Virchows Archiv A Pathol Anat (1989) 414:415–422

Virchows Archiv A
Pathological Anatomy and Histopathology

Mechanism of death in avalanche victims

Helge Stalsberg[1], Claus Albretsen[1], Mads Gilbert[2], Michael Kearney[1], Einar Moestue[1], Ivar Nordrum[1], Morten Rostrup[3], and Anne Ørbo[1]

Departments of [1] Pathology and [2] Anesthesiology, N-9012 Tromsø University Hospital, Tromsø, Norway
[3] Medical Department, Ullevål Hospital, N-0407 Oslo 4, Norway

Summary. The autopsies of 12 victims from two snow avalanches in North-Norway are reported. Supportive evidence from non-autopsied and surviving victims is included. Consistent autopsy findings were prominent lung oedema, moderate cerebral oedema, extreme contraction of the left ventricle, petechiae in the superior vena cava drainage area, and acute congestion in lungs and kidneys. In four cases in whom no resuscitation was attempted, aortic oxygen pressure was in the range expected in pure asphyxial-type deaths in one and in pure cardiac-type deaths in three. No air pocket was seen in front of the mouth and nose in any of the fatal cases. Three fatal cases had fractures. It is concluded that the immediate cause of death in most cases was general body compression with acute respiratory and circulatory failure.

Key words: Avalanche – Asphyxia – Autopsy – Accidents – Traumatology

Introduction

When a snow avalanche stops, a completely buried victim is assumed to have an 80% average chance of survival if uncovered immediately. For each succeeding hour of burial, the chance of survival is reduced by one half, to 40% after one hour, 20% after two hours and so on (Schild 1976).

Although several different mechanisms of death may be considered, including mechanical trauma, hypothermia, and suffocation, and although these are of importance both to the surviving relatives and for the consideration of rescue and resuscitation procedures, little has been published on autopsy findings in avalanche victims, particularly in the English literature. We report here our experience from the autopsy of 12 victims from two avalanches in Vassdalen and Nord-Lenangen in Troms county in North-Norway. Examination included aortic oxygen tension, which has not been reported previously in avalanche victims.

Observations on the survivors, rescue operations and resuscitation procedures in the Vassdalen accident will be reported separately and are included here only to the extent that they are relevant to the consideration of what happened to those who died.

In Vassdalen on March 5, 1986, a troop of 31 soldiers were preparing a track for belt wagons up the bottom of a small valley at the foot of a 600 m mountain when they were hit by an avalanche. The whole troop and their two belt wagons were carried up to 100–150 m down the valley with the snow masses. The weather was bad with strong winds, falling snow and air temperatures around −10° C. The soldiers noticed no warning signs before they were hit. One soldier was not buried by the avalanche, 13 were partially buried, and 17 were completely buried in the snow. Six of those partially buried were able to free themselves from the snow rapidly. While two ran for assistance, the rest managed to bring out the other seven who were only partially buried, all of whom survived.

When the first rescue team arrived about one hour after the accident, 17 soldiers were still missing. The last survivor was brought out three hours after the avalanche impact. The remaining 16 were found without evidence of life after 80 min to 74 h under the snow.

Investigations showed that the avalanche had started as a dry-snow slab avalanche at 470 m above sea level where the snow density was about 400 kg/m^3. The avalanche hit the first victims at 270 m elevation with a speed of about 110 km/h

Offprint requests to: H. Stalsberg

and stopped at 215 m elevation (Haug et al. 1986). There were no large trees in the area where the victims were taken, and no ice or stone blocks were noted in the avalanche masses. When the rescue personnel arrived, the snow was extremely hard packed and hardly penetrable by manual digging.

In Nord-Lenangen on 29 March 1987, two boys 12 and 13 years of age were hit by a 300 m wide slab avalanche while playing with their sledges on a wooden hillside. The slab was 2 m high at the most where it broke loose, probably less than 50 m above the victims. The avalanche seemed to have carried the victims about 100 m down the hillside, where they were recovered in a wooded area. The victims were reported missing four hours after the assumed time of impact, and their bodies were recovered with no signs of life two and three hours later.

Methods

In Vassdalen, two of the authors participated in the rescue operations on the spot as batallion medical officer (MR) and chief of medical operations (MG), respectively. Further information on rescue operations was obtained one month after the accident by a questionnaire mailed to and answered by all 12 physicians participating on the spot. The helicopter pilots who evacuated the victims from the avalanche area also answered the questionnaire. Medical information was also obtained from the record of the military and general hospitals to which the victims were taken, and from the psychiatric team which treated the surviving victims. Further information was obtained from the local police report and from the report of a governmental commission of inquiry (Haug et al. 1986). In Nord-Lenangen, one of the authors (MG) participated in the rescue operation on the spot, and additional information was obtained from the local police. In six of the Vassdalen victims, the relatives objected to autopsy, and medical examination was restricted to external examination, performed by the city medical officer of Narvik, Dr. Harald Andreassen. Eight of the Vassdalen victims were autopsied 45–53 h after the accident, and the last two after 92 h. The Nord-Lenangen victims were autopsied 41 h after the accident. A standard protocol was followed in all cases, including the withdrawal of blood for gas analysis from the aortic arch under standard anaerobic conditions, and opening of the thorax under water to test for the presence of pneumothorax.

For the comparison of lung weight in fatal hypothermia, eight consecutive cases of death from general hypothermia (five men and three women, age 22–70, mean 48 years) were drawn from the autopsy files of 1977–1982. For the comparison of microscopic evidence of acute congestion in parenchymatous organs, 21 consecutive forensic cases were drawn from the autopsy files of 1986, excluding deaths from heart disease and cases in which major external or internal haemorrhage had preceded death (15 men and six women, age 22–70, mean 40.7 years). Histological slides from lungs, liver, kidney, and myocardium from avalanche victims and the consecutive forensic cases were studied after the slides had been randomly mixed and the identification labels covered. Evidence of congestion was recorded as absent, moderate, or marked.

Mean weights were compared by two-tailed t-test and presented as mean $\pm$ standard deviation. P-values < 0.05 were considered significant.

Results and clinical findings

A summary of observations in the 12 autopsied victims is given in Table 1.

The 17 fully buried victims in Vassdalen were found at depths of 1–3 m. Most were in horizontal position, face up or face down. Four were in more distorted positions, and two had one arm extended into the snow. One of the non-autopsied victims was found in a vertical position, head down in a stream with only his feet above water. The only one of the completely buried victims who survived was found after three hours at a depth of 1.5 m. He had his back against a belt wagon and his right arm in front of his face, around which an air pocket had formed. As far as it could be observed under the prevailing conditions and ascertained by the methods used, no air pocket was present in any of the others. An ice shield was noted over the nose and mouth of most of the victims. All victims wore warm winter clothing. Food remnants in the mouth or signs of aspiration were noted in eight.

In Nord-Lenangen, one victim was partially buried and the other was found at a depth of 1 m. In the former, gastric contents were present in the mouth, pharynx and trachea.

In Vassdalen, cardiopulmonary resuscitation (CPR) was started in the first nine victims recovered without vital signs. Resuscitation in the field and during transportation was performed or supervised by experienced physicians and included external chest compression and mouth-to-mouth or bag-to-mouth ventilation. Following hospital admission, rewarming was performed by peritoneal lavage in case 7 and extracorporeal circulation in case 1. In case 7, large amounts of fluid were drained from the endotracheal tube. CPR was unnecessary in the survivors. Due to extreme, deteriorating weather conditions, the victims recovered during the evening and night following the avalanche could not be evacuated, and CPR was not initiated. In Nord-Lenangen, CPR with endotracheal intubation and sporadic chest compressions was started on site by experienced medical personnel. CPR was continued during transport to the University Hospital and was then stopped after clinical assessment. In no case did the resuscitation efforts lead to any spontaneous heart or respiratory activity.

The survivor who was brought out after having been completely buried for three hours was con-

Table 1. Autopsied avalanche victims

Case	Age years	Time from accident to autopsy (h)	Burial time (h)	Burial depth (m)	Resuscitation attempted	Mechanical injury	Lung weight (g)	Brain weight (g)	Aortic pO_2 kPa (mm Hg)
1	29	45	5	NR	+	–	1700	1770	1.0 (8)*
2	19	48	4.5	2–3	+	Parietal skin laceration	1430	1530	3.8 (29)
3	19	48	18	NR	–	–	1700	1775	8.1 (61)
4	20	49	2.3	2	+	Rib fracture Haemothorax	1050**	1500	NR
5	24	50	21	2	–	–	1200	1420	4.7 (35)
6	20	51	2.3	1.5	+	–	1205	1620	1.1 (8)
7	20	53	2.5	2	+	–	940	1450	6.4 (48)
8	19	53	3.5	1.5	+	–	1800	1560	1.7 (13)
9	21	92	74	1	–	–	1580	1490	5.2 (39)
10	20	92	73	3	–	–	1980	1530	0.4 (3)
11	12	41	6	0.5	+	Skull fracture	630	1220	3.7 (28)
12	13	41	7	1	+	Leg fracture	1200	1500	1.6 (12)

* Measured before treatment on heart-lung machine. At autopsy, pO_2 was 7.6 kPa (57 mm Hg); ** Right lung 800 g, left lung 250 g; NR, not recorded

scious, but confused and agitated. On admission to hospital 15 min later, rectal temperature was 32° C, arterial pO_2 6.7 kPa (50 mm Hg) and pH 7.09. Chest X-ray showed a right lung infiltrate considered to represent lung contusion. One other survivor had haemoptysis, radiographic evidence of lung contusion and arterial pO_2 7.4 kPa (56 mm Hg) on admission. Other mechanical trauma was pneumothorax in one, knee ligament injury in one, fracture of the humerus in one, and fracture of the leg in two, one of which also had a facial fracture. There was no record of tympanic membrane rupture or other lesions to the ears. Most of the survivors felt snow enter their mouth, nose and ears. Ten had difficulty in breathing because of snow in the nose and pharynx. Compression of the thorax was felt by six, and possibly by a further three. One compared his feelings to lying under 40 mattresses. Most reported a loss of sense of time and space. Four lost consciousness, and six more may have lost consciousness, but the information is less certain in these cases. The survivor who was buried for three hours had total amnesia from the moment when the avalanche hit until he woke up in the hospital.

Two of the six dead on whom external examination only was performed, had superficial skin abrasions in the face. No fractures or other significant mechanical trauma were identified in any of these.

In general, the findings at autopsy in the 10 Vassdalen cases were strikingly similar. The main exception was case 4, who had fractures of left ribs 8–10 with a tear of the lung and 1500 ml of blood in the left pleural cavity. Case 2 had a stellate skin laceration in the parietal region of the head, but no fracture. In the remaining eight, no fracture or other mechanical trauma except superficial skin abrasions were identified.

Both Nord-Lenangen cases had fractures. Case 11 had a linear fracture through the left anterior cranial fossa without dislocation or significant haemorrhage, and case 12 a fracture of the right leg.

Postmortem lividity was pronounced in the head and chest region in 10 cases, frequently with petechial haemorrhages. In one, facial congestion was so marked that a basal skull fracture was at first suspected. On the trunk, areas of hypostasis were in most cases distinctly patterned after the fabric of the underwear.

A moderate number of petechial haemorrhages in mucous and serous membranes were seen in all cases, distributed to the conjunctiva in 6, pleura in 11, pericardium in 10, and tracheal mucosa in 3. The brain was moderately oedematous in all cases. A froth was present in the trachea and bronchi in cases 1, 2, 3, 8, 10 and 12. Aspiration of food to peripheral bronchi was seen in case 7. The lungs were dark and heavy with oedema fluid oozing from the cut surface in all cases. In case 4, in whom a left-sided haemothorax was present, this picture was seen in the right lung, whereas the left lung was collapsed. There was no pneumothorax in the autopsy cases. The left ventricle was very hard and maximally contracted in all cases. In con-

Table 2. Histologic evidence of acute congestion in avalanche victims compared to consecutive forensic autopsies excluding deaths from cardiac disease and cases with major haemorrhage

Organ	Microscopic congestion	Avalanche victims	Consecutive forensic autopsies	P*
Lung	None	0	3	
	Moderate	1	12	<0.002
	Marked	11	6	
Liver	None	4	15	
	Moderate	6	4	0.051
	Marked	2	1	
Kidney	None	0	9	
	Moderate	5	8	0.055
	Marked	7	4	
Heart	None	5	11	
	Moderate	4	7	0.82
	Marked	3	3	

* χ^2-test for 2 × 2 tables with Yates' correction. Because of small numbers, the 'moderate' group was combined with the smallest other group for each organ for statistical analysis

trast, the right ventricle was more relaxed and dilated. Other organs were normal on gross inspection.

Microscopic examination of the lungs showed acute congestion, alveolar oedema and focal alveolar haemorrhage in all cases. Microscopic evidence of aspiration was present in case 7. Acute congestion was also seen in the kidneys in all cases. In slides from the myocardium stained with Luxol Fast Blue, contraction band necrosis was demonstrated in six (cases 1, 4, 6, 7, 9 and 10), including two in whom no resuscitation had been attempted.

Uncoagulated aortic blood could be withdrawn from all except case 4. The pO_2 varied from 1.1 to 7.6 kPa (8 to 57 mm Hg) in the cases in whom resuscitation had been attempted, and from 0.4 to 8.1 kPa (3 to 61 mm Hg) in the non-resuscitated cases.

Mean lung weight was significantly higher in the adult avalanche victims (1459 ± 348 g) than in the cases of death from general hypothermia (940 ± 251 g) drawn from the autopsy files (p = 0.003).

Microscopic evidence of acute congestion is more prominent in the avalanche victims than among the consecutive forensic cases (Table 2). The difference is most marked and highly significant in the lung and of borderline significance in the liver and kidney. No significant difference is seen in the heart. Brain sections were only available in selected cases in the consecutive forensic series and therefore not suitable for statistical comparison.

Discussion

Severe mechanical trauma other than that resulting from general compression of the body was present in both of the Nord-Lenangen victims but in only one of the 16 who died in Vassdalen. The difference may be due to the presence of more trees in the Nord-Lenangen avalanche area against which the victims were thrown. Severe pulmonary congestion with alveolar oedema and haemorrhage was present in all cases. The mechanical trauma may be considered as a contributory, but not necessarily main cause of death in cases 4 and 11. The circulatory disturbances discussed below may have aggravated the effect of the trauma by causing more profuse bleeding than would have resulted from the injuries alone. In Vassdalen, mechanical injury was strikingly more common among the survivors than among the dead, probably because the survivors were more exposed to moving forces in the uppermost layers of the avalanche.

Hypothermia seems less likely to have played any role as a cause of death in the present cases. All victims wore warm winter clothing, and snow also has considerable insulating properties. The victim who survived three hours of snow burial had a rectal temperature of 32° C on hospital admission. Presumably, it would not take many minutes of adequate breathing to melt enough snow to create an air pocket in front of the mouth and nose. Since no air pocket was seen in any of the dead victims, they cannot have breathed for long enough to have died from gradual general hypothermia. Heavy, oedematous lungs, which was a marked finding in all cases, was not typically found in the cases of death from general hypothermia drawn from the autopsy files for comparison.

Possible mechanisms of suffocation in avalanche victims are (Markwalder 1976) aspiration of snow aerosol, resulting in either laryngeal spasm or drowning; aspiration of vomited stomach contents; external compression of the thorax which makes inspiratory movements impossible; or lack of oxygen in the inspired air. If aspiration of snow aerosol were an important factor, we would have expected this to have affected the partially buried victims. Many of these said that they had to spit out snow from the mouth, but we have no record of this causing laryngeal spasm or obstruction of the lower airways. Doubts have been expressed about the possible mechanism of drowning by in-

halation of powder snow (Markwalder 1976). The density of the aerosol is supposed to be between 2 and 10 kg/m^3 (de Quervain 1976) which is probably too scanty to bring enough water into the air passages and cause drowning.

Food remnants in the mouth and possible signs of aspiration were recorded in nine victims during rescue operations. Seven of these were autopsied, and only in one was aspiration to peripheral bronchi demonstrated. The findings may be due to passive postmortem regurgitation of stomach contents, or more likely an effect of external abdominal compression as it was so commonly seen. Inhibition of inspiratory movements by thoracic compression may have prevented the stomach contents from entering the lower air passages.

We consider external compression of the thorax to have been the most important cause of suffocation. The lack of recognizable air pockets in all cases indicates that virtually no effective respiration was maintained following burial. The distinct patterning of the postmortem hypostasis with imprints of the fabric of the underwear indicates that the snow had exerted a firm generalized pressure against the body. The very firmly packed snow, likened to concrete by the rescue personnel, also fits with this concept. A documentation of this mechanism of suffocation was recently provided as a case report in which a 32 year old woman survived complete burial in a snow avalanche. When only the head was uncovered, mouth to mouth resuscitation was not possible owing to the snow surrounding her chest. However, when the chest was uncovered, ventilation was easy (Gray 1987). It seems significant that the victim in our material who survived three hours of complete burial had his back against one of the belt wagons. This apparently shielded him from having the densely packed snow completely surrounding his chest. Combined with the fact that he held one arm before his face, this allowed him to breathe and to develop a distinct air pocket.

Avalanche snow consolidates during the first hour after impact (de Quervain 1976). Thus some of the victims were able to free themselves and their partially buried comrades immediately after impact, whereas the snow was hardly penetrable to manual digging when the rescue personnel arrived.

Some gas exchange is possible even through densely packed snow, but is greatly dependent on the size of the air pocket and is reduced by icing of its walls. Holding hands or arms before the face substantially improves the situation by increasing the interface between the air pocket and the snow and by reducing icing (de Quervain 1976). In our three-hour survivor, possible air pockets in and around the belt wagon may also have helped.

The ice-shield over the nose and mouth of most of the Vassdalen victims was probably due to extrusion of lung oedema or stomach contents. Breathing would have been expected to produce an air pocket rather than an ice shield in front of the mouth and nose.

Postmortem blood gas analysis is seldom reported in autopsy material except for measurement of carbon monoxide in cases of suspected carbon monoxide poisoning. Whereas acidity and partial carbon dioxide pressure of the blood always increase post mortem, irrespective of the cause of death, oxygen tension in aortic blood may reflect the oxygenation of the blood when circulation ceased reasonably well. Coe (1977) reported a study in which arterial pO_2 measurements were performed at autopsy in 11 respirator patients who died when the respirator was shut off, and in eight patients who died from irreversible circulatory arrest while ventilation was artificially maintained. The interval between death and autopsy was 1.5 to 22 h, and there was no systematic relationship between arterial pO_2 and the time after death. The measurements showed pO_2 of 0.4 to 1.6 kPa (3 to 12 mm Hg) in the asphyxial-type deaths and 4.7 to 10.9 kPa (35 to 82 mm Hg) in the cardiac-type deaths. The findings were consistent with previous experimental data (Mithoefer et al. 1967).

Although as much as 41–92 h elapsed from death to autopsy in our cases, the results of the oxygen measurements in themselves lend some support to their validity. The finding of pO_2 1.0 kPa (8 mm Hg) before treatment on heart-lung machine in case 1 confirms that pO_2 values in this low range occurred during the early postmortem period, and the value of 7.6 kPa (57 mm Hg) at autopsy 40 h later indicates that the oxygenation produced by the treatment on the heart-lung machine was preserved. The different findings of pO_2 0.4 kPa and 5.2 kPa in the two last recovered Vassdalen victims also indicate that little or no equilibration of the oxygen pressure had taken place before autopsy. Presumably, the rapid cooling of the bodies contributed significantly to prolonged preservation of blood oxygen tension. Compared to the findings of Coe (1977), four of our cases in whom resuscitation was attempted and one in whom resuscitation was not attempted had pO_2 values in the range of the pure asphyxial-type deaths. One of the resuscitated and three of the non-resuscitated avalanche victims had pO_2 values in the range of the pure cardiac-type deaths. Two

cases had intermediate values. Resuscitation had no obvious and systematic effect on the oxygen values, except in case 1, who was rewarmed by cardiopulmonary by-pass on a heart-lung machine. On the basis of these data, it seems that the five cases with the lowest oxygen pressures may have died from acute respiratory insufficiency with hypoxic hypoxaemia and hypercapnoea. In the remaining six cases in which measurements were obtained, circulation seems to have stopped and death ensued before the oxygen pressure had become critically low, at least in the three of these in whom no resuscitation had been attempted. The lack of air pockets indicates that no effective respiration had taken place, suggesting that death occurred within the first few minutes after burial. A protective diving reflex, stimulated by cooling of the face and resulting in bradycardia and reduced oxygen consumption, may have resulted in a more protracted course, but this is speculative (Harries 1986).

All cases had prominent signs of circulatory disturbances with pulmonary oedema, pulmonary haemorrhages, microscopic evidence of acute congestion in lungs, liver and kidney, a moderate cerebral oedema and firmly contracted left ventricle and dilated right ventricle. The petechial haemorrhages in the superior vena cava drainage area may have been of either hypoxic or circulatory origin. The mechanism of circulatory disturbances and failure is complex (Schmid 1981). Hypoxia results in contraction of pulmonary arterioles and leads to pulmonary hypertension and lung oedema. Cooling of the body surface leads to peripheral vascular contraction and increased arterial resistance. According to Starlings law, the general compression of the body by the pressure of the snow from all sides leads to increased tissue pressure, resulting in a net transfer of fluid to the blood. Sympathetic stimulation may give splanchnic vessel contraction. Arrest of respiratory movements abolishes the haemodynamic pump effect of the lungs. The only possible place in the body where the pressure is not increased due to the general external pressure, is within the lung alveoli and the air passages, where atmospheric pressure will prevail. Presumably, all of these effects lead to a centralization of the blood volume and a high pressure gradient over the pulmonary capillary-alveolar membrane with resulting acute alveolar oedema and haemorrhage. The finding of as much as 1500 ml of blood in the pleural cavity seems to us to be a somewhat unusual result of a costal fracture and superficial tear of the lung and may be another expression of centralization of blood volume and increased intravascular pressure.

The cranial contents are also protected from direct pressure from the surrounding snow, and as the general increase in pressure of body fluids is transferred via the blood vessels, increased transcapillary pressure gradient leads to cerebral oedema.

The combination of pulmonary arteriolar contraction and the cessation of respiratory movements may have reduced both arterial oxygen content and return of blood to the left heart with resultant ischaemic contracture of the left ventricle. The very hard left ventricle was a striking finding in all cases and is considered to represent a very pronounced cardiac rigor mortis. Althaus et al. (1982) were struck by a similar finding in two avalanche victims, both of whom survived following open cardiac massage. This intensive myocardial contraction may therefore be an early manifestation of ischemia in these patients, present from the moment of cardiac arrest and identical to the stone heart syndrome well known from cardiac surgery (Cooley et al. 1972). Contraction band necrosis as demonstrated in six of our cases is a typical but non-specific finding in the stone heart syndrome (Hutchins and Silverman 1979). We agree with Althaus et al. (1982) that this contraction of the heart may severely have reduced or even totally nullified the effect of external chest compression in cardiopulmonary resuscitation. This may explain why resuscitation efforts seem to have had so little effect on aortic oxygen pressure.

With the great similarity of the findings in all autopsied cases, including pulmonary and cerebral oedema, central congestion, Tardieu spots, and intense left ventricular rigor, the pathophysiology leading to death was probably also similar. Consciousness was definitely lost in four of the survivors including the one who was completely buried for three hours, and was possibly lost in six more. The loss of sense of time and space reported by most of the survivors also indicates that consciousness was impaired. We conclude that the observations are consistent with the following sequence of events in the fatal cases. Thoracic and whole body compression with restriction of respiratory movements and concomitant severe circulatory disturbances probably has lead to rapid loss of consciousness, followed by circulatory arrest within the first few minutes in some and after the blood oxygen had fallen to fatal levels in others. All probably died within the first 20–30 min. One of the six non-autopsied dead in Vassdalen is considered

to have died from drowning in a stream. In the remaining five, there was no external or circumstantial evidence that the mechanism of death was different from that in the majority of the autopsied cases. In summary, the mechanism of death is concluded to be general body compression with acute respiratory and circulatory failure in 15, combined general body compression and mechanical trauma in two, and drowning in water in one.

In avalanches occurring under other conditions, the effects on the victims may be different. The presence of trees and other objects towards which the victims may be thrown increases the risk of mechanical injuries, as illustrated by the Nord-Lenangen cases. Rocks and ice blocks may detach from the mountainside, blocks of dense snow may persist, and big rolling snowballs may be formed when the snow is wet, all of which increase the risk of mechanical injuries. Under such conditions, or when the victims are in automobiles or houses hit by the avalanche, the snow masses may be more unevenly packed and give more breathing possibilities. The victim who survived three hours of burial illustrates that under special circumstances which permit breathing, life can be sustained for much longer (Schild 1976; Markwalder 1969).

Under special circumstances, powder snow avalanches may attain very high, even supersonic speeds (Eliakis 1974). In such cases, barotrauma may be caused by the wave of positive pressure travelling in front of the avalanche and the following wave of negative pressure, causing tympanic membrane rupture and pneumothorax (Markwalder 1969; Eliakis 1974). Except for pneumothorax in one of the surviving victims, no such lesions were observed in our cases. The significance of the pressure wave as a cause of death seems doubtful, except when victims are hit by flying objects or thrown towards objects (Markwalder 1969, 1976).

In 20 autopsied avalanche victims over a seven-year period in Innsbruck in Austria, the cause of death was concluded to be atlanto-occipital dislocation in one and a combination of thoracic compression, lack of oxygen, aspiration, hypothermia and circulatory disturbances in 19 (Lugger and Unterdorfer 1972). In 24 autopsied victims in Davos, Switzerland, the cause of death was concluded to be mechanical trauma in one, hypothermia in two, snow inhalation in one, and suffocation under the weight of snow in 20 (Eliakis 1974). Among 43 cases in the Swiss Alps over a 10-year period, the cause of death was concluded to be asphyxia in 21, mechanical injury in five, combined asphyxia and injury in two, hypothermia in 1, psychogenic shock in nine and unknown in five. The diagnosis of psychogenic shock, supposed to result from seeing the oncoming life-threatening avalanche, was doubtful, and there is little to support that this may cause death in otherwise healthy individuals (Markwalder 1969). Among 41 victims in the French Alps, the cause of death was assumed to be fracture of the cervical spine in four, multiple fractures in seven, suffocation in 16, hypothermia in two, drowning in one und unknown in one. No cause of death was indicated in the remaining 10 (Lapras 1980). With the addition of the present report, this gives a total of 136 cases in which the immediate cause of death was concluded to be some form of suffocation in 67.6%, a combination of suffocation and mechanical trauma in 2.9%, drowning in 1.5%, mechanical trauma in 13.2%, hypothermia in 3.7%, and unknown (including assumed psychogenic shock) in 11.0%.

Acknowledgements. We thank Drs. Pål Herlovsen and Håvard Levang for information from the psychiatric team and Dr. Ole C. Ingebretsen for assistance in performing the blood gas analyses.

References

Althaus U, Aeberhard P, Schüpbach P, Nachbür BH, Mühlemann W (1982) Management of profound accidental hypothermia with cardiorespiratory arrest. Ann Surg 195:492–495

Coe JI (1977) Postmortem chemistry of blood, cerebrospinal fluid, and vitreous humor. In: Tedeschi CG, Eckert WG, Tedeschi LG (eds) Forensic Medicine. A study in trauma and environmental hazards, vol 2. WB Saunders, Philadelphia, pp 1042–1043

Cooley DA, Reul GJ, Wukasch DC (1972) Ischemic contracture of the heart: "stone heart". Am J Cardiol 29:575–577

Eliakis E (1974) La mort violente par avalanche. Mise au point médico-légale. Médecine Légale et Dommage Corporel 7:83–87

Gray D (1987) Survival after burial in an avalanche. Br Med J 294:611–612

Harries M (1986) Drowning and near drowning. Br Med J 293:122–124

Haug AN, Schrøen IL, Berntsen P, Tellefsen T, Husby S, Due J, Lied K, Hansen G, Collett M (1986) Skredulykken i Vassdalen 5. mars 1986. NOU 1986:20. Universitetsforlaget, Oslo

Hutchins GM, Silverman KJ (1979) Pathology of the stone heart syndrome. Am J Pathol 95:745–752

Lapras A (1980) Pathologie des ensevelis. La Nouvelle Presse Médicale 9:3124–3130

Lugger L, Unterdorfer H (1972) Obduktionsergebnisse bei Lawinenverunfallten. Ärztliche Praxis 24:28–31

Markwalder K (1969) Medizinische Aspekte bei Lawinenunfällen. 66 Fälle aus den Schweizer Alpen von 1958 bis 1967. City-Druck AG, Zürich

Markwalder K (1976) Die Pathophysiologie des Lawinenunfalles. In: Lawinen. Tagung über die medizinischen Aspekte des Lawinenunfalles. Juris Druck und Verlag, Zürich, pp 71–76

Mithoefer JC, Mead G, Hughes JMB, Iliff LD, Campbell EJM (1967) A method of distinguishing death due to cardiac arrest from asphyxia. Lancet 2:654–656

Quervain de M (1976) Für den Lawinenunfall wesentliche Lawineneigenschaften. In: Lawinen. Tagung über die medizinischen Aspekte des Lawinenunfalles. Juris Druck und Verlag, Zürich, pp 23–33

Schild M (1976) Problemstellung, Statistik, Standortbestimmung. In: Lawinen. Tagung über die medizinischen Aspekte des Lawinenunfalles. Juris Druck und Verlag, Zürich, pp 13–21

Schmid F (1981) Zur Pathogenese des Lungenödems nach Lawinenverschüttung. Schweiz Med Wochenschr 11:1441–1445

Received January 13, 1989 / Accepted January 27, 1989