area may indicate the presence of a tumour, usually a craniopharyngioma. Further delineation of a hypothalamic tumour depends on special x-ray techniques such as radioisotopic brain scans, cerebral angiography, and pneumography. An electroencephalogram may occasionally provide confirmatory evidence but is rarely of much localizing value.

Nature of Lesions

Hypothalamic lesions are not always followed by clinical effects, presumably because bilateral destruction of a particular vital area is necessary before loss of function becomes apparent.

VASCULAR LESIONS

Severe vascular damage to the hypothalamus can follow a ruptured aneurysm of the circle of Willis.³ This may fairly often give rise to biochemical changes in pituitary function⁴ and much more rarely clinical disturbances, such as hypothermia and adipsia.²

TRAUMA

Severe head injuries or surgery affecting the hypothalamic area may lead to diabetes insipidus, disturbances of temperature control, obesity, and hypopituitarism. Crompton⁵ has shown that in many cases head injury causes haemorrhagic lesions of the anterior hypothalamus affecting the supraoptic and other nuclei. In some instances trauma results in transection of the pituitary stalk, thereby severing the neural connexions between the hypothalamus and posterior pituitary and, much less commonly, the vascular connexions with the anterior lobe.

INFECTIONS

Very occasionally basal meningitis due to tuberculous or virus infections may be followed by diabetes insipidus, temperature disturbances, a grossly excessive appetite, or hypopituitarism. Chronic granulomas, especially sarcoidosis, and histiocytosis may affect the hypothalamus usually as part of more generalized tissue disease.

TUMOURS

The commonest lesions to cause widespread hypothalamic damage are tumours, which may be primary or secondary.

Metastatic deposits in the area of the supraoptic nuclei from carcinoma of the breast or bronchus occur, resulting in diabetes insipidus. On rare occasions the hypothalamus is the site of a glioma, or more often the tumour arises from the optic nerve and invades the adjoining anterior hypothalamus. Much more common is a craniopharyngioma or a large pituitary adenoma which has extended to compress the hypothalamus.

Craniopharyngioma

This tumour comprises up to 13% of all intracranial neoplasms of childhood, though it may also manifest itself in adult life. It is a benign tumour arising not within the brain tissue but from the remains of the embryonic anterior pituitary (Rathke's pouch). Usually a cystic tumour, the craniopharyngioma is most often suprasellar in position and compresses the hypothalamus and optic chiasma. Sometimes there is obstruction to the third ventricle causing an internal hydrocephalus. Impairment of vision is a dominant clinical presentation often accompanied by various degrees of hypopituitarism, of which hypogonadism is the most common. As well as failure to develop puberty, retardation of growth often occurs and is due to the interference with the hypothalamic control of the secretion of growth hormone. Diabetes insipidus is present in about 30% of cases. In some instances, particularly with large tumours, obesity and somnolence are also part of the clinical picture. When there is internal hydrocephalus, symptoms of raised intracranial pressure such as headache and vomiting may be presenting features.

In about 60-70% of cases plain radiographs of the skull reveal suprasellar calcification. Full ophthalmological, neuro-radiological, and endocrinological investigations are required in all cases of suspected craniopharyngioma. The treatment is surgical decompression of the optic chiasma followed by intensive radiotherapy and the replacement of any hormone deficiencies. Useful recent reviews of the hypothalamus are provided by Sheehan,⁶ and the *British Medical Bulletin*.⁷

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Second Opinion, Please

Pulmonary Embolus

A DISCUSSION BETWEEN DR. W. DEWI REES AND DR. HOWARD JENKINS

British Medical Journal, 1972, 2, 102-104

Case History

W.D.R.: I find assessing and treating other doctors' patients particularly difficult, especially when the problem posed is unusual. Such was the case when I was asked to visit a

53-year-old man, who was staying with his sister. The sister was slightly apologetic for asking me to visit as she considered him to be nervous person. Eight days previously he had been discharged from a teaching hospital and had come to Wales to recuperate. In hospital, he had been treated for swelling of the left leg. The swelling had been extensive and had reached

the groin. On leaving hospital, his maintenance treatment had been Lasix and potassium tablets.

The man was lying in bed looking worried. He had remained well until morning, but had awoken breathless and frightened. He found the breathlessness increased when he walked to the toilet. He experienced no pain and, though still short of breath when moving, felt less breathless than earlier in the morning.

At first, I was not greatly impressed by the severity of this man's illness. He looked anxious but not otherwise ill. The only abnormalities I could find were tachycardia and slight oedema of the left leg. His B.P. was 100/70.

Bearing in mind his sister's opinion and because of the paucity of clinical signs, my main impression was that this man was over-anxious. The possibility of transient left ventricular failure or a pulmonary embolus did occur to me. The latter diagnosis seemed unlikely for two reasons. If an embolic episode had occurred, I thought it likely that there would be progressive and not lessening dyspnoea. Secondly, it seemed unlikely that the illness treated in hospital had been a deep-vein thrombosis, as he had been discharged from a teaching hospital without maintenance anticoagulant therapy.

Progress

I was not completely happy about this man, however, and thought it best to admit him to our local cottage hospital. A male bed was vacant and it was the morning the radiographer visited the hospital, so I arranged for the chest to be x-rayed and took an E.C.G. I looked at the x-ray and E.C.G. and regret to say could detect no abnormality. I also read the section on pulmonary embolism in Cecil-Loeb's *Text-book of Medicine* and examined the patient again.

By this time the patient was more agitated and did not look well. He was dyspnoeic at rest, sweating, and his extremities were cold. The pulse rate was 150 with B.P. 100/70. I did not consider the J.V.P. to be elevated. The heart sounds remained normal and the lung fields clinically clear. His condition had obviously deteriorated since I first saw him about one hour previously. I thought it likely that he had a pulmonary embolus and that it was no longer safe to keep him in a general-practitioner unit. Consequently, he was transferred to the medical ward at Bronglais General Hospital, Aberystwyth, where the changes present in the E.C.G. and chest x-ray, that I failed to detect, were noted.

ELECTROCARDIOGRAPHS

H.J.: Yes, the E.C.G. was slightly abnormal. It showed an inverted T wave in leads II, III, and avf without a Q wave. In leads V₁ and V₃ the T wave again was inverted. This combination of an apparent inferior cardiac infarct in the standard leads and an apparent anterior cardiac infarct in the chest leads is suggestive that the real cause is a pulmonary embolus.

CHEST RADIOGRAPHS

As regards the chest x-ray, I think the apparent normality was important. Here was a man collapsed, intensely cyanosed, and now with a J.V.P. raised 2 cm. If this had been due to left heart failure or pulmonary infection, the x-ray changes would by this time have been more apparent and probably diagnostic. What was present was an avascular area at the left base, and this strongly suggested a block of the pulmonary artery to this area.

I suppose one really started off with a bias. The history was suggestive of a deep-vein thrombosis and the left leg still being swollen lent support to this diagnosis. An attack of

acute dyspnoea and especially recurrent attacks must therefore have been regarded as being due to pulmonary emboli until proved otherwise.

EFFECT OF MORPHINE

W.D.R.: The patient had to travel 30 miles by ambulance, from Llanidloes to Aberystwyth, so because of his anxiety and some developing chest pain I gave him 10 mg i.m. morphine. I know his condition had deteriorated and he was cyanotic when he arrived at Aberystwyth. I wonder whether this deterioration was due entirely to his illness or enhanced by the morphine.

H.J. I think it could have been both. He certainly had further emboli after coming to hospital and he might well have had them in the ambulance. Morphine does depress respiration and as the patient is already anoxic it is contraindicated in pulmonary embolus.

Could I raise one point? You mentioned earlier that with a pulmonary embolus you expected progressive dyspnoea. I think this is correct in that with each embolus a further portion of lung shows impaired function. The dyspnoea of the acute phase however does lessen, presumably as the embolus moves peripherally, and may disappear entirely.

W.D.R.: Once a definite diagnosis was established at Aberystwyth, anticoagulant therapy was started. However, his condition continued to deteriorate and the next day you arranged his transfer to a cardiothoracic unit.

H.J.: Yes, he was very ill. The problem now was one of distance and transport. The nearest cardiothoracic unit is 120 miles by road and the man was too ill to travel by ambulance. Consequently a helicopter lift was arranged.

FURTHER INVESTIGATIONS

W.D.R.: I am wondering what further investigations were done after he was admitted to the cardiothoracic unit.

H.J.: He had pulmonary arteriograms. These showed two-thirds obliteration of the right superior and left inferior pulmonary arteries. Cardiac catheterization showed low cardiac output, raised pulmonary artery resistance, filling defects in both main pulmonary arteries, and right heart failure.

On reaching the thoracic centre, his condition had improved. During the night he collapsed and became pulseless. An immediate thoractomy was done, when the heart was found to have virtually stopped. He was placed on a by-pass and large clots were removed from both pulmonary arteries.

W.D.R.: I learnt he had a rocky immediate convalescent period. The final outcome was excellent, and he resumed work six months after embolectomy.

I always imagined that embolectomy needs to be done immediately a large embolus lodges in the pulmonary arteries. This man was able to survive 48 hours before this was done. Is this unusual?

H.J.: What you say is probably true of a very large embolus but Professor P. R. Allison at Oxford has demonstrated experimentally that large emboli do get absorbed quite quickly provided the pulmonary circulation can be maintained and protected from the overload of further emboli.

You must remember that both pulmonary arteries were found blocked at operation and his heart acutely dilated and in asystole. There is no doubt that his final embolus would have been fatal had it not occurred where they were geared up to expect it and deal with it. He was of course extremely lucky that it had not happened in the air, but not only that: he improved to such a degree that on arrival at the unit they could afford to stand back and watch the response to streptokinase. Quite frankly, in spite of the happy outcome of this case, I doubt if I would care to take such a risk again.

Glossary

<i>Lasix</i>	Frusemide.
<i>B.P.</i>	Blood pressure.
<i>E.C.G.</i>	Electrocardiogram.
<i>J.V.P.</i>	Jugular venous pressure.
<i>i.m.</i>	Intramuscular.

Appointments of Speakers

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Dr. S. T. Howard Jenkins, M.B., F.R.C.P., Consultant Physician (Bronglais Hospital, Aberystwyth, Card.)

New Appliance

pH Profile of Gut as Measured by Radiotelemetry Capsule

British Medical Journal, 1972, 2, 104-106

Mr. S. J. Meldrum, Dr. B. W. Watson, and Mr. H. C. Riddle, of the Department of Medical Electronics, and Dr. R. L. Bown and Dr. G. E. Sladen, of the Department of Gastroenterology, St. Bartholomew's Hospital, London E.C.1, write: The relative inaccessibility of the lower gastrointestinal tract and the semi-solid nature of its contents precludes the ready measurement of luminal pH by conventional techniques. It is possible to measure the pH of small-bowel contents obtained by aspiration¹ but it has been shown that the pH of these contents does not always agree with the true luminal pH as measured by intraluminal electrodes.² Normal colonic contents cannot be aspirated and, as shown below, stool pH is a poor reflection of intracolonic pH. A radiotelemetry capsule ("radio pill") has been developed which is swallowed by the subject and continuously measures the pH of surrounding gut contents.

A radiotelemetry capsule for measuring pH was described by Jacobson and MacKay.³ It used as the transducer a copolymer resin which changed its dimensions with pH. The extremely slow response time of this device made it unsuitable for routine clinical use. A similar telemetry capsule later described by Nöller⁴ used an antimony electrode as the transducer. This device suffered from serious drift problems, as antimony is oxidized quickly in the presence of body fluids. The drift and short life of this device make it suitable for use only in the stomach and small intestine.⁵

The pH telemetry capsule described here was developed from the original specification of Watson and Kay⁶ and measures pH by means of a glass electrode. This responds to true changes in hydrogen ion concentration, and the stability of the device is such that it may be used to investigate pH throughout the entire gastrointestinal tract.

Description of Capsule

The device consists of a glass pH electrode, a transistor oscillator, and a replaceable mercury battery encapsulated in a polyacrylate body. A silver chloride reference electrode is situated in the battery cap of the capsule. The overall measurements are 10 mm in diameter and 25 mm long. The potential difference developed between the glass and reference electrodes is proportional to the hydrogen ion concentration of the fluid in which the device is immersed, and this voltage is used to modulate the frequency of the radio transmitter. The device thus transmits at a frequency proportional to the pH of its surroundings. A circuit diagram is shown in Fig. 1.

The response of the device in various buffer solutions is shown in Fig. 2, indicating the linear response between pH 1 and pH 12.

When swallowed the device is at body temperature, but in situations where this is not the case the temperature error introduced is of the order of 0.1 pH unit per °C. The life of the capsule is limited to about 10 days by diffusion of electrolyte from the reference cell, but this is more than enough for transit through the human alimentary system. The end-cap containing the reference electrode may be replaced and the device used again.

FIG. 1—Circuit diagram of telemetry capsule.

FIG. 2—Transmission frequency of capsule at various pH values.