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Pathological Spectrum of the Lung in Cases of Violent Death: Part II. Clinicopathologic Correlation

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ABSTRACT: The correlation between the type of pathological lesion of the lung and the circumstances which encompass their evolution in 66 cases of violent death have been examined.

Pulmonary lesions have been classified into four groups.

1. Inflammatory alveolar lesions without a diffuse interstitial involvement (IAL) which result from direct aggressions in subjects of advanced age.
2. Inflammatory alveolar lesions with a diffuse interstitial affectation (IALW) which are more frequent in younger subjects having a higher defense capacity and with severe lesions requiring admission to an intensive care unit.
3. Edemohemorrhagic lesions (EHL) appearing as a precocious lesion at any age.
4. Unspecific chronic lesions (UCL) previous to the aggression and without any relationship to death.

The most obvious feature noted was the frequent occurrence of certain types of acute pulmonary lesions indicative of the rapid and extensive capacity of the lung to react to a lesion agent even when death follows rapidly after the aggression. The scarcity of acute interstitial lesions among the older group can be related to a diminution of biological defense activity leading to a less vigorous response; on the contrary, diffuse lesions of the wall seem to be related to an excess of defense mechanisms, determined more by age, severity of lesion, and type of medical assistance received than by a specific type of aggression.

KEYWORDS: pathology and biology, death, lungs, lesions, diffuse alveolar damage

Violent death is characterized by the intervention of an unexpected and sudden external agent independent of the natural course of human pathology.

According to Holinger [*1*], it is the third most frequent cause of death in the general population of the United States and the first among the adolescent population of that country.

From the forensic science point of view, three etiologies can be distinguished, namely: accidental, suicidal, and homicidal.

The mechanisms that produce lesions can also be distinguished as follows: traumas of either a physical or mechanical origin, mechanical asphyxia, and intoxication.

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There is a clear predominance of the accidental form of death in a traumatic way, most of them caused by road accidents [2], which constitute a real and increasing preoccupation for the authorities in all developed countries.

The increase in frequency of violent death has been accompanied, throughout the twentieth century, by the equally rapid development of diagnostic and therapeutic regimes in the treatment of such urgent situations.

While there has been a diminution in the incidence of immediate deaths in cataclysmic situations thanks to control of acute hemorrhage, renal failure, and other acute severe situations, this has been matched by the appearance of new complications, of which Adult Respiratory Distress Syndrome stands out [3,4]. We consider the pulmonary parenchyma as a target for this type of complication related to all of the external agents mentioned above: traumas [5–10], asphyxia [11–13] and intoxication [14–18].

In this work, we have correlated in 66 cases of violent death the type of pathological lesions found in the lungs with all the circumstances that characterized and encompassed the evolution of the general organic lesion.

Materials and Methods

We have studied 66 victims of violent death, upon which judicial autopsies were carried out at the Forensic Institute in Zaragoza, Spain.

The distribution by age was quite homogeneous with a slight predominance in the sixth, ninth, and third decades of life; men predominated over women.

The most frequent cause of death was trauma followed by asphyxia and intoxication (Fig. 1). Among traumatic situations traffic accidents predominated (Fig. 2). Submersion was the main cause among asphyxias (Fig. 3), and heroin in cases of intoxication (Fig. 4).

Death was immediate in nearly half of the cases, the other half having been admitted to an intensive care unit (ICU) or to an ordinary ward (Fig. 5). Of those admitted to ICU, there was a predominance of polytraumatisms and cranial and encephalic injuries (Fig. 6).

Having opened the thorax, a specimen measuring 3 by 3 by 3 cm was taken from each pulmonary lobule and prepared by conventional methods for microscopic examination.

Pulmonary lesions were classified into four groups:

(1) Inflammatory alveolar lesions without diffuse interstitial affectation (IAL),
(2) Inflammatory alveolar lesions with diffuse interstitial affectation (IALW),

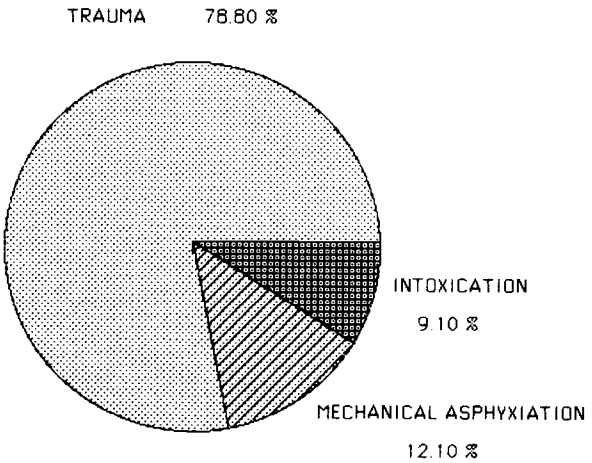

FIG. 1—*Etiology of violent death.*

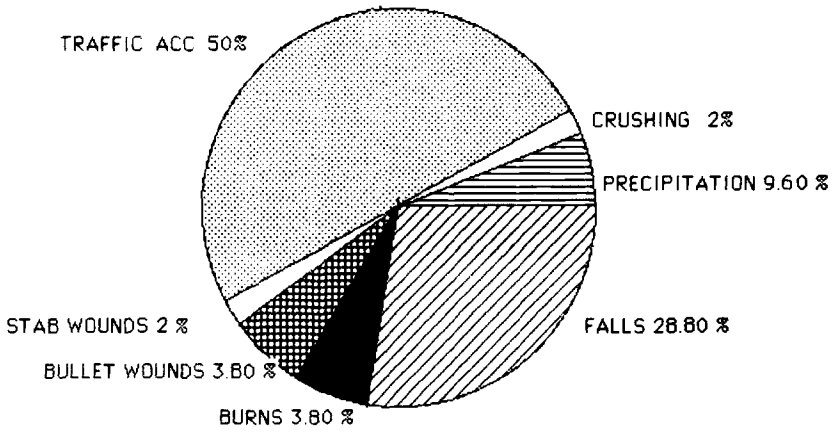

FIG. 2—*Type of trauma over 52 cases.*

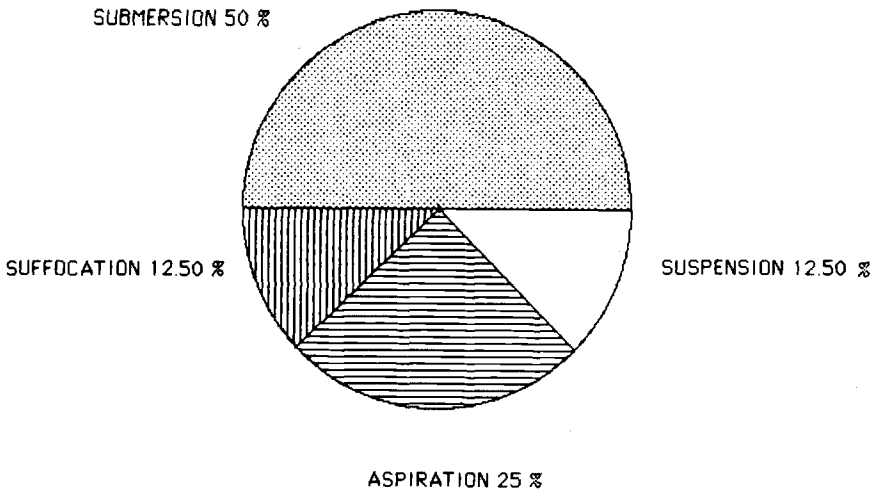

FIG. 3—*Type of mechanical asphyxia over eight cases.*

(3) Edemohemorrhagic lesions (EHL), and
(4) Unspecific chronic lesions (UCL).

We have related those lesions to the circumstances characterizing the evolution by employing the statistical proof *X*.

Results

Of the 66 cases studied, there were only 9 (13.64%) in which we did not find the presence of acute lesions. The rest were distributed in the following way: IAL: 12 cases (18.18%), IALW: 20 cases (30.30%), and EHL: 25 cases (37.87%).

In Table 1 we relate the presence of pulmonary lesions within three age groups. Note that

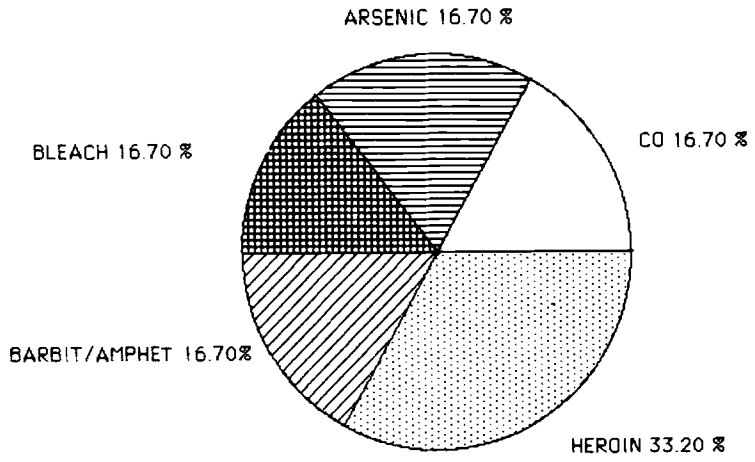

FIG. 4—*Type of intoxication over six cases.*

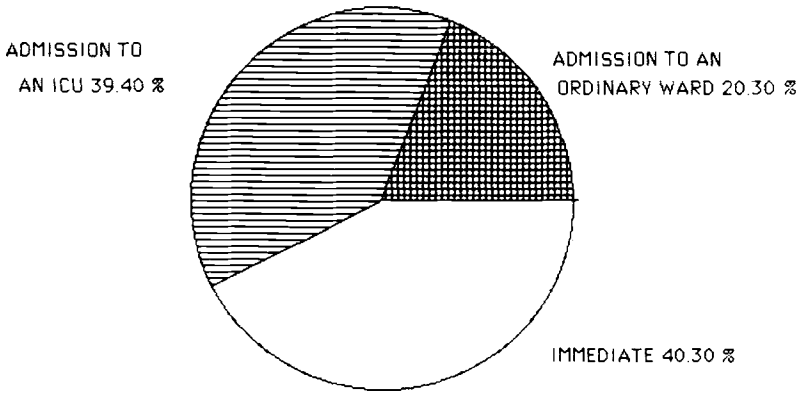

FIG. 5—*Interval between lesion and death and admission place.*

IALW and UCL predominate under 60 years, IAL is more frequent above that age, and EHL is distributed in a homogeneous manner.

In Table 2 we relate the age to the postlesion circumstances: immediate death and hospital admission place (ICU or ward). We found that the highest proportion of those admitted to ICU were under 60, while those admitted to ordinary wards were predominantly over 60. Cases of immediate death were divided in a homogeneous manner between the two groups of age.

In Table 3 we relate the type of pulmonary lesion to the place of admission. Of those admitted to ICU, cases of IALW predominated, while of those admitted to ordinary wards, cases of IAL predominated. In those instances where there was not any admission, EHL predominated.

The circumstances noted in the last two tables necessarily imply a certain degree of abstraction: thus, instances of nonadmission indicate that general lesions were so grave that the patient died before any possibility of specialized medical care; admittance to ICU indicates the presence of a lesion requiring treatment by specialized equipment, while admit-

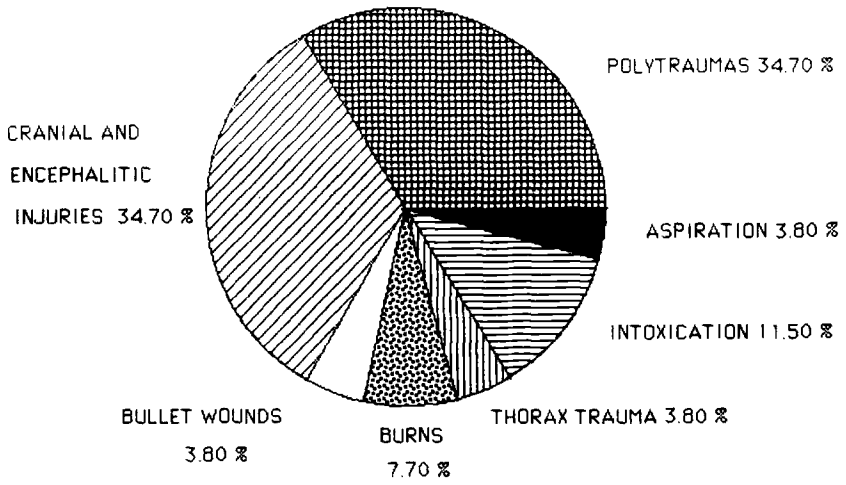

FIG. 6—*Type of lesions in the patients admitted to an ICU.*

TABLE 1—*Correlation between pulmonary lesions and age groups.*[a]

	Age Groups			
Lesions	0 to 29 Years	30 to 59 Years	60 to 99 Years	Totals
	0.87[b]	0.87	2.37	
IALW	8[c]	8	4	20
	5.76[d]	5.76	8.48	
	0.09	3.45	1.66	
IAL	4	0	8	12
	3.45	3.45	5.09	
	0.01	0.09	0.04	
EHL	7	8	10	25
	7.20	7.20	10.61	
	2.59	0.06	1.24	
UCL	0	3	6	9
	2.59	2.59	3.82	
Totals	19	19	28	66

[a] $X^2 = 13.34$. 6 degrees of freedom. $p = 0.05$.
[b] Top value is expected frequency.
[c] Middle number is observed frequency.
[d] Bottom value is partial contribution to the statistical value X^2.

tance to an ordinary ward suggests that the treatment regime was pharmacological and supervisory only.

In Table 4 we attempt to relate the type of pulmonary lesion to the type of aggression, but this table is not statistically significant with the proof *X* and it must be stated that to this number of cases the pathological lung results cannot be correlated with the type of violent death.

Discussion

Upon analysis of the results, the most obvious feature noted was the frequent occurrence of certain types of acute pulmonary lesions. This is indicative of the rapid and extensive

TABLE 2—*Correlation between postlesion circumstances and age groups.*[a]

Postlesion Circumstance	Age Groups			Totals
	0 to 29 Years	30 to 59 Years	60 to 99 Years	
	0.85[b]	0.57	2.02	
ICU	10	10	6	26
	7.48	7.88	10.64	
	2.01	2.19	6.06	
Ward	1	1	11	13
	3.74	3.94	5.32	
	0	0.08	0.10	
No admittance	8	9	10	27
	7.77	8.18	11.05	
Totals	18	20	27	66

[a] $X^2 = 13.88$. 4 degrees of freedom. $p = 0.01$.
[b] See footnotes in Table 1.

TABLE 3—*Correlation between pulmonary lesions and postlesion circumstances.*[a]

Lesions	Postlesion Circumstances			Totals
	ICU	Ward	No Admittance	
	8.37[b]	0.96	4.67	
IALW	16	2	2	20
	7.88	3.94	8.18	
	1.58	5.61	0.17	
IAL	2	6	4	12
	4.73	2.36	4.91	
	3.47	0.17	4.48	
EHL	4	4	17	25
	9.85	4.92	10.23	
	0.06	0.33	0.03	
UCL	4	1	4	9
	3.55	1.77	3.68	
Totals	26	13	27	66

[a] $X^2 = 29.90$. 6 degrees of freedom. $p = 0.005$.
[b] See footnotes in Table 1.

capacity of the pulmonary parenchyma to react when faced with a violent exogenous agent, this being so even in those cases where the lung has not been directly affected and where death quickly followed the aggression. Asbaugh et al. [*3*] pointed out this phenomenon of pulmonary reactivity.

In the group not requiring admission, death followed rapidly in relation to lesion. These deaths took place either at the scene of the accident or during the journey to hospital. EHL predominates in this group being the quickest form of pulmonary response. However, two cases of IALW and four cases of IAL demonstrate that the pulmonary parenchyma is also capable of response within just a few minutes by the transmission of a significant volume of inflammatory cellular elements to the interstice and to the alveolar lumen, especially when facing direct aggressions, traumatic or asphyxial.

Those admitted to ICU presented severe lesions, especially of a traumatic type, and IALW predominated in these cases. As stated by Blaisdell and Scholobohm [*19*], grave traumatic situations, with polytraumatism and affectation of the central nervous system or ischemic

TABLE 4—*Correlation between type of aggression and pulmonary lesion.*[a]

Type of Aggression	Lesions			Totals
	IALW	IAL	EHL	
	0.08[b]	0.1	0	
Trauma	14	10	19	43
	15.09	9.05	18.86	
	0.23	0.28	0.63	
Asphyxiation	2	1	5	8
	2.81	1.68	3.51	
	1.69	0.05	1.01	
Intoxication	4	1	1	6
	2.11	1.26	2.63	
Totals	20	12	25	57

[a] $X^2 = 4.08$. 4 degrees of freedom. Not statistically significant.
[b] See footnotes in Table 1.

lesions produced by shock, are associated in those cases admitted to ICU with the pathogenous effect that ventilators, oxygen, and the accumulation of liquids have upon the lungs.

Of those admitted to ordinary wards we noted a tendency to develop lesions of a pneumonic type. They fell predominantly within the older age group, and they had a tendency to die of complications to a trauma which was not in itself serious enough to produce death.

We can thus establish a classification of pulmonary response to aggression with three variables:

- EHL which appears as a precocious reaction at any age.
- Lesions predominantly affecting the alveolar lumen, the appearance of which, in the face of either direct or indirect aggressions, occurs more frequently in older people.
- Lesions of the whole alveolocapillary wall that appear more frequently in younger subjects who have a higher capacity for defense and who have suffered serious lesions requiring admission to ICU. The relative scarcity of this type of lesion in the more advanced age group could be interpreted as an expression of the diminution of organic reactivity in this group when faced with aggression, thus permitting a less vigorous response.

Thus, the diffuse lesion of the alveolocapillary wall comparable to the profile defined by Katzenstein [*20*] as diffuse alveolar damage, seems to be a response to an excess of the defense mechanisms. In this respect, several studies have emphasized the role of the defensive cells of the alveoli, neutrophils, and macrophages in the pathogenia of the lesion [*21,22*]. This response seems to be determined more by age, severity of lesion, and type of medical assistance received than by specific type of aggression.

References

[*1*] Holinger, P. C., "Violent Deaths Among the Young: Recent Trends in Suicide, Homicide and Accidents," *American Journal of Psychiatry*, Vol. 136, No. 9, 1979, pp. 1144-1147.
[*2*] Council on Scientific Affaires, "Automobile-Related Injuries. Components, Trends, Prevention," *Journal of the American Medical Association*, Vol. 249, No. 23, 1983, pp. 3216-3222.
[*3*] Asbaugh, D. and Petty, T., "Sepsis Complicating the ARDS," *Surgery, Gynecology and Obstetrics*, Vol. 135, No. 6, 1972, pp. 865-869.
[*4*] Baue, A. E., "The Pushmi-Pullyn Syndrome," *Surgery*, Vol. 72, No. 4, 1972, pp. 655-656.
[*5*] Moseley and Dotty, "Hypoxemia During the First 12 Hours After Battle Injury," *Surgery*, Vol. 67, No. 5, 1970, pp, 765-772.
[*6*] Collins, J. A., James, P. M., Bredenberg, C. E., Anderson, R. N., Heisterkamp, Ch. A., and

Simmons, R. L., "The Relationship Between Transfusion and Hypoxemia in Combat Casualties," *Annals of Surgery*, Vol. 188, No. 4, 1978, pp. 513-520.

[7] Carmona, R. H., Lim, R. C., and Clark, G. C., "Morbidity and Mortality in Hepatic Trauma. A 5 Year Study," *American Journal of Surgery*, Vol. 144, No. 1, 1982, pp. 88-94.

[8] Santatecla, V., Verdejo, J., and Montero, R., "Alteraciones de la Fisiopatologia Pulmonar en la Reanimación de los Traumatismos Abdominales," *Revista Española de Anestesia y Reanimación*, Vol. 31, No. 2, 1984, pp. 55-58.

[9] Luisada, A. A., "Mechanisms of Neurogenic Pulmonary Edema," *American Journal of Cardiology*, Vol. 20, No. 1, 1967, pp. 66-68.

[10] Hirsh, E., Clarke, J. R., Gomez, H. E., and Clowes, G. H. A., "The Lung: Responses to Trauma, Surgery and Sepsis," *Surgical Clinics of North America*, Vol. 56, No. 4, 1976, pp. 909-928.

[11] Overland, E. S. and Severinhaus, J. W., "Noncardiac Pulmonary Edema," *Advances in Internal Medicine*, Vol. 23, 1978, pp. 307-326.

[12] Hudson, L., "Causes of ARDS. Clinical Recognition," *Clinics in Chest Medicine*, Vol. 3, No. 1, 1982, pp. 195-212.

[13] Cahill, J. M., "Ahogamiento: el Problema de la Sumersion no Mortal y del Paciente sin Conocimiento," *Clinicas Quirurgicas de Norte America*, Vol. 48, 1968, pp. 423-430.

[14] Louria, D. B., Hensle, T., and Rose, J., "The Major Medical Complications of Heroin Addiction," *Annals of Internal Medicine*, Vol. 67, No. 1, 1967, pp. 1-22.

[15] Duberstein, J. L. and Kaufman, D. M., "A Clinical Study of an Epidemic of Heroin Intoxication and Heroin Induced Pulmonary Edema," *American Journal of Medicine*, Vol. 51, No. 6, 1971, pp. 701-714.

[16] Frand, U. I., Shim, Ch. S., and Williams, H., "Methadone-Induced Pulmonary Edema," *Annals of Internal Medicine*, Vol. 76, No. 6, 1972, pp. 975-979.

[17] Allred, R. J. and Ewer, S., "Fatal Pulmonary Edema Following Intravenous "Freebase" Cocaine Use," *Annals of Emergency Medicine*, Vol. 10, No. 8, 1981, pp. 441-442.

[18] Frejaville, J. P., "Oedeme Pulmonaire Non-hemodinamique des Toxiques," *Annales d'Anasthesie Française*, Special II et III, 1975, pp. 67-77.

[19] Blaisdell, W. and Scholobom, R., "The Respiratory Distress Syndrome: A Review," *Surgery*, Vol. 74, No. 2, 1973, pp. 251-262.

[20] Katzenstein, A. L. and Askin, F. B., *Surgical Pathology of Non-Neoplastic Lung Disease*, 1st ed., Saunders Co., Philadelphia, 1982.

[21] Johnson, K. J., Chapman, W. E., and Ward, P. A., "Immunopathology of the Lung. A Review," *American Journal of Pathology*, Vol. 95, No. 3, 1979, pp. 725-845.

[22] Reynolds, H. Y., "Lung Inflammation: Role of Endogenous Chemotactic Factors in Attracting Polimorphonuclears Granulocytes," *American Review of Respiratory Disease*, Vol. 127, Supplement, 1983, pp. 16-25.

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