ACUTE PULMONARY ŒDEMA WITH HYPOGLY- CÆMIC COMA

AN EXAMPLE OF ACUTE PULMONARY ŒDEMA OF NERVOUS ORIGIN

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(RECEIVED 10TH APRIL, 1942)

THE patient, a man (A. N.), aged 40 years, was receiving 60 units of protamin-zinc insulin daily for his diabetes mellitus, when he was infected with influenza. As a result he fell into a sub-comatous condition, but he recovered quickly with daily 90 units of ordinary insulin and 100 gm. of carbohydrate (porridge), the urine becoming free from sugar and ketone bodies in 3 days. About a week later, on 1st February, 1942, at 6 p.m., after an injection of 30 units of ordinary insulin, the patient complained of a disagreeable feeling of hunger, which was got rid of by 20 c.c. of a 20 per cent. solution of glucose together with a meal of porridge. Six hours after the insulin injection the patient during apparently ordinary sleep fell into a condition of coma, which commenced with typical pulmonary Œdema. There were the severest symptoms of hypoglycæmic shock: unconsciousness; extremely contracted pupils which did not react to light; muscular rigidity; restlessness; ankle and patellar clonus; bilateral plantar reflex of Babinski type; psychotic screaming. One might describe the condition as one of "screaming coma" with tetanus-like rigidity of limbs and trunk (opisthotonus). Treatment for the hypoglycæmic coma did not remove the pulmonary Œdema, but a hyoscine-atropine injection worked immediately and brought the severe attack to an end, though the patient seemed rather weak for 2 or 3 days afterwards.

Discussion

In the present case there was no evidence of mitral stenosis, coronary sclerosis, myocardial anæmia, arterial hypertension, or any chronic disease of the lungs or cardio-vascular system, and it seems clear that the acute pulmonary Œdema must have been of nervous origin. We would compare it to the attacks of pulmonary Œdema sometimes associated with epileptic attacks and occasionally constituting the fatal termination. In rare cases of paroxysmal pulmonary Œdema (Riesman, 1907, Case 2) cerebral symptoms, such as fleeting aphasia, paralysis of limbs, stupor, or coma, have been associated with the attacks. Shanahan (1908) recorded a number of cases illustrating acute pulmonary Œdema as a serious complication of epileptic seizures, and Ohlmacher (1910) described five epileptic cases in which acute pulmonary

œdema constituted the terminal event. Langeron (1925) discussed the case of a woman in whom epileptic crises were regularly followed by an attack of acute pulmonary œdema. One of us (Weber, 1922) would also compare as an analogous phenomenon eclamptic attacks in children during which auscultation of the chest reveals rapid "filling up" with coarse moist râles, passing away when the attack subsides. Such temporary bronchorrhœa in children during eclamptic states is probably due to irritation of the bronchosecretory fibres of the vagus nerve. Moutier (1918) has drawn attention to occasional cases of fatal pulmonary œdema in soldiers suffering from cerebral wounds and the possible danger of adrenalin injections in such cases. Aubertin (1908) experimentally in animals produced convulsions and acute pulmonary œdema by lead poisoning. Bezançon and others (1932) recorded the case of a woman, aged 39 years, with post-encephalitic Parkinsonism, without any cardiac, aortic, or renal lesion, who suffered from recurrent attacks of acute pulmonary œdema, one of which proved fatal. Porter and Greenfield (1941) at a Meeting of the Neurological Section of the Royal Society of Medicine, communicated a case of the "Arnold-Chiari malformation" of the brain-stem with a fatal termination resembling acute pulmonary œdema. It was suggested that the clinical picture was produced by an excessive secretion of the bronchial mucosa glands (? a central autonomic phenomenon) and that the response to atropine was in favour of that view.

Langeron (1925) in the account of his case, above referred to, regarded the attacks of acute pulmonary œdema in that case as a "visceral manifestation of epilepsy." He stated that this "sympathetic epilepsy with visceral manifestations" was from his point of view a spreading out (extension) of the "cortical epilepsy with muscular motor manifestations."

Manunza (1935) has drawn up an elaborate classification of pulmonary œdema of nervous origin. His first class comprises those cases connected with cerebral affections (inflammations, hæmorrhages, tumours, epilepsy), whilst he makes a second class of the cases associated with medullary affections (myelitis, hæmatomyelia, tabes dorsalis) and a third class with affections of the vegetative nervous system and "dysneurotonic" states; his fourth class consists of cases of traumatic nervous origin. Here one might mention that Müller (1891) described an attack of acute pulmonary œdema associated with angioneurotic œdema of the face.

It is certain that the origin of attacks of acute pulmonary œdema varies considerably in different cases and that in most cases multiple factors are at work, but it must be admitted that in some cases an attack may arise in the absence of any true weakness of the right side of the heart (as judged from ordinary and X-ray and electro-cardiographic examination) and without any sudden congestion of the pulmonary blood-vessels, due only to reflex or other disturbance of nervous centres. Hess (1931, 1932, 1933, 1939) lays stress on the probable intervention of nervous reflexes. Impulses, he thinks, which arise sometimes, but not always, in the heart, irritate the vasodilators of the lungs and produce active congestion together with increased transudation into the pulmonary alveoli. In this process, according to his views, the vagus

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nerve plays an important part. Wassermann's observation (1933) is interesting, namely, that in some cases pressure on the vagus fibres in the carotid (carotid sinus) can suppress the development of an attack—as it may likewise suppress some attacks of acute paroxysmal tachycardia. Certain observations in experimental pharmacology speak in favour of a vagal causative mechanism in acute pulmonary Œdema: vagus irritation from cholines and acetylcholine (Dale, 1914); vagotropic effect of pilocarpin with provocation of an attack of pulmonary Œdema; the effect of Sympatol during an attack in diminishing vagotonus through excitation of sympathetic fibres.

We suggest that in our case the attack of acute pulmonary Œdema was of pure central nervous origin, due to cerebral Œdema and irritation of the vagal nervous system. The clinical symptoms and the rapid response to hyoscine and atropine support this view.

Summary

A case is described of acute pulmonary Œdema combined with hypoglycæmic coma, as analogous to the cases connected with epilepsy and other cerebral disturbances. Various nervous origins of attacks of acute pulmonary Œdema are shortly considered.

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Addendum

Dr. H. Pullar Strecker (*J. ment. Sci.*, 1938, **84**, 146) knew of only four cases of acute pulmonary Œdema occurring as a (most dangerous) complication in connection with the insulin shock therapy for schizophrenia, etc., and referred to the following papers: M. Müller, *Schweiz. med. Wschr.*, 1936, **66**, 929; W. Beiglböck and Th. Dussik, *Schweiz. Arch. Neurol. Psychiat.*, 1937, **39** (Ergänzungsheft), 38; N. Beno, *ibid.*, 149; A. Kronfeld and E. Sternberg, *ibid.*, 187. Dr. Pullar Strecker kindly also gave us later references to the subject: L. A. Finiefs, *J. ment. Sci.*, 1938, **84**, 678; S. W. Gillman and D. N. Parfitt, *ibid.*, 718; W. R. Thomas and I. G. H. Wilson, H.M. Stationery Office, 1938, 22.