5	A	64	GOE	15	0	A
14	PGOEF	19	7½	A	65	GOEF	14	14	A
15	PGOEF	14	7	D	66	GOE	21	7	A
16	GOE	11½	2½	G	67	GOE	20	0	H
17	GOE	16	70	C	68	GOE	14	0	E
18	GOE	13	0	B	69	GOE	19	0	A
19	GOE	9	0	H	70	GOEF	18	0	C
20	GOE	16	2	A	71	GOEF	8	1	C
21	PGOEF	10	12	A	72	GOEF	17	0	E
22	PGOEF	6	12	B	73	GOE	10	7½	A
23	CyGOE	16	13	I	74	GOE	11	0	B
24	PGOEF	15	SBa	L	75	GOE	9	0	C
25	GOE	10	1	L	76	GOEF	22	5	E
26	GOE	12½	2½	I	77	GOE	12½	0	A
27	GOE	20	4	C	78	PGOEF	16	14	B
28	GOE	14½	7	C	79	GOEF	19	7	E
29	PGOEF	5	7	H	80	PGOEF	18	8	B
30	PGOEF	4	90	C	81	GOE	10	½	C
31	GOE	15	0	C	82	GOE	18	0	E
32	GOE	11½	4	I	83	GOE	13	17	B
33	GOEF	19	0	D	84	GOEF	18	0	B
34	GOE	11½	7½	D	85	PGOEF	17	12	B
35	GOE	17	5	D	86	GOE	12½	9½	E
36	GOEF	25	6	C	87	PGOEF	6	0	E
37	GOE	14	0	E	88	GOE	12	5	E
38	GOE	13	3	C	89	GOE	12	5	B
39	GOEF	13	0	J	90	GOE	15½	2	I
40	GOEF	15	6	C	91	GOE	15½	4	E
41	PGOEF	13	13	C	92	GOE	16	0	E
42	GOE	18	12	C	93	GOE	19	SBd	J
43	GOE	9	2	C	94	GOE	16	4	E
44	GOE	28	3	C	95	GOE	18	1	J
45	GOE	19	8	C	96	GOE	8	½	B
46	GOEF	12	SBb	I	97	ChGOE	33½	5½	K
47	GOE	6	0	J	98	GOE	11	0	B
48	GOE	7	0	J	99	GOE	13½	0	C
49	GOE	18½	5½	I	100	GOE	23½	4	C
50	GOE	19	20	E	101	GOE	14	0	B
51	GOE	10	6	D	102	GOE	15	0	E

Ch=chloroform. Cy=cyclopropane. E=ether. F=gallamine triethiodide. G=nitrous oxide. O=oxygen. P=thiopentone. SB=stillborn: (a) case of placenta praevia; (b) case of foetal distress; (c) case of "failed forceps"; (d) case of prolapsed cord.

TABLE III.—Anaesthesia

Anaesthetic Agents	Induction	Gallamine Triethiodide	Maintenance
N ₂ O, O ₂ , ether	81	16	N ₂ O, O ₂ , and ether in all cases
Chloroform	1	—	
Cyclopropane	1	—	
Thiopentone	17	16	

Premedication in all the cases was confined to atropine, 1/100 gr. (0.65 mg.). However, several of the mothers had had sedatives within the accepted dangerous time limit—for example, morphine within four hours of delivery. In all cases the baby was alive at the time of induction of anaesthesia.

Anaesthesia (Tables II and III).—Anaesthesia was induced in 81 cases with nitrous oxide, oxygen, and ether; in 1 with cyclopropane; in 1 with chloroform; and in 17 with thiopentone sodium. Sixteen of the 17 were also given gallamine triethiodide, and so too were 16 of those who had nitrous oxide, oxygen, and ether. In all cases maintenance was carried out with nitrous oxide, oxygen, and ether.

Timing.—Two periods were measured (Table II): (1) from start of induction

of anaesthesia to delivery of the baby from the uterus, and (2) from delivery of the baby from the uterus to establishment of continuing respirations. This end-point—the establishment of continuing respirations—was chosen instead of the more usual "crying-time" or "breathing-time," because many of the infants would take one or two gasps or even cry once or twice and then cease breathing for a varying period before either dying (Baby 93) or beginning to breathe again.

All infants were subjected to some degree of resuscitation. The measures adopted varied from simple wiping of the mouth to the administration of intragastric oxygen. We would commend the method of intragastric oxygenation (Åkerrén and Fürstenberg, 1950), which we feel is what permitted Babies 17 and 30 to survive.

Results

The results are listed in Table II and presented graphically in Figs. 1 and 2.

Two sets of twins were born: Babies 47 and 48, and Babies 52 and 53. Four babies are listed as never having breathed—Babies 24, 46, 63, and 93—though, in fact, Baby 93 gasped several times but never breathed continuously before dying.

The results, *excluding* the four babies that died without breathing, were subjected to statistical analysis. When anaesthetic time was related to the delay in onset of continuing respirations, a correlation coefficient (*r*) of 0.12 was

FIG. 1.—Scatterplot of time of exposure to anaesthetic related to delay in onset of "continuing" respirations.

FIG. 2.—Histogram showing time of exposure to anaesthetic related to delay in onset of "continuing" respirations. The three groups are similarly distributed throughout the range.

obtained: $t=1.158$, and with this value $0.25 < P < 0.30$. This value of r does not differ significantly from zero, and therefore the evidence for the correlation is inadequate.

The babies were divided into three groups according to whether they were exposed to anaesthesia lasting up to 10 minutes and under, 10 to 15 minutes, or over 15 minutes (Fig. 2). The variability in the behaviour in delay in onset of continuing respirations of these three groups was similar: $\chi^2=27.94$, $n=34$, $\sqrt{2\chi^2} - \sqrt{2n-1} = -0.713$.

Discussion

Our results are apparently in conflict with established teaching. It appears that with duration of anaesthesia between 5 and 30 minutes there is no correlation between duration of anaesthesia to which the child is exposed and the delay in onset of respiration. However, closer investigation reveals: (1) Our cases were almost entirely limited (93 out of 102) to those exposed to anaesthesia for a duration of 5 to 20 minutes. No comment can be made on what happens outside these limits. (2) One hundred caesarean sections is not a very large sample on which to base dogmatic statements, as a large number of variables enter into the causation of apnoea neonatorum. However, even a trend towards prolongation of delay in breathing with increased time of exposure to anaesthesia is not evident. (3) All anaesthetics given to the mother may delay somewhat the initial respirations of the infant (Cole *et al.*, 1939). However, if the level of anaesthesia be kept light, the amount of anaesthetic passing to the infant may be kept to the minimum, and thus dangerous—from the point of view of respiratory activity—levels of ether in the blood (Smith and Barker, 1942) of the foetus be avoided.

Conclusion and Summary

In cases of caesarean section, provided that the child is extracted within 20 minutes of induction of anaesthesia of the mother, we feel that a calm unhurried approach by the operating team will not adversely affect the child. 102 infants born by caesarean section were investigated regarding the correlation between the duration of anaesthesia to which their mothers were exposed and the delay in breathing which the infants showed. When the duration of anaesthesia did not exceed 20 minutes, no such correlation was observed.

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The Royal Society of Health has informed the Ministry of Health that it is concerned about the pollution of water supplies by copper, galvanized, and polythene pipes. It suggests that the Water Pollution Research Laboratory should investigate the matter. The society states that since the war there have been instances of severe corrosion in copper pipes, particularly in the west of England, and that in other districts there have been reports of an alarming content of copper in piped water. "Illness," the society continues, "has been attributed to the poisoning of the water." Such a hazard is denied by the Copper Development Association, which states: "In this country alone about 30,000 miles of copper pipe are at present installed annually for domestic purposes, and the association has not yet come across any authentic case of copper poisoning from normal water supplies."

EXPERIENCE WITH "NOBECUTANE" IN MINOR SURGERY

BY

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The new plastic dressing "nobecutane" has already been used in general surgical and abdominal operations, and a survey on these lines has been made by Rob and Eastcott (1954). There has, however, been no report on its use in minor and casualty surgery. Dissatisfaction with the progress of wound healing in many military personnel treated in the casualty department of a large military hospital led to a trial of other forms of dressing. This report describes such a trial of nobecutane.

The conventional gauze dressings with a bandage or zinc oxide plaster in minor operations and in the treatment of the lacerations of minor trauma have always been open to criticism. They are easily disturbed by normal use of the affected part and soon become soiled and dirty, with resulting maceration and contamination of the wound. Service men in general are exposed to many sources of trauma in the course of their duties, and conditions for the maintenance of a clean and dry wound are rarely available. To expect a cookhouse orderly whose hands are frequently cut by knives and edges of tins to keep his upper limbs free from moisture and dirt is to expect the impossible.

Nobecutane is an acrylic resin dissolved in a mixture of acetic esters. When applied to the skin this solvent evaporates, leaving a transparent, adherent, and elastic film. The dressing is inert, impervious to bacteria, but pervious to evaporation from the skin surface (Olow and Hogeman, 1953). It is flexible and adheres to the edges of the wound without irritation or maceration of the underlying skin. The principal disadvantages of the dressing are stated to be haemorrhage from the wound edges if these come into contact with the dressing, and difficulty in suture removal if the plastic film is too thick (Wallgren, 1954). The haemorrhage is thought to be due to both a haemolysing and a vasodilating effect of the solvent.

Method of Trial.—Over a six-months period 48 wounds resulting from minor surgical operations and 60 sutured lacerations were dressed with nobecutane. All the operations were performed in a small casualty theatre, using the "no touch" technique and a full aseptic procedure. If the lacerated wound was found to be grossly contaminated, a prophylactic course of penicillin was given; nine of these cases received such a course. A form was prepared on which the patient's name and occupation could be entered together with a description of the wound and the suture technique. The wounds were inspected on the first, fifth, seventh, and fourteenth days after initial treatment and the state of the wound and the treatment it required were recorded.

Results and Complications

At the end of the six-months period of trial it was apparent that the wounds treated with nobecutane were healing quickly and soundly, and that while this was taking place all the patients had been able to continue with their normal duties without running the risks of maceration and contamination of their wounds. The impression was gained that there had been a decrease in the incidence of wound infection, though a comparable control series was not taken.