Malformations Recorded on Birth Certificates Following A2 Influenza Epidemics

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INTEREST in the relationship of viral infections to malformations was first aroused by the discovery that the human embryo could be damaged by rubella (*1*), and several attempts were made in studies following the appearance and worldwide spread of Asian influenza (type A2) in 1957–58 to determine whether this disease also might be teratogenic.

A review (*2*) of most of these studies suggested that their results were inconclusive. The reported malformations in children whose mothers might have had influenza in early pregnancy were much less homogeneous than those observed in children following exposure to known teratogens such as rubella and thalidomide. Although statistically significant increases in the incidence of particular types of defects in such children were reported from Dublin, Ireland (*3*), and Birmingham, England (*2, 4*), the types of defects were different in the two cities. Anencephalus, meningocele, encephalocele, and spina bifida were especially common among the children of Dublin women who reported an attack of influenza during pregnancy. In Birmingham the incidence of cleft lip, esophageal atresia, anal atresia, and exomphalos, and especially of cases exhibiting combinations of these malformations with each other or with other defects, was significantly increased among children who had been in the early stages of intrauterine life when influenza was epidemic.

Data from other sources apparently have not been examined for consistency with either set of findings. In this study we examined U.S. birth records for evidence of increases in the incidence of defects following influenza epidemics.

Material Used

At the National Communicable Disease Center (NCDC), Public Health Service, weekly returns of the number of deaths ascribed to in-

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fluenza and pneumonia are received from 122 cities of the United States. For the analysis of these figures the country is divided into nine regions, and the number of deaths reported from the cities of each region is compared with the number to be expected if the normal secular trend and seasonal fluctuation in mortality were to continue undistorted by epidemics. Influenza is assumed to be epidemic if two or more consecutive weekly totals exceed the expected figures by at least 1.64 standard deviations (*5*).

At the Dental Health Center of the Public Health Service, San Francisco, live-birth certificates selected from the 1956–61 files of four States and from the 1962–65 files of up to 29 States and two other reporting areas, including 63 of the 122 cities surveyed by NCDC, were abstracted and coded on punchcards or magnetic tape (*6*, *7*). The selected certificates comprised (*a*) all those on which clefts of the lip and palate were recorded, (*b*) those for 1961–65 on which malformations of any kind were noted, and (*c*) a control sample consisting of the certificates of 0.5 percent of the related births in 1956–60 and 1 percent of those in 1961–65. Whenever a certificate in group (*a*) or (*b*) was selected for group (*c*), the next certificate filed was substituted for it in this sample. The data abstracted from each selected certificate included month, county of mother's residence, and any malformations that were reported. For births in 1961–65, the day was also recorded.

The times and places at which A2 influenza was epidemic during the period when the children represented by these certificates were at risk of malformation were estimated from NCDC's influenza and pneumonia mortality statistics, checked against records of the strain of virus most prevalent when mortality was high. By far the most severe epidemic that occurred when any of the children born in 1962–65 were at risk was in early 1963. Substantial increases in influenza and pneumonia mortalities for 2 or more consecutive weeks of this period occurred in 17 of the 63 cities for which data on malformed births as well as influenza deaths were available. At approximately the same time that each of these increases occurred, influenza was epidemic (according to NCDC's formal criteria) in the NCDC region containing the city concerned. The 1962–65 birth records of children whose mothers resided in the Standard Metropolitan Statistical Areas (SMSA's) containing these 17 cities were divided into high and low risk groups. The high risk group comprised cases in which, according to the birth dates recorded, the period of high mortality in the city concerned might well have coincided with early pregnancy.

Complete mortality data for the 122 cities surveyed by NCDC were not available for all years when the children born in 1956–61 were at risk. The dates when A2 influenza was epidemic during this period were estimated for each State by reviewing the mortality and absenteeism statistics and reports of epidemics received by NCDC from State health departments. The 1956–61 birth records were divided accordingly into high and low risk groups.

Statistical Methods

The incidence of various types of malformations in the 1956–61 and 1962–65 series was examined for possible post epidemic increases by computing two ratios for each epidemic and type of defect:

1. *Crude incidence ratio.* Ratio between the malformation incidence rates observed among high risk births after the epidemic concerned and among low risk births in the same population.

2. *Standardized incidence ratio.* Ratio of the incidence rate observed in the high risk group (i_1) to an estimate of the incidence rate to be expected during the season and year when these children were born (i_e). Use of such a ratio is desirable because seasonal and secular variations in incidence unrelated to influenza are known to occur (*8*, *9*) and would tend to distort the crude ratio if the timing of an influenza epidemic were such that the high risk group were born during a season or year of high or low incidence.

Even under these conditions, it might be expected that, if influenza had no effect, the ratio of incidence when the high risk group was born (i_1) to incidence during the remainder of the post epidemic year (i_2) would be of the same order as the equivalent ratio for low risk years $\left(\frac{I_1}{I_2}\right.$ where I_1 is the incidence rate in the

months corresponding to those in the post epidemic year when the high risk births occurred, and I_2 is the rate during the other months of the low risk years). The value of i_1 to be expected in these circumstances (denoted i_e before) can therefore be estimated from the equation $\left(\frac{i_e}{i_2}=\frac{I_1}{I_2}\right)$, provided that i_2, I_1, and I_2 are calculated first. The estimates of i_e that were used in calculating the standardized incidence ratios were obtained in this way. The division of time into post epidemic and low risk years that this method requires was carried out by reference to the midpoint of each period when high risk children were born. The time from 6 months before to 6 months after each of these midpoints was defined as a post epidemic year.

For each crude or standardized ratio above unity, indicating a post epidemic excess of malformed births, the significance of the excess was evaluated by computing χ^2 with Yates' correction from the relevant basic data and halving the statistical probability ascribed to it in the standard tables. Increases and decreases in incidence after epidemics each account for half of this probability, and one is not concerned with the decreases, since the hypothesis under test is that incidence rises after epidemics.

For each crude ratio, χ^2 was computed from the numbers of malformed and other children in the high risk group and those born at other times. For each standardized ratio, χ^2 was computed for a contingency table containing in the following sequence the numbers of malformed births (n_1, n_2, N_1, N_2) from which the incidence rates i_1, i_2, I_1, and I_2 were derived:

Period	*Post epidemic years*	*Low risk years*
High risk months	n_1	N_1
Low risk months	n_2	N_2

This method of testing significance is not entirely valid except in the absence of interaction between season and year of birth in the related population. The number of malformed births in the high risk group (n_1) would, for example, tend to be higher than expected if during the high risk year a greater proportion of all births than in other years occurred in high risk months.

There was some evidence of interaction of this kind in 1962–65, when the number of control births to be expected in the high risk period (given the numbers of controls actually born in low risk months or years) was only 95.6 percent of the number observed. To offset the effects of this excess of all high risk births on the numbers of malformed births, each χ^2 test that suggested a significant increase in one of the standardized ratios for 1962–65 was repeated after

Table 1. Live-born children classified according to likelihood of exposure to A2 influenza in early intrauterine life, 1956–61

Reported dates of epidemics	Months with midpoints 26–40 weeks after epidemics (high risk)	Live births [1] A: In high risk months listed	Live births [1] B: In low risk months of 1956–61
California			1, 980, 752
Jan 10–Feb. 13, 1960	July–November 1960	162, 838	
Pennsylvania			1, 216, 584
Oct. 6–Dec. 21, 1957	April–September 1958	190, 678	
Feb. 2–Mar. 22, 1958	August–December 1958		
Mar. 22–May 2, 1959	October 1959–January 1960	80, 882	
Wisconsin			466, 024
Oct. 6–Dec. 21, 1957	April–September 1958	72, 824	
Jan. 26–Mar. 29, 1958	August–December 1958		
Dec. 27, 1959–Mar. 5, 1960	July–November 1960	43, 520	
Total		550, 742	3, 663, 360

[1] Figures derived from "Vital Statistics of the United States," 1956 through 1961.

Table 2. Number of clefts reported among live births in high and low risk months,

State	Cleft palate alone				Cleft lip alone			
	a High risk births	b Low risk births	Crude incidence ratio [1]	Standardized incidence ratio [2]	a High risk births	b Low risk births	Crude incidence ratio [1]	Standardized incidence ratio [2]
California	64	703	1. 11	0. 97	59	695	1. 03	0. 94
Pennsylvania:								
First high risk period	72	391	1. 17	1. 33	52	395	. 84	. 71
Second high risk period	22		. 85	. 96	28		1. 07	1. 37
Wisconsin:								
First high risk period	25	205	. 78	. 87	19	151	. 81	. 76
Second high risk period	14		. 73	. 80	19		1. 35	1. 44
All three States:								
With exomphalos, esophageal, or anorectal defect	2	19	. 70	3. 00	1	5	1. 33	----------
With other defects only	42	314	. 89	1. 26	16	72	1. 48	2. 80
Without other defects	153	966	1. 05	. 83	160	1, 164	. 91	1. 00
Total	197	1, 299	1. 01	. 94	177	1, 241	. 95	1. 08

[1] Crude incidence ratio equals $\frac{a}{A} \div \frac{b}{B}$. For explanation of A and B, see table 1.

[2] For explanation of standardized incidence ratio, see text, p. 972. Standardized incidence ratios for the 3 States combined cover 1959–61 only.

replacing n_1 by $0.956n_1$. All estimates of statistical significance given for these ratios in the results were obtained in this way. The 1956–61 data did not require any such correction.

Results

As the data for births in 1956–61 are considerably less extensive than those for 1962–65, the findings for the two periods are presented separately.

Births in three States in 1956–61. Data on births in 1956–61 were available for California, Hawaii, Pennsylvania, and Wisconsin. In Hawaii, no epidemic was observed during the years when the children born in 1956–61 were passing through early intrauterine life. One outbreak of A2 influenza was defined in California and two each—the first occurring in two waves—in Pennsylvania and Wisconsin. The distribution of births in relation to these epidemics is shown in table 1. Separate figures are given for the months with midpoints 26 to 40 weeks after epidemics, since the children who were born then are considered to have been at high risk of exposure to maternal influenza during the teratogenic period. The assumption that this period might be at any time between 26 and 40 weeks before birth was also made in earlier papers (*2*, *4*), to allow for variations in length of gestation and in the age at which teratogenesis occurs.

The frequency of clefts occurring in each of the five high risk groups and in other children is compared in table 2. Because of the report that influenza epidemics may be followed by a particularly marked increase in the incidence of clefts associated with other defects, especially esophageal atresia, anal atresia, and exomphalos, separate figures are given for such cases. The standardized ratios for the three States combined were computed only for 1959–61 because the method used is not suitable for analyzing data in which any community is represented by more than one high risk group of births unless these births occurred in the same year or in the same months of different years. The basic data used in computing the expected values for the standardized ratios are not given since this material would occupy more space than its importance warrants.

as defined in table 1, 1956–61

Cleft palate with cleft lip			
a High risk births	b Low risk births	Crude incidence ratio [1]	Standardized incidence ratio [2]
80	1, 065	0. 91	0. 96
95	631	. 96	. 84
40		. 95	. 88
51	303	1. 08	1. 48
33		1. 17	1. 57
2	29	. 46	. 40
28	253	. 74	. 56
269	1, 717	1. 04	1. 11
299	1, 999	. 99	1. 01

No ratio listed in table 2 is significantly in excess of unity. The only malformation with any evidence of a consistent pattern is cleft lip, the incidence of which was relatively high after the second epidemics in both Pennsylvania and Wisconsin but not after the first widespread outbreaks in any of the three States.

Births in 17 SMSA's in 1962–65. Figures for the 17 areas yielding data on the frequency of malformations and the duration of the influenza epidemic in 1963 are shown in table 3. Birth statistics from all 17 areas were available for 1963–65, and from 13 for 1962. Because of a misunderstanding, the high risk periods for these births were defined as starting 188 days after the onset of the epidemic, whereas the interval allowed in the other studies was 182 days. This discrepancy is not likely to have appreciably biased our findings.

The original material included details of all malformations reported on birth certificates, coded according to the Dental Health Center

Table 3. Children born alive in 1962–65, classified according to likelihood of exposure to 1963 influenza epidemic in early intrauterine life

Standard Metropolitan Statistical Area	Reported dates of 1963 epidemic	Live births 188–280 days after epidemic (high risk)		Other live births (low risk)		Crude incidence ratio $\frac{a}{A} \div \frac{b}{B}$	Standardized incidence ratio (see text)
		a Affected [1]	A Total [2]	b Affected [1]	B Total [2]		
Baltimore, Md. (part)	Jan. 27–Feb. 23	61	11, 900	714	129, 700	0. 93	0. 73
Birmingham, Ala	Mar. 3–16	3	4, 200	87	45, 900	. 38	. 51
Chattanooga, Tenn	Mar. 10–30	8	1, 700	65	19, 200	1. 39	1. 43
Detroit, Mich	Feb. 24–Mar. 16	145	26, 500	1, 528	303, 600	1. 09	1. 18
Grand Rapids, Mich	Mar. 10–Apr. 6	13	3, 200	165	31, 000	. 76	1. 13
Little Rock, Ark	Feb. 10–Mar. 16	8	2, 600	114	22, 100	. 60	. 58
Louisville, Ky., and Indiana (part).	Mar. 3–30	9	4, 600	101	53, 900	1. 04	1. 13
Memphis, Tenn	Mar. 3–16	3	5, 100	25	57, 400	1. 35	1. 40
Milwaukee, Wis	Feb. 17–Mar. 23	58	9, 000	674	98, 000	. 94	1. 30
Montgomery, Ala.[3]	Feb. 24–Mar. 16	4	1, 000	40	10, 000	1. 00	1. 35
New Orleans, La	Mar. 3–16	24	7, 500	229	81, 800	1. 14	1. 08
New York, N.Y. (part) [3]	Jan. 27–Mar. 2	292	58, 300	2, 170	428, 700	. 99	1. 12
Norfolk-Portsmouth, Va	Feb. 17–Mar. 9	19	5, 100	220	54, 200	. 92	. 87
Philadelphia, Pa., and New Jersey (part).	Feb. 17–Mar. 23	164	27, 000	1, 451	275, 000	1. 15	1. 02
Pittsburgh, Pa	Mar. 3–23	94	16, 700	859	155, 400	1. 02	1. 06
Richmond, Va.[3]	Feb. 3–23	11	3, 400	116	23, 600	. 66	. 82
St. Louis, Mo.[3], and Illinois (part).	Mar. 3–30	54	12, 200	452	94, 600	. 93	1. 06
Total		970	200, 000	9, 010	1, 884, 100	1. 01	1. 05

[1] Exhibiting 1 or more of the malformations listed in table 4.
[2] Estimated from 1 percent sample of controls.
[3] 1963–65 births only.

classification (*10*). Categories in this classification that comprise miscellaneous or ill-defined defects grouped according to site were not included in the present inquiry, except for two groups (esophageal and rectal) in which a single type of malformation (atresia, sometimes combined with fistula) was apparently predominant. Minor defects and those reported in less than 0.05 per 1,000 live births were also excluded. The remaining conditions are listed in table 4, and the affected children enumerated in table 3 are those for whom these defects were described. The incidence of affected children did not increase significantly following the epidemic in any of the 17 areas.

Findings for specific types of defects are summarized in table 4. The standardized ratio for cleft lip and both ratios for defects of the upper limbs are significantly in excess of unity. More than half of the reduction deformities of upper limbs apparently involved digits only (table 5). Most of the increase in reduction deformities observed among the high risk births was in defects of this type. The slight excess exhibited by supposedly more extensive deformities may only indicate that some digital defects were inadvertently allocated to this group—a likely occurrence in view of the incompleteness of information on many birth certificates.

Defects limited to the thumbs and radii are enumerated separately in table 5 because they showed an increase, although not a statistically significant one, after epidemics in Birmingham, England (*2*). In the present series, they showed less increase than reduction deformities involving other parts of the upper limbs.

The high risk births included no children with cleft lip combined with reduction deformities of the limbs, such as might have been expected if the increases shown by these two types of defects had a common cause.

The 1962–65 data are used in table 6 to explore the suggestion (*2*, *4*) that influenza epidemics may be followed by a particularly marked increase in the proportion of births in which cleft palate with cleft lip, cleft lip alone,

Table 4. Numbers of selected malformations reported among live births in 17 Standard Metropolitan Statistical Areas, 1962–65

Type of malformation	a High risk births	b Low risk births	Crude incidence ratio $\left(\frac{a}{200{,}000} \div \frac{b}{1{,}884{,}100}\right)$	Standardized incidence ratio (see text)
Anomalies of nervous system:				
Anencephalus	52	466	1. 05	1. 32
Spina bifida, encephalocele	126	1, 097	1. 08	1. 13
Hydrocephalus	57	579	. 93	. 93
Microcephalus	2	105	. 18	. 24
Anomalies of digestive system:				
Cleft palate	55	556	. 93	. 93
Cleft lip	62	464	1. 26	[1] 1. 47
Cleft palate with cleft lip	74	779	. 89	1. 10
Esophageal defects	11	135	. 77	1. 12
Anorectal defects	39	378	. 97	. 80
Hypospadias	123	1, 218	. 95	. 93
Anomalies of musculoskeletal system:				
Clubfoot	232	2, 247	. 97	. 89
Reduction deformities:				
Upper limbs only	51	320	[2] 1. 50	[2] 1. 91
Lower limbs only	13	129	. 95	1. 23
Upper and lower limbs	6	45	1. 26	1. 32
Limbs unspecified	2	22	. 86	. 75
Congenital dislocation of hip	14	150	. 88	. 77
Diaphragmatic hernia	10	114	. 83	1. 01
Down's disease	92	810	1. 07	1. 16
Exomphalos	26	272	. 90	1. 16

[1] $0.05 > \frac{P}{2} > 0.01$. [2] $0.01 > \frac{P}{2} > 0.001$.

Table 5. Extent of reduction deformities of upper limbs

Extent of deformity	Number of children at high risk			Number of children at low risk			Incidence ratios	
	Legs also affected	Legs not affected	Total	Legs also affected	Legs not affected	Total	Crude	Standardized
Limited to digits:								
Thumbs only	0	4	4	3	26	29	1. 30	1. 70
Other	4	30	34	21	160	181	1. 77	2. 54
Not limited to digits:								
Radial sides of limbs only	0	0	0	0	6	6	0	0
Other	2	17	19	21	128	149	1. 20	1. 39

Table 6. Frequency of single and multiple defects among children listed in table 5

Defects of special interest in each child	Children with multiple defects [1]				Children with single defects			
	Number at high risk	Number at low risk	Crude incidence ratio	Standardized incidence ratio	Number at high risk	Number at low risk	Crude incidence ratio	Standardized incidence ratio
2 or more special-interest defects	5	38	1. 24	1. 67				
Only 1 special-interest defect	32	347	. 87	. 95	170	1, 605	1. 00	1. 14
Cleft lip alone	1	40	. 24	. 62	61	421	[2] 1. 36	[2] 1. 51
Cleft palate with cleft lip	14	87	1. 52	[2] 2. 92	57	682	. 78	. 94
Esophageal defects	3	21	1. 35	2. 46	6	92	. 61	. 80
Anorectal defects	8	124	. 61	. 42	27	225	1. 13	. 96
Exomphalos	6	75	. 75	. 69	19	185	. 97	1. 50
No special-interest defects	110	1, 105	. 94	1. 09	653	5, 915	1. 04	1. 03
Total	147	1, 490	. 93	1. 07	823	7, 520	1. 03	1. 05

[1] Multiple defects are defined as combinations of malformations from 2 or more of the 62 categories of the Dental Health Center classification. Source, reference 10.

[2] $0.05 > \frac{P}{2} > 0.01$.

esophageal atresia, anal atresia, and exomphalos are associated with each other or with other defects. In the present series, the influenza epidemic was followed by increases in the incidence of combinations of these five defects and of cases in which esophageal defects or cleft palate with cleft lip were associated with other malformations. None of these increases was significant, however, except for the high standardized ratio for children exhibiting both cleft palate with cleft lip and other defects. The increase in incidence of cleft lip without cleft palate (table 4) was limited to children with no other defects.

Discussion

The study results raised two methodological problems that must be noted before any biological implications can be discussed. The first problem, since the reporting of malformations on birth certificates was incomplete (*11*), is that differences may have existed between the proportions reported 26 to 40 weeks after epidemics and at other times. If true, the cited incidence ratios would have been biased. The fact that influenza is not reputed to be a potent teratogen makes it unlikely that an epidemic itself would have such delayed effects on the quality of reporting, but regular seasonal or more prolonged changes in reporting habits not resulting from epidemics may very possibly have occurred. However, although changes of this kind may have biased the crude incidence ratios, any such bias would have tended to be eliminated when the standardized ratios were calculated.

The second problem is whether it is right even

to consider biological explanations when the number of statistically significant results observed (six, with a probability below 5 percent, among the 161 crude and standardized ratios shown in tables 2–4 and 6) is no greater than the number that might be expected to occur by chance. Certainly, it would be unwise to attach much importance to findings of this kind unless they confirmed trends observed in other data.

Unfortunately, we have no data with which to compare the excess of finger defects following the influenza epidemic of 1963 (table 5). The increase at this time in the incidence of cleft lip without cleft palate (table 4), however, can be compared with observations made on four other occasions when a population previously affected by the 1957–58 pandemic of A2 influenza was reexposed—Pennsylvania in 1959 and Wisconsin in 1959–60 (table 2) and Birmingham, England, in 1959 and 1960–61 (*2*). Data covering the 1957–58 epidemic in these three communities and the first widespread outbreak in California (in 1960) are available from the same sources. All five reexposures were followed by increases in the incidence of either cleft lip alone (in the American series) or cleft lip with or without cleft palate (treated as a single entity in the Birmingham series), but no increase occurred after the first major exposure in any of the four communities from which data for this period were analyzed. If these observations are meaningful, they may indicate that cleft lip can be caused by something that happens when pregnant women who already have some immunity to the A2 virus are reexposed.

The U.S. data provide little support for the view that influenza was responsible for the excessive number of certain other defects, notably esophageal atresia, anal atresia, and exomphalos, observed in children following the epidemics in Birmingham, or for the high incidence of neural tube defects among the children of mothers with a history of influenza in Dublin, Ireland (*3*). With one exception (anorectal defects) the standardized incidence ratios for all the corresponding categories in the U.S. data for 1962–65 exceeded unity, but no excess was significant (table 4). In contrast to the Birmingham data on cleft lip, esophageal atresia, anal atresia, and exomphalos, no significant increase occurred in the incidence of children with two or more of these defects following the 1963 epidemic in the present series, and no increase was noted in the overall incidence of combinations involving only one of these defects (table 6). The proportion of children with multiple defects increased among those with cleft lip and palate and decreased among those with cleft lip alone following the 1963 epidemic (table 6), but the reverse was true following the epidemics to which the 1956–61 series of births was exposed (table 2).

There is little in this report to suggest that A2 influenza causes a syndrome of defects. The only malformation that showed a significant association with more than one outbreak was cleft lip.

Summary

Records of the National Communicable Disease Center, Public Health Service, were used to identify periods when A2 influenza was widespread in California, Pennsylvania, and Wisconsin in 1955–61 and in 17 Standard Metropolitan Statistical Areas in the eastern United States that were affected by the epidemic of early 1963. Encoded abstracts of the birth certificates of children born in the three States in 1956–61 and in the 17 metropolitan areas in 1962–65 were subdivided according to whether or not birth occurred approximately 26 to 40 weeks after the epidemics. The incidence of clefts of the lip and palate in these subdivisions of the 1956–61 data was compared, and the 1962–65 data were used for similar comparisons of all the common major malformations that were distinguished in the records used.

Reduction deformities of the fingers were especially common among births following the 1963 epidemic. As in a previous series from Birmingham, England, the incidence of cleft lip did not increase after the first widespread epidemic of A2 influenza but was higher after subsequent outbreaks. The other defects examined showed no significant increase in incidence after epidemics.

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Tearsheet Requests

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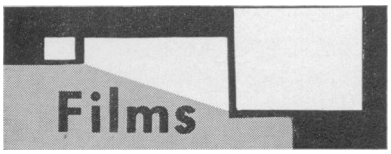

Blood Pressure Readings. *Motion picture, 16 mm., color, sound, 18 minutes, 1968. Order No. M–1582.* Produced for the Heart Disease and Stroke Control Program, National Center for Chronic Disease Control, by the National Medical Audiovisual Center.

AUDIENCE: Field investigators employed in studies involving the measurement of blood pressure, medical and nursing students, and paramedical personnel.

SUMMARY: Developed to provide a test of the reliability of blood pressure readings by one observer or a group of observers. Presents a series of clinical blood pressure measurements using a mercury sphygmomanometer and stethoscope. Each scene shows a column of mercury descending on a sphygmomanometer scale with accompanying stethoscopic sounds. Following a practice reading, 14 separate readings are presented as test segments. Viewers record their observations during the pause between segments. The film was made during actual measurement of blood pressure on persons selected to provide a variety of responses.

AVAILABLE: Free short-term loan from the National Medical Audiovisual Center (Annex), Chamblee, Ga. 30005, Attention: Film Distribution. Purchase from DuArt Film Laboratories, Inc., 245 West 55th Street, New York, N.Y. 10019.

Intensive Respiratory Care. *Motion picture, 16 mm., color, sound, 30 minutes, 1968. Cleared for television. Produced by John Sutherland Productions, Inc., for the Chronic Respiratory Diseases Control Program, Health Service and Mental Health Administration.*

AUDIENCE: Restricted to physicians, nurses, medical and nursing students, and paramedical personnel to acquaint them with techniques for diagnosing and treating acute respiratory failure.

SUMMARY: Presents an overview of an intensive respiratory care unit, with emphasis on the roles of doctor, nurse, and laboratory technician in diagnosis and treatment. The story is presented in dramatic form through short sequences showing various treatments of actual patients, with a 2-minute animated segment showing lung pathology and physiology.

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