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 oedema: vagus irritation from cholines and acetylcholine (Dale, 1914); vagotropic effect of pilocarpin with provocation of an attack of pulmonary oedema; 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 oedema was of pure central nervous origin, due to cerebral oedema 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 oedema combined with hypoglycæmic coma, as analogous to the cases connected with epilepsy and other cerebral disturbances. Various nervous origins of attacks of acute pulmonary oedema are shortly considered.

REFERENCES


Addendum

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