PULMONARY OEDEMA WITH NEUROLOGICAL SYMPTOMS AFTER THE FAINTING LARK AND MESS TRICK

BY

Air Vice-Marshal Sir AUBREY RUMBALL, K.B.E., Q.H.P., F.R.C.P.

Consultant in Medicine, the Central Medical Establishment, Royal Air Force

The mess trick and the fainting lark are performed by young people for excitement. Unless complicated by head injury from a precipitate fall, they terminate almost invariably in complete and rapid recovery.

Very occasionally non-traumatic complications are encountered to which no reference in the literature has been found. This communication refers to a non-traumatic complication in which syncope, somewhat prolonged, terminated with neurological symptoms and with acute pulmonary oedema. Both were followed by complete recovery.

Case 1

A youth aged 20 decided to improve on the usual performance of the mess trick by its repetition in quick succession and without interval. Though apparently normal after his first and second episodes, unconsciousness in the third sequence was prolonged. His companions estimated the period of unconsciousness to be at least two or three minutes. Clonic twitches affected his arms and legs. On recovery he struggled with difficulty to a sitting position on the floor. Unsupported he could not stand. Descriptions of his attempts to walk were consistent with his being ataxic as well as having motor weakness. His speech, though intelligible, was dysarthric. The doctor who was summoned arrived in about 15 minutes; in that time control of articulate speech and control of locomotion had steadily improved, in contrast to his complaint of becoming increasingly "winded." At first this complaint was attributed to clumsiness of his two assistants, whose part in the trick had been to compress his chest bimanually on his completion of the hyperventilation phase, and then to maintain compression until the rise in intrathoracic pressure so created ended in syncope. Physical examination before he was moved to hospital recorded respirations of about 30 a minute. Neck veins were full, but there was uncertainty about skin cyanosis. Neither his ribs nor his rib movements were painful. There was occasional cough with thin watery expectoration. Resonance to percussion was impaired over both lower regions of the chest, where bubbling rales were heard in abundance. Tendon reflexes were present

and brisk in all four limbs. His plantar reflexes were recorded as bilaterally extensor in type.

On arrival in hospital about 45 minutes later, he had recovered from dysarthria while motor power and ataxia were not remarked on, though it was observed that his plantar reflex responses had become predominantly flexor. Cranial nerves were intact, but defective ocular convergence caused diplopia with closely overlapping vertical images for objects nearer than 2-3 ft. (0.6-0.9 m.). His symptom of "being winded" was unchanged. His pulse was 115 a minute; his blood-pressure was elevated to 155/92 mm. Hg. Physical signs in his chest were as already recorded.

Chest Radiography.—The rib cage was devoid of bony damage. The upper lobes of the lungs were spared the non-homogeneous loss of translucency that was evident in the lower lobes (Fig. 1). This radiological appearance together with dyspnoea and cervical venous engorgement improved rapidly without treatment. Within 24 to 36 hours his thoracic x-ray was virtually normal, and apart from a slight defect in ocular convergence he had no symptoms. He was detained in hospital for three days.

Case 2

A youth aged 19 was persuaded by a group of friends of like age to perform the fainting lark for the first time. His subsequent experience was not dissimilar to that already described in Case 1. His own account of the episode was as follows:

"We had just finished our midday meal; the room was hot and stuffy. My friends who knew about the fainting lark showed me how to squat on the floor, where for two or three minutes I breathed in and out moderately deeply every three or four seconds. Then I closed my mouth, pinched my nose, and while doing this tried hard to breathe out. Both my ears 'plopped.' My friends beckoned me to stand upright, which I did. I don't remember falling, but everyone was positive that I did not hit my head on the wall, furniture, or floor. When I came round I had the sensation of drowning. I could only breathe in small short breaths. I was able to see and hear without difficulty but I could not move my limbs or body. My

FIG. 1

FIG. 2

FIG. 3

FIG. 1.—Case 1. Thoracic radiograph taken about one hour after completing the mess trick. FIG. 2.—Case 2. Thoracic radiograph taken about one hour after completing the fainting lark. FIG. 3.—Case 2. Thoracic radiograph taken when signs of pulmonary oedema had subsided.

friends said I had remained unconscious for some three minutes. When they had pulled me into a sitting position on the floor my breathing became easier, but I could not speak or move my body or limbs. My face, at first pale, was said to have gone blue, and veins in my neck were swollen; in fact, my whole neck seemed to be bigger. Shortly afterwards I moved my arms and legs but I couldn't stand. I could speak, but my voice was too slurred to be understood. It was still hard to breathe, but I coughed some frothy, slightly blood-stained stuff. The doctor who was called sent me to hospital. My skin felt numb on my legs and body more than on my arms and chest. I felt physically weak in bed, though I could move my legs and arms. I could see 'double' when I looked straight ahead. For nearly two days I had some difficulty in passing water, and even in bed I felt chilly."

Though not recorded in particular detail, his hospital notes confirmed the substance of the foregoing account. He was apyrexial. At first his pulse rate was 120 a minute but became normal in 36 to 48 hours. Blood-pressure was 158/90 mm. Hg. Cyanosis of face with thin watery expectoration was recorded. Respirations were all quick and shallow. Physical signs in his chest were described as "consolidation, with alveolar fluid."

Tendon reflexes were bilaterally brisk; plantar responses elicited probably an hour or more after the onset of the condition were bilaterally flexor. Appreciation of cutaneous and deep sensation appears to have been intact. His notes mentioned diplopia when looking forward. Vertically parallel double images were then present for near objects and for objects seen across the room. This was still slightly present for near objects on the fifth day.

Chest Radiography.—The thoracic cage was devoid of bony injury. Prominent in the mid-zone with sparing of the apices and bases was a lobular pattern of reduced radiotranslucency and a measure of cardiac dilatation with prominent pulmonary arteries (Fig. 2). It was an appearance consistent with pulmonary oedema. Chest symptoms had improved markedly in 24 hours and had virtually subsided after 48 hours. A chest x-ray film (Fig. 3) taken when pulmonary oedema had subsided disclosed restoration of normal lung appearances.

Discussion

The subjects of these post-syncopal complications have remained well since the foregoing events, which had happened nine and seven years previously. They have not repeated the trick, nor could they be persuaded to do so. Only because each was medically examined for fitness for flying (pilot duties) did their earlier history come under close scrutiny. The original hospital diagnosis was uncertain, but lung trauma was postulated in one of them.

It must be established whether or not their respiratory symptoms could have resulted from lung trauma. External pressure on the thorax may damage lungs through fractured rib ends, while "blast lung" and pulmonary contusion may arise without damage to the rib cage. Interstitial pulmonary emphysema may complicate damage to lung alveoli, whatever the cause of damage. In Case 1 hand pressure applied to the chest cannot be assessed with any certainty, but neither the radiological appearance, nor the clinical course, nor the quick recovery points to lung trauma as a likely cause of the lung symptoms.

In Case 2 intrapulmonary pressure was sustained from the Valsalva manoeuvre, but there is ample evidence to show that rupture of healthy lung tissue must be exceptional as a sequel, if indeed it occurs at all. Haldane and Priestley (1935) observed that muscular fixation of the chest wall necessary to sustain the manoeuvre provides automatic counterpressure to traumatic overdistension from internal pressure. In practice the manoeuvre has formed part of a test-battery used by more than 50,000 Royal Air Force pilots who have regularly sustained pressures equal to 40 mm. Hg for a period of not less than half a minute and

often exceeding a minute. At the same examination they reached, for brief periods, pressures of 80 to 120 mm. Hg. It may be accepted that since no pulmonary incident complicated this large series of recorded tests the manoeuvre can be performed in healthy men with safety. Neither the clinical course nor radiological appearances suggest that trauma took part in the genesis of this man's pulmonary condition.

Concept of Neurogenic Pulmonary Oedema

The combination of unconsciousness followed by brain-stem symptoms with pulmonary oedema merits consideration of a single unifying cause. The association of intracranial lesions and pulmonary oedema is well described, notably by Weisman (1939), Luisada (1940), Cameron (1948), and Haddy *et al.* (1949). Clinical observers have postulated the existence of abnormal autonomic cerebral impulses that alter the permeability of lung capillaries (Cameron and De, 1949). This concept is not supported by animal studies (Sarnoff and Berglund, 1952; Gamble and Patton, 1953; Maire and Patton, 1956). These workers demonstrated that specific cerebral damage led consistently to an outflow of uninhibited vasomotor impulses with consequent displacement of blood from vessels possessing high constrictor potential into lung vessels, where such potential is slight. Gamble and Patton (1953) considered the term neuro-haemodynamic pulmonary oedema to be preferable to neurogenic pulmonary oedema. This haemodynamic concept receives clinical support in man, though not in all instances.

Baker (1957) reported at post-mortem examination focal bilateral lesions of the dorsal nucleus of the vagus and the medial reticular nuclei of a patient with bulbar poliomyelitis, whose life had terminated in pulmonary oedema and high tachycardia from vagal damage. Heart failure was inferred as the cause of pulmonary oedema. Campbell and Visscher (1949) reported raised intracranial pressure associated with hypertension that would conform to Gamble's description of neuro-haemodynamic pulmonary oedema. Richards (1963) found neither constant nor consistent disturbance in circulatory haemodynamics to account for pulmonary oedema in patients in whom that condition occurred in the research unit of a neurosurgical centre. The impression is left that perhaps more than one neural trigger may cause pulmonary oedema in these patients.

Experimental Neuro-haemodynamic Pulmonary Oedema

Consideration of animal experimental work is illuminating in connexion with neuro-haemodynamic pulmonary oedema that may arise in man. Gamble and Patton (1953) and Maire and Patton (1956) demonstrated in rats that discrete bilateral basal lesions to the pre-optic hypothalamic region caused swiftly fatal pulmonary oedema. The affected region failed in its normal inhibitory control over vasomotor impulses that emerge from cells more caudally situated in the hypothalamus or neuraxis. Ganglion-blocking drugs conferred protection on these animals, and more revealing was the observation that splanchnicectomy alone gave protection. These workers tentatively suggest that overloading of the pulmonary circuit with blood resulted from splanchnic-mediated constriction of the visceral venous reservoir. The animal's viscera, including spleen and liver, weighed significantly less than in control animals which had not succumbed to pulmonary oedema. This mechanism depends more on constriction of visceral veins and venules than on constriction of arterioles in the systemic vascular bed.

It is uncertain whether this mechanism, if present in man, would be associated with a smaller-than-expected rise in mean systemic arterial pressure, or even whether the result of animal experiments can be applied in detail to man; but without violating any principle it can be inferred that such a hypothalamic centre in man probably exists. The role of veins in the haemodynamic shift of blood has been carefully reviewed by Landis and Hortenstine (1950) and by Gollwitzer-Meier (1932). Closed venous segments in the forearm (Ruch and Fulton, 1960) may develop pressures under sympathetic constrictor impulses that rise to 40 or 50 mm. Hg. There is much to support this concept of neuro-haemodynamic pulmonary oedema in which veins play an important part.

Cerebral Blood-flow Changes in Fainting Larks

We know that mean systemic blood-pressure when reduced to 50 mm. Hg or less will reduce speed of cerebral flow below the level needed by the reticular formation to alert the brain to full and conscious activity (Jefferson, 1961). When a deliberate syncopal manoeuvre is carefully conducted the resultant depression in speed of cerebral blood flow may be significantly greater than occurs in spontaneous fainting. Hyperventilation that avoids unnecessary muscular effort in deliberate syncopal manoeuvres will reduce arterial carbon dioxide tension (P_{CO_2}) by 20 or even 25 mm. Hg (Campbell, 1961). With this degree of hypocapnia cerebral veins as well as arteries constrict vigorously to reduce cerebral blood flow by 35% (Ruch and Fulton, 1960), while cerebral hypoxia is pre-disposed by a shift to the left of the oxygen-dissociation curve (Bard, 1960).

The Valsalva manoeuvre will add substantially to depression in cerebral flow rate by isolating central venous pressure from peripheral inflow through retrograde closure of valves, with a resultant sharp fall in mean systemic pressure. Cerebral blood flow is thus depressed below its critical level for consciousness and the Valsalva manoeuvre becomes responsible finally for precipitating syncope. The postural effect of standing after squatting, however, adds its quota to the foregoing changes. According to Patterson and Warren (1952) and to Scheinberg and Stead (1949), blood flow is depressed by 21% or more on standing after squatting, but one-third of this is offset by a simultaneous fall in jugular pressure except in the presence of the Valsalva manoeuvre, which will abolish this normal postural arterio-venous pressure gradient. Serious cerebral hypoxia will result when cerebral flow is reduced by 40% or more (Ruch and Fulton, 1960). This can be reached and exceeded in syncopal manoeuvres; but fortunately most performers are not obsessional enough, or are unaware of what is needed to press the manoeuvre to its full extent.

In the cases under discussion only speculation is possible both on the location and on the nature of cerebral vascular changes, which, had they been present, might have caused some or all of the symptoms already detailed. Defective ocular convergence possesses localizing value. An identical symptom in cerebral concussion is at post-mortem examination associated with punctate haemorrhages located at or near the central grey matter of the upper brain-stem. It is reasonable to assume that this location is applicable to the present cases, and, further, the closely adjacent formation reticularis was probably abnormally disturbed, because unconsciousness persisted longer than is usual in uncomplicated syncope. The positive Babinski responses detected in Case 1, and the grouping of neurological symptoms in both cases, would be consistent with neural disturbance

localized to the foregoing region. Should precise location of an "oedemagenic centre" be considered on the basis of experimental animal work, the adjacent basal hypothalamus near the midline would be involved. Assuming that a vascular disturbance was responsible for the whole symptom-complex, we must consider what peculiarities in these cases could exist when most people under similar circumstances would have escaped.

Anatomical and Morphological Aberrations in Basal Cerebral Arteries

Severe and even gross aberrations in anatomy and morphology of basal cerebral arteries may exist unsuspected for years, especially when congenital and in young people; indeed, they are rarely diagnosed clinically until after atherosclerotic changes have affected them in later life. In the event of their suffering a major overall reduction in cerebral blood flow, unexpected poverty of supply is revealed by symptoms related to the region involved. The origins of the anterior cerebral arteries are normally regions of particular vulnerability through being at points of "no flow," described by Symonds (1955). The proximal segment of the anterior cerebral artery has been investigated by Webster *et al.* (1960). This segment may be completely absent or it may be no more than a tenuous thread-like vessel, while ganglionic branches arising from it are similarly affected. In the series of Webster *et al.* (1960) unilateral defects of a major kind affected the proximal segment and its ganglionic branches in 11%, while they were bilaterally affected in 0.5%.

Although it is conjectural to consider whether or not the subjects of this discussion had unsuspected cerebrovascular anomalies, not to consider them is to leave an important possibility unexplored. Thrombosis of one or more arterial branches is unlikely, while haemorrhage in a condition that recovered so quickly and completely is most improbable. Branches from the proximal segment of the anterior cerebral artery along with branches from the basilar and posterior cerebral arteries supply the basal hypothalamus and upper brain-stem (Mitchell, 1953). The effect on blood flow in these vessels, and especially their terminal and ganglionic branches, may be more profound as the result of a "fainting lark" than in spontaneous syncope. The existence of certain major anatomical and morphological vascular aberrations in this basal region may, under the influence of a fainting lark, reduce blood flow in branches most affected to be tantamount to vascular "shut-down." The neurological symptoms in the cases referred to here were almost certainly a result of anoxic dysfunction, and it is reasonable to suppose that an "oedemagenic centre" involved in anoxia would react, though less severely and not irreversibly, as would be expected in experimental animals after a focal and discrete lesion to the basal pre-optic region of the hypothalamus. Whether the foregoing hypothesis is correct or not, the onset of pulmonary oedema in both cases had the abruptness that has characterized the experimentally produced condition.

Conclusions

Neither the fainting lark nor the mess trick can be regarded as safe. They are potentially hazardous parlour games. Risk of head injury from a sharp fall with sudden unconsciousness predominates, especially when the lark is not conducted on a soft or carpeted surface, well away from walls and hard furniture. To my knowledge disregard of these precautions has accounted for cerebral concussion by impact with parquet flooring in two instances and with walls and hard furniture in four more.

Two skulls were also cracked through lack of precautions. Now there is evidence that non-traumatic risks are present which if numerically fewer are no less unpleasant or hazardous.

Summary

The foregoing occurrence of acute pulmonary oedema with neurological disturbance followed promptly after completing the "mess trick" and the "fainting lark." Syncope in each case occurs through well-established physiological vascular changes. Lung trauma is considered to be an improbable cause of pulmonary oedema. Neither cerebral thrombosis nor cerebral haemorrhage is thought to be likely as a cause of neurological disturbance. A proved connexion exists between pulmonary oedema and cerebral damage: both conditions have been confirmed at post-mortem examination. With lesser degrees of cerebral damage recovery from pulmonary oedema may occur.

The infliction of discrete lesions to the hypothalamic pre-optic nuclei in experimental animals is regularly followed by acute and severe pulmonary oedema. Uninhibited vasomotor impulses in these animals will move blood from regions of high constrictor potential into lung vessels possessing low constrictor potential. The pulmonary oedema that follows is essentially neuro-haemodynamic in origin. By applying to man the principle elicited by this experimental work, an unusual symptom-complex after the fainting lark or mess trick becomes understandable. The presence is postulated, in these people, of unsuspected anatomical and morphological anomalies in the ultimate arterial supply to the hypothalamus and upper brain-stem; this, coupled with the very marked reduction in overall cerebral blood flow that accompanies a dedicated performance of the fainting lark, is calculated to deprive of blood both the oedemagenic centre in the hypothalamus

(presumed to exist in man) and parts of the upper brain-stem. It would be tantamount to their sustaining arterial shutdown. Therefore this reversible neural disturbance most probably causes neuro-haemodynamic changes which result in pulmonary oedema and a variable neural dysfunction in the upper brain-stem.

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PULMONARY OEDEMA AND INTRACRANIAL LESIONS

BY

PETER RICHARDS,* M.A., M.B., M.R.C.P.

Research Assistant to the Neurosurgical Unit of St. George's Hospital at Atkinson Morley's Hospital, Wimbledon

Pulmonary oedema is a frequent necropsy finding in subjects with a variety of intracranial lesions. Weisman (1939) found it at necropsy in 70% of 686 patients with traumatic or spontaneous intracranial haemorrhage and in only 2% of 200 controls. Cameron (1948) found a similar incidence and also noted that it was common at necropsy in patients dying from hypertensive disease (86%), chronic nephritis (74%), complete coronary obstruction (68%), and mitral stenosis (65%). In contrast, Paine *et al.* (1952) claimed that pulmonary oedema and congestion was less common in patients dying with "significant disease of the central nervous system" (46%) than in those who died with other major illnesses (60%). Moreover, they found evidence of hypertension and/or heart disease in three-quarters of those with lesions of the central nervous system and concluded that an essentially cardiac origin of the oedema was likely.

Little attempt has been made to correlate particular lesions of the central nervous system in man with the occurrence of pulmonary oedema, with the exception of Baker (1957), who demonstrated consistent bilateral damage to the dorsal nuclei of the vagus and to the medial reticular nuclei in 10 patients with bulbar poliomyelitis dying with pulmonary oedema, but not in five others without oedema.

In experimental animals a variety of injuries to the central nervous system provoke pulmonary oedema in different species. These procedures include non-specific rise in intracranial pressure in guinea-pigs (Campbell and Visscher, 1949) and dogs (Harrison and Liebow, 1952), intracisternal injections of thrombin and fibrinogen in rats and rabbits (Cameron and De, 1949) and dogs (Sarnoff and Berglund, 1952), discrete bilateral lesions of the anterior hypothalamus or paraventricular nuclei in rats (Gamble and Patton, 1953; Maire and Patton, 1956), and either bilateral lesions of the vagal nuclei, transection of the cervical cord at C7, or bilateral vagotomy in guinea-pigs (Borison and Kovacs, 1959). Bilateral vagotomy has long been known to precipitate pulmonary oedema, and this, as well as oedema produced by the other methods mentioned, is often accompanied by multiple discrete haemorrhages throughout the lungs which were first described by Valsalva (1740).

This paper presents clinical and pathological observations on pulmonary oedema complicating lesions of the central nervous system made in the course of a study of respiratory infections in this neurosurgical unit. Particular attention has been paid to the incidence, the associated lesions of the central nervous system, the lobar distribution of oedema, associated heart disease and hypertension, and to the clinical and radiological features.

*Now at the Royal Hampshire County Hospital, Winchester.