THE "MESS TRICK" AND THE "FAINTING LARK"

BY

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The following trick is sometimes played in a mess or at a party: a victim is persuaded to hyperventilate for about a minute and then his chest is suddenly compressed by someone standing behind him. He usually loses consciousness in a few seconds. A procedure with a similar outcome has been described by a practitioner in a question to the *British Medical Journal*:

"A colleague has recently mentioned to me a practice which is current in the school attended by his son, aged 12; the practice is known as the 'fainting lark.' According to the description given, a boy desiring to indulge in this 'lark' squats on his haunches and takes 20 deep breaths. He then rises quickly to his feet, closes his nostrils with his fingers (as for inflation of the Eustachian tube by Valsalva's method), and blows hard. The immediate result is alleged to be that he drops to the ground and 'passes out' for a period of about half a minute."

Previous work in this department suggested that the two procedures were closely related, and this paper describes some of the physiological mechanisms involved. All observations were made on ourselves.

The "Mess Trick"

Effect of Sudden Compression of the Chest

Fig. 1, A, shows the effect of suddenly squeezing the chest of a normal subject. The intrathoracic (oesophageal) pressure jumps to about 40 mm. Hg, and remains there during the squeeze. This is because the subject instinctively counters the pressure on his ribs by closing his glottis and contracting his abdominal muscles. In effect

FIG. 1.—Intrathoracic pressure in a normal subject recorded with a water-filled oesophageal tube attached to a capacitance manometer (Hansen, 1949). Chest squeezed at signal. (A) Natural response. (B) Negative response when subject takes care not to close glottis. (C) Valsalva manoeuvre at 40 mm. Hg.

FIG. 2.—Brachial blood pressure (capacitance manometer). (A) Effect of Valsalva manoeuvre. (B) Effect of sudden squeezing of chest.

he performs Valsalva's manoeuvre. With practice a subject may learn to avoid closing his glottis and to counter the squeeze by an inspiratory effort. There is then no change in intrathoracic pressure (Fig. 1, B). The identical effects on the brachial blood pressure of a Valsalva manoeuvre and of a chest squeeze are shown in Fig. 2.

The circulatory effects of Valsalva's manoeuvre are somewhat complex; the relevant point here is that the blood pressure difference across the cerebral circulation is decreased.

Effect of Overbreathing on the Response to Valsalva's Manoeuvre

Overbreathing is known to have two effects that are relevant to the present discussion: it increases muscle blood flow (Abramson and Ferris, 1940), and it increases cerebral vascular resistance (Kety and Schmidt, 1946). Fig. 3 shows that 20 deep breaths are enough to double the blood flow through the forearm. Our observations on variations in response to Valsalva's manoeuvre (which will be the subject of a later paper) indicate that the effect of the manoeuvre is increased when the peripheral blood flow is high. Thus when a Valsalva manoeuvre is performed after a period of overbreathing one may expect a greater than normal drop in effective cerebral blood pressure and, since the cerebral vascular resistance is simultaneously increased, a very substantial drop in cerebral blood flow.

Fig. 4 shows the effects of similar manoeuvres before and after a short period of overbreathing. The blood pressure drops further after overbreathing. The difference in the subjective effects was much more marked than the changes in pressure: the first manoeuvre was performed without effort; the second produced a partial "blackout." The striking difference in the "overshoot" probably reflects a difference in the degree of cerebral ischaemia induced. For this experiment the subject was seated. We can assert from previous experience that had he been standing the effects would have been more severe, and after overbreathing would in all

FIG. 3.—Record of spirogram and forearm volume; venous cuff applied at arrows. During the hyperpnoea the volume record was interrupted. Before hyperpnoea the flow is about 15 ml./min. and after about 40 ml./min.

likelihood have resulted in loss of consciousness. The "mess trick" therefore produces (1) muscle vasodilatation and cerebral vasoconstriction followed by (2) an acute decrease in differential pressure across the brain. This reduces the blood flow to levels causing loss of consciousness.

The "Fainting Lark"

The resemblance to the "mess trick" can now be seen. A voluntary Valsalva replaces the involuntary response to chest compression, and the effect of rising from the squatting position is added. Fig. 5 shows a comparison of the effect of rising from the squatting position with and without hyperventilation: the blood-pressure fall that normally accompanies rising after squatting is considerably enhanced by previous overbreathing. It is easy to see that the additional procedure of Valsalva's manoeuvre will rapidly bring the brain blood flow to the lowest levels. This has not been recorded, however, owing to the experience gained by a preliminary observation on one subject who followed the reported pro-

FIG. 4.—Effect on blood pressure of standardized Valsalva manoeuvre. Supporting 40 mm. Hg for 8 seconds (A) before and (B) after 20 deep breaths.

cedure exactly. After about five seconds of a moderate Valsalva he became suddenly unconscious. This was followed by generalized clonic convulsions and apnoea lasting 30 seconds, and it was considered that the violent movements made it impossible to obtain an accurate record of the blood pressure during the full attack.

Knowledge of the mechanism involved allows a number of variations on this scheme to be devised. Anyone, however, considering practising these tricks

on himself or his friends should be prepared for the consequences. The main risk is of injury during falling.

The schoolboys have combined three different methods of affecting the blood supply to the brain and devised a procedure for making any normal man unconscious in 60 seconds without outside interference. They are to be congratulated on their ingenuity.

Summary

The "mess trick" consists in suddenly compressing the chest after hyperventilation for about one minute. Hyperventilation leads to dilatation of muscle and constriction of

FIG. 5.—Effect on blood pressure of rising from squatting position. (A) Control and (B) after 20 deep breaths. For this observation the manometer was strapped to the subject's forearm, and the arm was held horizontally throughout. The pressure recorded is thus that at shoulder level. The pressure at brain level is about 15 mm. Hg less.

brain blood vessels. Compression of the chest causes the victim to perform an involuntary Valsalva manoeuvre, which in the presence of peripheral dilatation is sufficient to reduce effective blood pressure and consequently cerebral blood flow to levels which cause unconsciousness.

The "fainting lark" consists in a subject's squatting on his haunches and hyperventilating for 20 breaths, rising rapidly to the erect posture, and immediately performing the Valsalva manoeuvre. Consciousness is lost within a few seconds. The mechanism is the same as in the "mess trick," with the added factor of the fall in blood pressure produced by rising from the squatting posture.

REFERENCES

- Abramson, D. I., and Ferris, E. B., jun. (1940). *Amer. Heart J.*, **19**, 541.
Hansen, T. (1949). *Pressure Measurement in the Human Organism*. Teknisk Forlag, Copenhagen.
Kety, S. S., and Schmidt, C. F. (1946). *J. clin. Invest.*, **25**, 107.

PULMONARY HAEMOSIDEROSIS

BY

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[WITH SPECIAL PLATE]

Pulmonary haemosiderosis occurs in the main in two separate groups of cases—children and young adults—and presents in each different but characteristic clinical features. Certain of these features, together with the finding of remarkably similar focal lesions, lead us to suppose that the two groups must have at least some aetiological factors in common. It may therefore be permissible to describe the condition as a syndrome.

This paper, based upon a study of five adult cases which have come under the care of one of us (P. E.), all subjects of rheumatic heart disease, and also upon a perusal of the literature to date, is presented in the hope that it may encourage further clarification and unification of the pathological basis in the two groups; for it may well be that these have as yet remained completely separated only because the cases happen to come under different specialties of medicine.

Historical

Although the condition was first noted by Virchow in 1858 it was not until 1928 that Rosenhagen, basing his observations on three necropsies, described the condition of pulmonary haemosiderosis in association with mitral stenosis. Ceelen (1931) described a similar condition in necropsies on two children with severe anaemias.

The basic anatomical feature of each condition is the presence in the lung parenchyma of focal deposits containing pigment-laden histiocytes, and attention has been drawn to them principally because of the radiological changes to which they give rise, especially those of a miliary nature. Further observations have since been added to the literature by various workers, though

special mention must be made of the contributions of Scott *et al.* (1947), Laubry *et al.* (1948), and Lendrum *et al.* (1950), who have contributed greatly to our knowledge of the condition in cardiac cases, and to Waldenström (1940, 1944, 1948) and Wyllie *et al.* (1948) on the condition in children with anaemia.

There can be no doubt, therefore, that we are dealing with an established entity, but in spite of its characteristic nature the condition of pulmonary haemosiderosis is inadequately recognized and incompletely understood.

As an illustration of this, it is notable that, to our knowledge, it was not until some 16 years after Rosenhagen first drew attention to the condition that L. D. Scott made the first ante-mortem diagnosis in this country.

Although in this paper we are concentrating on pulmonary haemosiderosis it should be noted that a miliary radiographic pattern in the lungs is also a feature of *industrial siderosis*—a condition which is, pathologically, distinct from haemosiderosis (McLaughlin, 1951). In industrial siderosis, occurring in electric-arc welders and iron-ore workers, inorganic iron is inhaled in the inspired air and is therefore of *exogenous* origin, and histologically the aggregates of iron pigment are found mainly in the peribronchial and periarterial lymphatic channels and nodes. Moreover, no fibrotic reaction ensues. In contrast, in haemosiderosis aggregates of organic iron pigment are deposited in the lung tissues from the blood and are therefore of *endogenous* origin. Here the aggregates of iron pigment are intra-alveolar and cause a fibrosis which is sharply limited (McLaughlin, 1951). It is also noteworthy that Meiklejohn (1949) has recorded the co-existence of haemosiderosis and silicosis.

We have described pulmonary haemosiderosis as a syndrome and shall attempt to justify this by a separate consideration of the two main groups, from clinical, radiological and pathological points of view respectively.

Group 1: Children with Severe Anaemias

Hitherto some 25 cases have been described in the literature, and a remarkably good survey of the clinical and pathological features has been given by Wyllie *et al.* (1948). Our interest is due to the conspicuous absence, up to the present, of any aetiological correlation of this condition with that of the cardiac group of cases, and, indeed, the paucity even of recorded surmise about the general pathology of the disease.

This is certainly of more than academic interest, for according to reported cases the condition is characterized clinically by attacks of a rather alarming nature which not uncommonly terminate in death from acute cardio-respiratory failure. These consist typically of the sudden development of: moderate fever, gross tachycardia and dyspnoea, cyanosis, and haemoptysis. The last-mentioned symptom may be profuse and repeated, but is not always obvious, for the blood may be swallowed and therefore manifest only as occult blood in the stool.

The subjects may be of either sex, and are often small and underweight for age. Clubbing of the fingers may be present, and may regress during the remissions featured in the disease. Peripheral hypertension is never found.

Apart from the obvious respiratory distress, which may even call for the use of an oxygen tent, the most striking feature is a marked and often profound