Z. Rechtsmed. 83, 303—312 (1979)

Zeitschrift für
Rechtsmedizin

Is the Aeration of the Lungs a Reliable Sign of Live Birth?

L. Buris, I. Töröcsik, and S. Poczkodi

Institute of Forensic Medicine, Medical University of Debrecen, 4012 Debrecen, Hungary

Ist die Luftfüllung der Lungen ein zuverlässiges Zeichen einer Lebendgeburt?

Zusammenfassung. Ob ein Kind lebend oder tot geboren wurde, ist eine der wichtigsten rechtsmedizinischen Fragestellungen. Entfaltete, luftgefüllte Lungen sind nicht immer ein sicherer Hinweis dafür, daß das Neugeborene geatmet hat. Unter verschiedenen Umständen können zuerst luftgefüllte Lungen luftleer werden, andererseits aber auch eine Totgeburt als beatmet erscheinen lassen. Nur die kritische Bewertung histologischer und makroskopischer Befunde kann zur Lösung des Problems beitragen.

Schlüsselwörter: Neugeborenes, lebend oder tot – Totgeburt, Lungenbefunde

Summary. One of the most important medico-legal questions is whether an infant was born alive or not. The aerated lungs do not mean in every case that the newborn had taken a breath. Under various conditions the aerated lungs may turn into unaerated ones and, on the other hand, a stillborn's lungs may seem aerated. The critical evaluation of the histological and macroscopical examinations may aid in solving the question.

Key words: Newborn, living born or not – Stillborn, estimation of lungs

At the time of birth the alveoli of the lungs are undeveloped and their structure is tubular, containing a few desquamated cells in proteinaceous fluid (Spencer, 1962).

The elastic tissue is undeveloped and it is situated only in the wall of the large bronchi. In the 3 months-old infant the elastic fibres are developed both in the wall of the alveoli and in the blood vessels.

After the aeration of the lungs the alveoli expand, the volume of the lungs

Offprint requests to: Dr. L. Buris (address see above)

0044-3433/79/0083/0303/$ 2.00

increases significantly and their marginal parts round off. The alteration of the lungs is similar at normal and artificial aeration alike. From the cut surface of the aerated lungs—immersed in water—air bubbles press out. Together with the start of respiration the blood circulation also changes. The fetal circulation is characterised by blood flow from the a. pulmonalis to the aorta through the ductus arteriosus. This circulation changes because of the occlusion of fetal shunts immediately after the birth. As a result of occlusion, the blood volume, flowing through the lungs increases and the alveolar capillaries expand, together with the expansion of alveoli (Bonham-Cartner, 1957; Avery, 1959). This process results in the air getting into the lungs.

Changes preventing the suitable filling of the lungs' capillaries also prevent the normal expansion of the lungs. The lungs' expansion cannot take place if the right heart is not able to keep sufficient blood volume circulating. For a long time it had been the conception that the lungs expand progressively after the birth. The lungs' expansion after some respiratory movements and at the same time, the decrease of the size of the right heart chamber were verified by X-ray examinations (Lind, 1965).

The neonatal collapse of the lungs may develop from:

1. Congenital incomplete expansion, because of a defect of the normal expanding mechanism, or the insufficient development of lungs, in relation to the infant's immaturity.
2. Collapse due to obstruction of air passages from hyalin membrane disease, or glottic obstruction by mucus.
3. Congenital disease preventing the lungs' expansion (Spencer, 1962).

The above mentioned problems might cause neonatal collapse of the lungs. We found expressed collapse with those infants who died a few days after birth and none of the afore mentioned problems with them. The collapse of these neonates may result from agonal air absorption and insufficient respiration.

The hydrostatic test was first described by Rayger in 1675; the test was applied widely in forensic medical practice, without sufficient criticism. The floating lungs means, the infant was born alive and a negative test means still-birth. In many cases there was a contradiction between the hydrostatic test and anamnestic data and because of these the test was almost completely forgotten in practice. The test verifies only the lungs' density is less than that of water. False positive reaction may develop after putrefaction, fixation with alcohol etc. The floatation test may be appreciated in forensic medical practice as a screening procedure.

The fluorescence microscopical examination of the infant lungs' elastic fibres was made by Prokop (1971). The fibres do not give characteristic changes which would be typical of live birth.

In our cases when we examined infant lungs, we had reliable anamnestic data. Among these experimental materials there were live born infants whose lungs were atelectatic, but conversely, there were infants who died intrauterine and their lungs appeared aerate, even microscopically. In view of the above mentioned cases we examined lungs, originating from live born and intrauterine died infants alike.

Material and Methods

Seventy-two infants' lungs were examined. The lungs were removed with bronchi and trachea and fixed in 10% formaline.

Samples were taken from each lobe, embedded in paraffin. The slides were stained haemalaun-chromothrop, van Gieson, trichrome by Endes and silverimpregnation.

We signed +, when there were only a few opened alveoli, at ++ sign many opened alveoli and expanded bronchi were seen, at +++ sign atelectatic parts were only seldom found in the slides. We examined the capillarisation and the number of opened capillaries of lungs. Autolytic processes were observed carefully. We divided our material into three groups.

I. Mature Newborns

Nineteen newborns were in this group, 3 h—10 days age of life. Table 1 contains the data of this group.

II. Premature Infants

Twenty-five prematures were examined, the birth weight was 900—1800 g after 3—7 days age of life (Table 2).

III. Stillborn Fetuses

Among 28 fetuses 2 were macerated (Table 3).

Table 1. Matured newborns

Name	Age	Section no.	Examinations		Cause of death
			Histological	Macroscopical	
1. T.A.	5 h	261/1969	–	+	Inhalation of amniotic fluid
2. M.J.	1 d	502/1969	–	–	Infanticide
3. E.I.	2 d	716/1969	–	+	Atelectasis of lungs
4. D.A.	2 d	169/1970	–	+	Atelectasis of lungs
5. R.A.	75 min	653/1969	+	++	Prolapsed umbilical cord
6. V.V.	3 h	407/1970	++	–	Hyaline membrane disease
7. M.B.	3 h	696/1970	+	+	Strangulation by cord
8. D.F.	7 h	778/1969	++	–	Hyaline membrane disease
9. S.K.	8 h	85/1969	+++	–	Hyaline membrane disease
10. P.M.	10 h	364/1970	++	–	Hydrocephalus
11. S.I.	13 h	512/1970	+++	++	Prolapsed umbilical cord
12. C.G.	14 h	616/1970	++	–	Hyaline membrane disease
13. S.T.	1 d	592/1970	++	+	Atelectasis of lungs
14. K.L.	1 d	508/1970	++	+	Intracranial haemorrhage
15. V.E.	1 d	789/1969	+	–	Intracranial haemorrhage
16. B.S.	11 h	302/1970	+	–	Polycystic kidneys
17. G.M.	1 d	231/1969	+	+	Inhalation of amniotic fluid
18. B.G.	2 d	21/1970	+	–	Hyaline membrane disease
19. S.O.	2 d	591/1970	+	++	Atelectasis of lungs

Table 2. Premature infants

Name	Age	Section no.	Examinations		Cause of death
			Histo-logical	Macro-scopical	
20. K.J.	3 h	730/1970	–	–	Atelectasis of lungs
21. M.Z.	14 h	510/1970	–	–	Intracranial haemorrhage
22. J.C.	1 d	553/1970	–	–	Immaturity
23. S.A.	7 d	354/1970	–	–	Pneumonia
24. M.P.	6 h	778/1969	+++	+++	Hyaline membrane disease
25. O.T.	17 h	117/1970	++	++	Intracranial haemorrhage
26. H.H.	22 h	665/1970	+++	++	Hyaline membrane disease
27. C.I.	1 d	372/1969	+	+	Intracranial haemorrhage
28. S.J.	2 h	753/1969	++	–	Immaturity
29. S.E.	2 h	643/1970	++	+	Hyaline membrane disease
30. B.M.	8 h	722/1970	+	+	Inhalation of mucus
31. M.Z.	10 h	402/1970	+	–	Immaturity
32. K.S.	16 h	202/1969	+	+	Hyaline membrane disease
33. O.L.	10 h	118/1970	+++	+++	Pneumonia
34. D.E.	17 h	692/1970	+++	+++	Hyaline membrane disease
35. M.J.	18 h	99/1969	+	+	Immaturity
36. M.C.	19 h	734/1970	+	+	Hyaline membrane disease
37. S.E.	23 h	258/1969	==	+	Pneumonia
38. D.F.	1 d	528/1970	+	+	Intracranial haemorrhage
39. K.S.	1 d	734/1969	+	–	Intracranial haemorrhage
40. O.J.	1 d	688/1969	+	–	Inhalation of amniotic fluid
41. B.L.	1 d	614/1970	+++	+	Intracranial haemorrhage
42. R.E.	2 d	523/1970	+	–	Hyaline membrane disease
43. F.S.	5 d	24/1970	++	+	Hyaline membrane disease
44. B.L.	5 d	98/1969	++	+	Pneumonia

Results

Mature Infant Group

Among them we diagnosed non aerated lungs in four cases. The life span in these cases was from 5 h to 2 days. In the anamnestic data asphyxia, toxemia, respiratory distress were found. At autopsy the lungs were macroscopically atelectatic. In the other cases the lungs were aerated and cause of death was either hyeline membrane disease, or intracranial haemorrhage, or meconium inhalation etc.

Immature Infant Group

The life span was from 3 h to 5 days. Three infants' lungs were non-aerated, their age was 3 h, 14 h, 1 day. Two of the lungs were macroscopically non-aerated, and

Table 3. Stillborn fetuses

Name	Section no.	Examinations		Cause of death
		Histo-logical	Macro-scopical	
45. Ö. M.	536/1970	++	+	Strangulation with umbilical cord
46. N. L.	590/1970	–	–	Inhalation of amniotic fluid
47. P. E.	735/1969	–	–	Immaturity
48. T. S.	704/1970	++	+	Inhalation of amniotic fluid
49. Z. L.	363/1970	+	–	Inhalation of amniotic fluid
50. B. S.	668/1969	++	++	Abortus
51. S. L.	488/1970	–	–	Inhalation of meconium
52. V. A.	702/1969	+++	++	Hydrocephaly
53. S. S.	634/1970	++	+	Immaturity
54. V. M.	700/1969	++	+	Inhalation of amniotic fluid
55. K. J.	226/1970	–	–	Immaturity
56. K. S.	626/1969	–	–	Abortus
57. F. G.	537/1969	–	–	Inhalation of mucus
58. S. S.	641/1970	–	–	Intrauterine death
59. R. S.	675/1970	–	–	Strangulation with umbilical cord
60. L. G.	489/1970	–	–	Abortus
61. N. K.	383/1970	–	–	Inhalation of amniotic fluid
62. B. S.	747/1969	–	–	Rh incompatibility
63. B. A.	22/1970	–	–	Inhalation of meconium
64. T. S.	20/1970	–	–	Inhalation of amniotic fluid
65. K. J.	506/1970	–	–	Inhalation of meconium
66. T. J.	505/1970	–	–	Inhalation of meconium
67. B. J.	156/1970	–	–	Anencephaly
68. S. L.	475/1970	–	–	Abortus
69. B. A.	658/1969	–	–	Inhalation of amniotic fluid
70. H. S.	680/1969	–	–	Inhalation of amniotic fluid
71. F. S.	686/1969	–	–	Intrauterine maceration
72. B. E.	514/1969	–	–	Abortus

the third was dystelectatic. In anamnestic data we found marked asphyxia and resuscitation in the premature infants. In the other cases the lungs were aerated. In microscopic slides opened alveoli and atelectatic parts alike were found. The bronchi opened only partially. Hyaline membrane disease occurred in greater number than in the former group.

Among the stillborn fetuses only those were examined, who had reliable anamnestic data. We examined 28 corpses. Two of them died intrauterine, many days before the parturition the cause of death was either inhalation of amniotic fluid, Rh incompatibility, or prolapsed umbilical cord etc. Of the 28 examined lungs we were able to identify 6 aerated lungs. Microscopically large "aerated" parts were found, with positive floating test. In the above mentioned cases there

was no resuscitation effort in the anamnestic data. In the 50th infant there was intrauterine trepanition for a malformation—hydrocephalus—and the infant was born dead. The lungs were aerated macroscopically and microscopically alike. In the lungs of two macerated fetuses we found an extreme inhalation of amniotic fluid, certifying the intrauterine respiratory movement.

Discussion

Among the mature infants there were found "non aerated" lungs in four cases (Fig. 1). The alveoli were not opened, the bronchi were collapsed and the congestion of the vascular system of the intraalveolar septae was not pronounced. The postmortem air absorption may have caused the development of secundary atelectasis. The separation of the primary and secondary atelectasis was practically impossible when the newborn lived only for a few minutes. The histological features of the lungs give a characteristic finding after artificial respiration. There were little aerated parts in mostly atelectatic lung tissue, and neither the alveolar epithelium, nor the congestion of capillaries of the interstitium was typical of aerated lungs. A few opened alveoli were seen after considerable artificial respiration, but there was an acute interstitial emphysema, resembling histologically putrefaction of the lung, without any cytological signs of putrefaction (Fig. 2). The lungs of the premature infants expanded imperfectly. In the premature infants the lungs may become atelectatic because the right heart is not able to keep enough volume of blood in circulation, thus causing imperfect

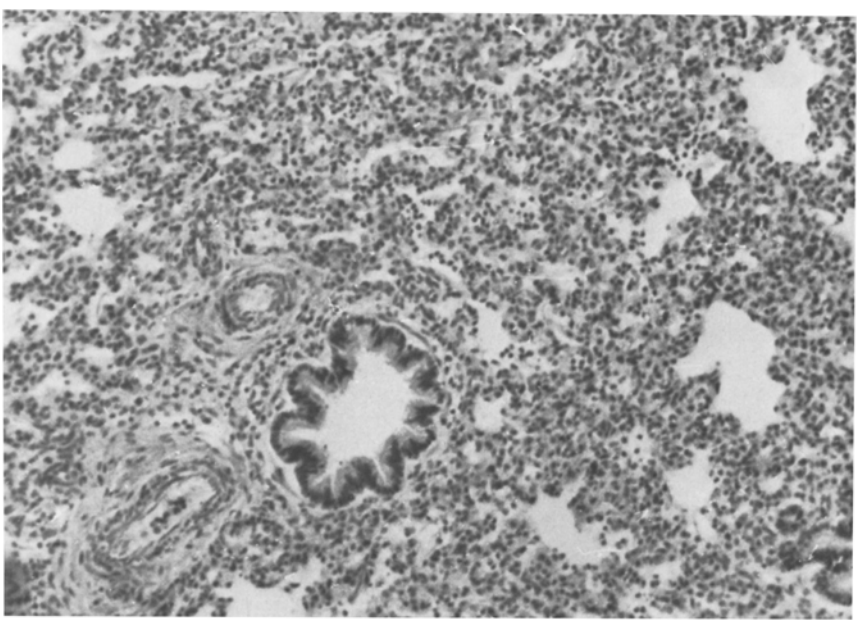

Fig. 1. Mature, live-born infant. "Non aerated" lung

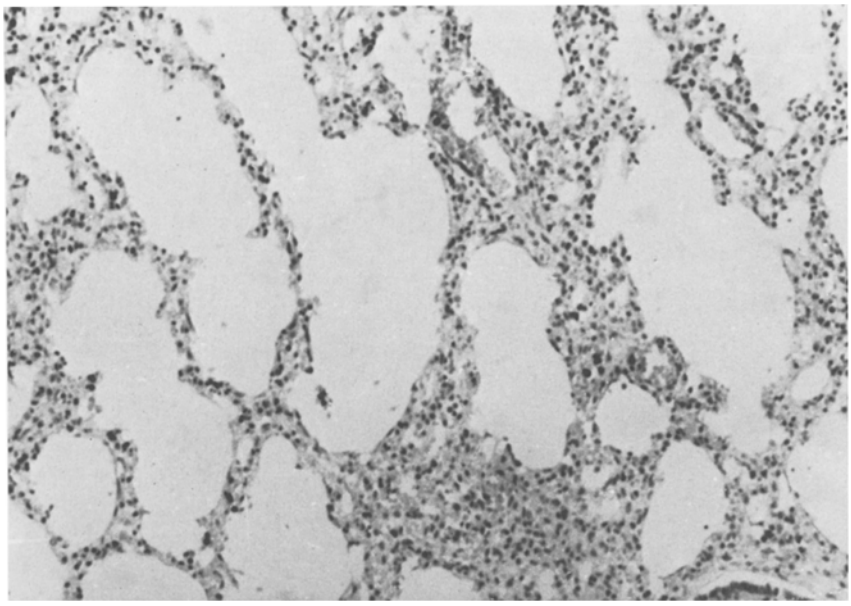

Fig. 2. Artificially respirated infant. Note: acute emphysema

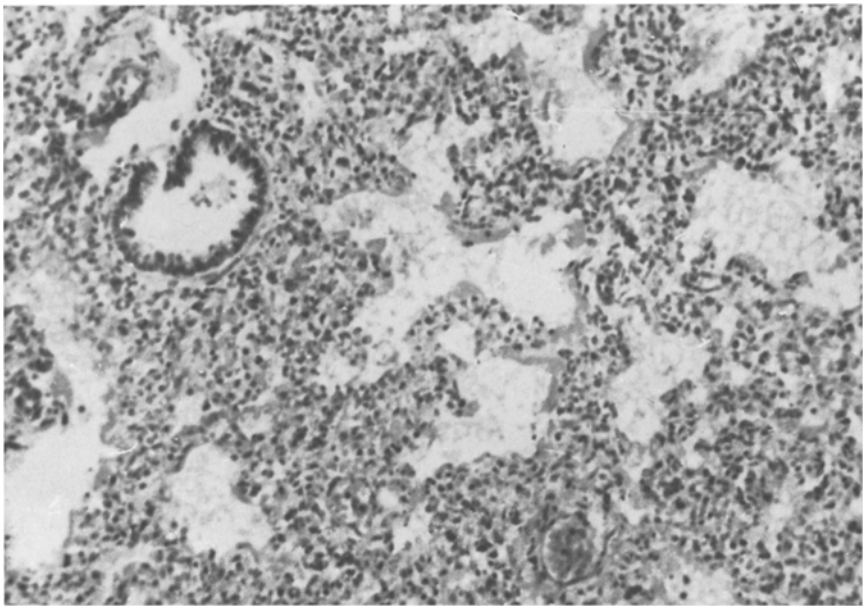

Fig. 3. Hyaline membrane disease. Premature infant

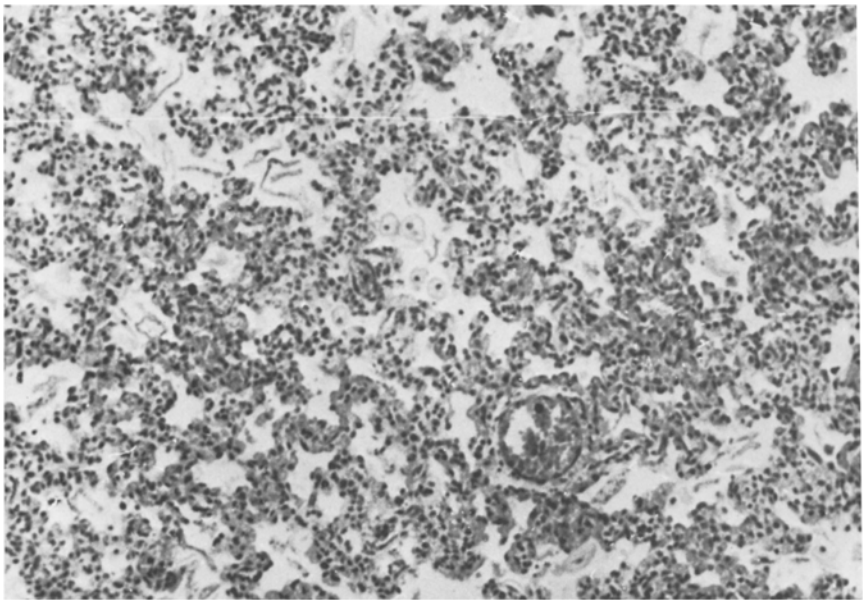

Fig. 4. Epithelial cells, fetal elements in alveoli. Inhalation of amniotic fluid

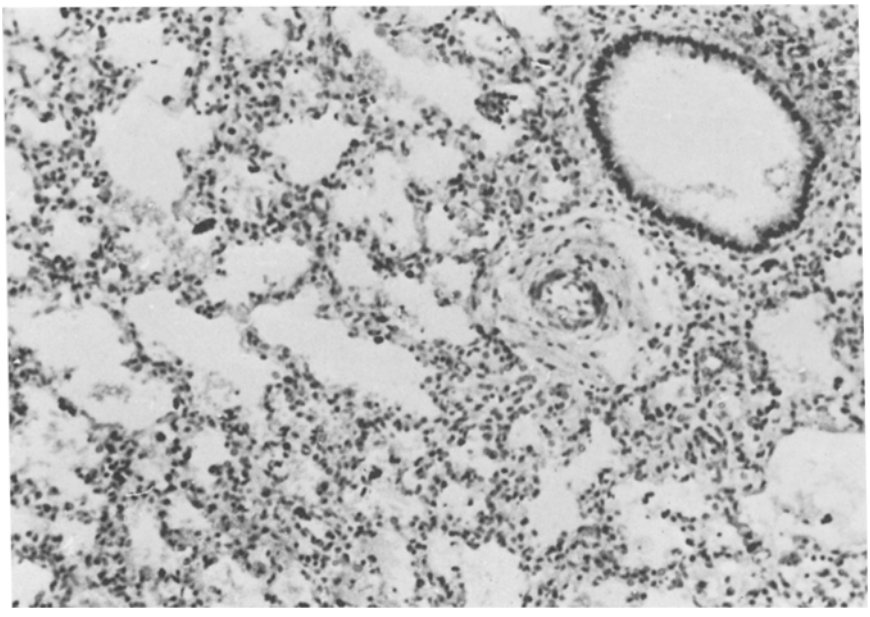

Fig. 5. Stillborn infant; pseudo-aerated lung. Case No. 52

expansion. The absence of the normal expanding mechanism also causes incomplete expansion. The premature infant's lungs are immature and the alveoli are unable to expand, following the first weak respiratory movements. In our autopsy material we found markedly atelectatic lung tissue with unaerated parts histologically in prematures who lived for hours after birth. Four lungs of 28 cadavers were macroscopically and microscopically "unaerated". We explained the secondary atelectasis in the region of the imperfectly aerated lung tissue with agonal air absorption. Hyalin membrane disease occurred in the premature infants more frequently than in the former group (Fig. 3).

In stillborn infants the cause of death was either inhalation of amniotic fluid (Fig. 4), prolapsed umbilical cord, Rh incompability, or malformation etc. The examination of the stillborn infants' lungs motivated our problem; can we justify exactly a life birth by histological processes? We found 6 "aerated" lungs of the examined 28 cases. In the majority of the cases the aerated parts were disposed in separate patches of lungs, but in the cases 48, 50, 52, 53, 54 there were expanded lungs histologically, with positive floating test. Among their anamnestic data were no resuscitating processes, and in the 52th case the infant with a malformation —hydrocephalus—was born dead, after cranioclassis, and its lungs were as aerated as those of a living born infant macroscopically and microscopically alike (Fig. 5).

Our examinations verified that the histological results of the newborn lungs in a significant part of the cases did not match the anamnestic data. There was a major problem in that a large part of the maturated infants' lungs seemed nonaerated. In the formation of the nonaerated lungs we attached great importance to agonal manifestations. From the imperfectly aerated lungs the air is absorbed after stopping of respiration, when the circulation is retained for a short time.

The cases where stillborn infants' lungs "aerated" have most important implications in forensic medical practice. We were able to resolve only with very detailed examinations whether these lungs were aerated or not. The examination of the alveolar epithelium, the congestion of intraalveolar capillaries may resolve the problem, but we have to be concerned about the less experienced examiner who mistakes an unaerated lung as aerated!

However, the knowledge of the anamnestic data, the critical evaluation of the histology, the recognition of the other signs of a live birth seem sufficient to answer the important question: was the newborn living or not!

References

Avery, M. E., Mead, J.: Surface properties in relation to atelectasis and hyaline membrane disease. Am. J. Dis. Child. **97**, 517—532 (1959)

Bonham, E., Carter, R.: The architectural function of pulmonary capillaries. Lancet **1957 I**, 1292—1294

Hirnvonen, J., et al.: Roentgenological and autopsy studies on the gas content of the lungs and gastro-intestinal tract in living and stillborn infants and sources of error in resuscitation. Dtsch. Z. Ges. Gerichtl. Med. **65**, 73—86 (1969)

Lind, J., Wegelius, C.: Proc. 8th International Congress of Pediatrics, Copenhagen 1956; Cit.: Spencer, H.: Pathology of the lung
Prokop, O.: Zur Brauchbarkeit der elastischen Fasern der Lunge im Rahmen der Lebensproben. Z. Rechtsmed. **69**, 177—184 (1971)
Spencer, H.: Pathology of the lung. Oxford: Pergamon Press 1962

Received March 20, 1979