MEDICAL PRACTICE

Clinicopathological Conference

A Case of Anorexia

DEMONSTRATED AT THE ROYAL COLLEGE OF PHYSICIANS, LONDON

British Medical Journal, 1973, 2, 158–163

On 25 January 1973 the Royal College of Physicians held the second of its quarterly clinicopathological conferences. It was presented by Dr. J. J. Daly (1) and the discussion was led by Dr. J. D. N. Nabarro (2).

Clinical Summary

The patient, a 22-year-old unmarried girl, was admitted to the Royal Hospital, Sheffield, in coma on the night of 21 September 1971. Since her early teens she had been obsessed with her weight and had often dieted to lose weight. In 1970 she had seen her doctor for sudden loss of menses but no pelvic abnormality was noted. During December 1970 her appetite had decreased further and her weight had fallen from 7 stone 10lb (49 kg) to 6 stone 9lb (44 kg). She was also vomiting after most meals. At Christmas 1970 she learned that her father had an inoperable carcinoma of the lung. On May 11 1971 she was referred for a psychiatric opinion with a diagnosis of anorexia nervosa. *X*-ray films of her chest and skull were normal but she was pale and had mild glossitis. She was given amitriptyline (25 mg three times daily). Her appetite improved temporarily and she gained some weight (7 stone 4lb) (46 kg). She returned to full-time work. Her employer, a physiologist, noticed that she had an excessive thirst, particularly for lemonade—she was drinking up to six litres daily—and that her anorexia had returned. She tested her urine but detected no glucose or albumin. She became weaker and lost more weight until her admission.

Apart from her father, her family (mother and 13-year-old brother) were well. She had had "poliomyelitis" when she was 2 with admission to the local fever hospital, where a total white blood count of 68,000 cells/mm^3 was found with considerable eosinophilia (this had returned to normal within six weeks). Her C.S.F. had been sterile, cells 10 lymphocyte/mm^3, protein 250 mg/100 ml, glucose 60 mg/100 ml.

On 21 September 1971 she was emaciated (6 stone, 38 kg), comatose, very pale, with slightly cyanosed upper eyelids and a sore, red, dry mouth. Her temperature was 33°C, rising to 36°C three days later. Her fingers were shiny and slightly reddened, and the skin was inelastic. Her breasts were normally developed but her pubic and axillary hair was scanty. There was no lymphadenopathy. No focal signs were found in her central nervous system and both fundi were normal, but some observers thought there was neck stiffness. Her pulse was thin and thready, 120/min, regular. The blood pressure was 90/50 mm Hg. The results of cardiovascular (including electrocardiogram), respiratory, abdominal and rectal examinations were otherwise normal.

The urine contained a trace of protein and some acetone but no glucose. There was a soft opacity in the right upper zone on the *x*-ray film (but this soon cleared and was thought to be inflammatory). Her skull *x*-ray film was normal. Her electroencephalogram showed widespread activity at 4 cycles per second, and scarcely any alpha rhythm, but occasional abnormally slow delta rhythm which might be hypothalamic in origin. Further investigations are shown in the table.

She was given 5% dextrose with hydrocortisone and L-thyroxine intravenously. The plasma sodium fell to 150 mEq/l. Her 24-hour urine output was 6-8 l. but this fell to 1·5 l. (specific gravity 1030) when she was given a daily dose of 5 units of pitressin tannate in oil intramuscularly. Her blood pressure rose to 110/80 mm Hg.

On 18 October she suddenly lost consciousness and had an epileptic fit. She recovered consciousness partially but developed right-sided hemiplegia. Her serum sodium gradually rose from 140 to 178 mEq/l., while her urine 24-hour output varied between 2 and 3·5 litres. She died in coma on 1 November 1971.

DR. DALY: I want to show two slides of the features and build (fig. 1) of this patient, weighing 38 kg a few days after admission. Without more ado I will ask Dr. Nabarro to introduce the case to us.

Clinician's Comments

DR. NABARRO: If we look at this girl's history, it certainly suggests that she was suffering from anorexia nervosa. We see, for

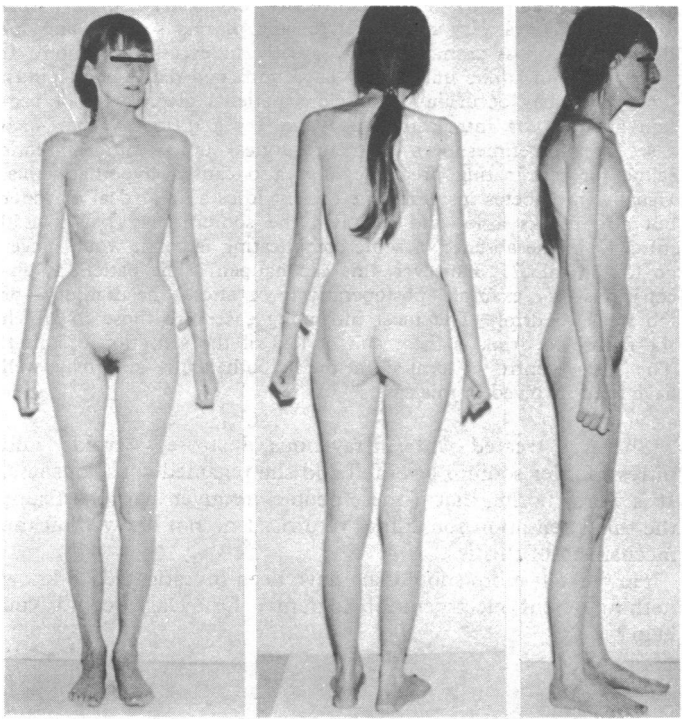

FIG. 1—View of patient a few days after admission (38 kg).

example, that she was obsessed by her weight, though her normal average weight was not really unreasonable. She had been interested in food production—the course in domestic science—a very common feature in anorexia nervosa. Moreover, her condition markedly deteriorated after the emotional shock of learning that her father had inoperable carcinoma. On the other hand, there are some unusual features in this history. The first is that at one stage she admitted to vomiting after every meal. Now patients with anorexia nervosa, particularly those who apparently have a good appetite, do in fact usually vomit—they may make themselves sick—but they do not tell anybody. The second thing which is unusual is that this photograph of her showed a remarkable degree of pallor and lack of pigmentation. Although she is thin, she is not as emaciated as most patients with anorexia nervosa are when they reach the comatose state. Finally, we are not told if she had the characteristic downy hair?

DR. DALY: No, that physical sign was not present.

DR. NABARRO: Then she started to get very thirsty. Her employer did not find any sugar or protein in her urine. What caused the thirst? Diabetes mellitus was apparently excluded and later ruled out by the investigations. Had she hypercalcaemia?—this too is ruled out later. Had she renal failure?—which might explain her weight loss and amenorrhoea—but the later blood urea concentration is not compatible with it. The last two possibilities were diabetes insipidus and hysterical polydipsia and these must remain possibilities—although, from what happens later, I believe there can be little doubt that she had true diabetes insipidus.

Now we come to 21 September, when she was admitted in coma. I think we could justly ask about the onset of coma?

PATTERN OF COMA

DR. DALY: It occurred over a matter of hours. She had continued to work to within a week of admission and had become drowsy the night before admission. She was admitted semiconscious and within an hour or two was unconscious.

DR. NABARRO: She was not only in coma but also markedly hypothermic. Had she taken some sedative or hypnotic drug?

DR. DALY: No. We looked for barbiturates and aspirin but found none.

DR. NABARRO: The next thought is was she hypoglycaemic? But the first blood sugar result was 65 mg/100 ml. What about meningitis? But the result of the lumbar puncture did not suggest this. One of the first things that was done was a chest *x*-ray film and I will ask Dr. Powell to comment on it.

DR. T. POWELL (3): It is a film taken supine in the ward, which tends to magnify the heart diameter but here the heart is still small. She has areas of poorly defined shadowing in the right upper zone—but no specific signs, no air bronchogram, no cavitation, no changes at the hila. We thought the shadows were of infective origin—perhaps aspiration secondary to coma or possibly tuberculous associated with meningitis—but there is no evidence of miliary tuberculosis. The appearance could have been due to collagen disease. The second film, taken some weeks later, shows clearing of the changes in the right upper zone. This lateral film of the skull is normal.

DR. DALY: The chest infection was thought to be staphylococcal and she was given both cloxacillin and penicillin in large doses for about a week.

DR. NABARRO: Thank you. That seemed to have cleared her pneumonia pretty efficiently. Now we will get on to the investigations that were done (see table I).

There is an alarming plasma sodium level, potassium a little low, blood urea a little raised; anaemic with some microcytosis; white cell count not striking; a very much raised sedimentation rate, but this falls later and may be attributed partly to her pneumonia. So we have a very gross hypernatraemia, but I will leave discussing this until later.

TABLE I—*Results of Laboratory Tests*

Blood	
Plasma Sodium	185 mEq/l.
Plasma Potassium	3·1 mEq/l.
Plasma bicarbonate	27 mEq/l.
Blood Urea	66 mg/ml
Haemoglobin	10·6 g/100 ml
W.B.C.	10,000/c.mm
Platelets	70,000/c.mm
E.S.R.	90 mm in 1 hour (Westergren)
Blood glucose	65 mg/100 ml
Cholesterol	126 mg/100 ml
P.B.I.	2·7 ug/100 ml
Serum Calcium	8·3 mg/100 ml
Serum Albumin	3·0 g/100 ml
Serum Globulin	4·1 g/100 ml
Plasma cortisol	8·4 ug/100 ml
24-hr Urinary Steroid Excretion:	17-ketosteroids, 1·5 mg/day 17-hydroxycorticosteroids, 2·3 mg/day. oestrogen, 17·7 µg/100 ml
Synacthen test	control plasma cortisol 5·3 µg/100 ml; 45 minutes after 1 mg Synacthen plasma cortisol 20·6 µg/100ml; 60 minutes after 23·3 ug/100 ml.
Cerebrospinal Fluid	
White cells 38/mm³. Lymphocytes 95%, Polysatu	
Red cells 7/mm³	
Protein 70 mg/100 ml	
Sugar 55 mg/100 ml	
Lange 0000000000	
Gram and Ziehl-Neilsen films: no organisms seen	

I think there are two possible explanations for her coma. It could have been due to her hypernatraemia, which does dehydrate the brain cells. Alternatively if—as I hope to show—this girl had hypopituitarism, the development of pneumonia may precipitate coma, and the hypothermia would be a feature of it.

The cerebrospinal fluid findings exclude a meningitis, although the white cells are increased. The sugar is present in reasonable concentration.

Additional results include the protein-bound iodine, which is a bit low, but patients who are starved, including patients

with anorexia nervosa, have a low level. If that was all we had we could not say she had hypothyroidism. The plasma cortisol (taken at midday the day after admission) was 8·4 μg/100 ml; one wonders if that is as much cortisol as she ought to have had in her circulation when she was severely stressed. The Mattingly technique gives rather higher readings than the protein-binding methods and this is rather low for her condition. The urine steroids are obviously very low; they indicate some disturbance of hypothalamic/pituitary or adrenal function. The Synacthen test indicates that the adrenal cortex was able to respond well to ACTH, so this puts the trouble back into the hypothalamus or the pituitary. Her oestrogen levels are pretty high for a girl who has amenorrhoea either from anorexia nervosa or from hypopituitarism.

In retrospect it is easy to ask for extra tests. There are other hormones made in the pituitary which we ought to look at—for example, her plasma luteinizing hormone, and her growth hormone production and growth hormone reserve. Low growth hormone levels may occur in a normal person, so one has to stimulate the production of more growth hormone, either by insulin hypoglycaemia or by other techniques, and see if the patient has a normal growth hormone reserve.

I believe, from the pallor of the girl on the first slide (fig. 1), the diminution of body hair, and the results of these investigations, that she was suffering from hypopituitarism. Was it hypothalamic or pituitary in origin? The radiograph of the skull showed a normal pituitary fossa; this does not exclude a destructive lesion of the pituitary, but it does make one wonder about a primary lesion in the hypothalamus. As this diagram (fig. 2) shows, we should not think of the hypothalamus and pituitary as a single unit.

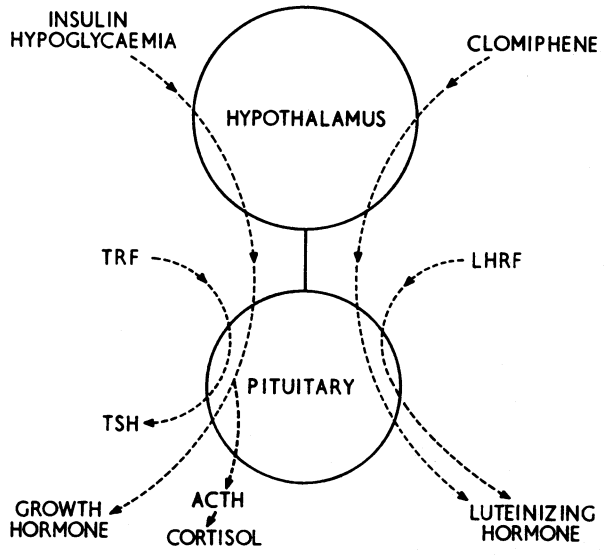

FIG. 2—Diagram showing separate functions of hypothalamus and pituitary glands.

The insulin hypoglycaemia test with measurements of growth hormone, and ACTH, or cortisol do not distinguish between a hypothalamic or pituitary lesion. Recent developments allow a stimulus to be put between the two, using either synthetic thyrotropin-releasing factor or synthetic luteinizing-hormone releasing factor. These will test whether the pituitary part of the hypothalamic pituitary axis is intact. In this way it is now possible to differentiate quite clearly between hypothalamic and pituitary disturbance. I believe this girl had diabetes insipidus and that would localize her trouble to the hypothalamus.

To lead into this question I would like to return to her hypernatraemia and refer to a comprehensive review article by Drs. Eric Ross and Christie of University College Hospital.[1] Now 185 mEq/l. is a very high level for the plasma sodium, and indicates water loss from the body greatly in excess of sodium. It is very rare in adults unless they have untreated diabetes insipidus. This level may occur in unconscious patients given a lot of protein and salt by intragastric tube and I regret to say that such cases are sometimes seen in neurosurgical units. Intra-amniotic saline for terminating pregnancy has also caused hypernatraemia. Usually in diabetes insipidus the patient loses a great deal of water but also drinks a lot and although the sodium may rise to 150 mEq/l., in the absence of some complicating factor it would never go to 185 mEq/l. However, this can happen if the patient is unconscious—for example, postoperatively or after a head injury—or too weak to drink. The most intriguing cases are those in which the failure to drink is the result of loss of the sensation of thirst. The thirst centre is located in the hypothalamus and may well have been involved in this case.

She was treated with intravenous dextrose, steroids, and pitressin. Her sodium level fell and she regained consciousness. It is worth noting that if these people are given hydrocortisone the thirst sensation sometimes returns. I do not know what the mechanism of this is.

How much more should she have been investigated? I know nothing about electroencephalograms; Dr. Daly, could you help?

FURTHER INVESTIGATIONS

DR. DALY: Our expert, Dr. J. Gumpert, thought that he could not pin down any abnormal locus but he felt that if there were trouble it was most likely to be hypothalamic. She was still very ill when it was done.

DR. NABARRO: I believe a brain scan was done. Would Dr. Powell like to comment?

DR. POWELL: It was not of very good quality. Pertechnicate was used. The result was thought to be within normal limits.

DR. NABARRO: I think I would have done another lumbar puncture at this stage with a specific request to the cytologist to look for malignant cells in the C.S.F. Arteriography and air studies obviously depended on decisions on the spot and I gather that she was really too ill for these to be done.

To conclude, I have to discuss the location of the lesion and its nature. I believe that this girl had some infiltrating condition of her hypothalamus. In 165 cases of lesions in this area which I reviewed some years ago there were 29 with suprapituitary lesions—six craniopharyngiomas, three who had had tuberculous meningitis in childhood, with calcification and destruction of the pituitary stalk; three had this curious infiltrating tumour of the hypothalamus, sometimes called an atypical pinealoma, sometimes an atypical teratoma; and a few with miscellaneous midbrain tumours, intracranial hypertension, collagen disease, trauma, Lawrence-Moon-Bied syndrome, and Hodgkin's disease. In view of the absence of any radiological abnormality I think this girl probably had the condition known as atypical pinealoma.

I had some anxiety about giving this as my firm diagnosis because she was anaemic, thrombocytopenic, had a raised sedimentation rate, and a high globulin (4·1g/100 ml). However, one of my cases of pinealoma had all these features except thrombocytopenia. Other things described as giving anterior and posterior pituitary deficiency are sarcoidosis, Hand-Schüller-Christian syndrome and arteritis. These are all possible but I would back rather strongly the atypical pinealoma.

Questions From Audience

DR. DALY: I am sure there are members of the audience who would like to ask questions before the answer is revealed.

DR. A. M. COOKE (4): The picture of the whole girl looked very pale but the pictures of her face were pigmented. Was there any pigmentation elsewhere to suggest Addison's disease?

DR. DALY: There was no pigmentation elsewhere. Dr. Ian Sneddon was asked to see her because she had a curious blue heliotrope discolouration of her upper eyelids.

DR. J. W. D. BULL (5): Why was she thought to be too ill to have an air encephalogram? Because Dr. Nabarro suggested a number of space-occupying masses just above the sella and in the region of the third ventricle where anything more than half a centimetre in diameter should be demonstrable by putting about 10 ml of air up by the lumbar route.

DR. DALY: Our problem at that time was linked to her high serum sodium and we had difficulty in controlling her water balance. She went in and out of coma about six times a day, whenever the sodium went above 152 mEq/l., and was never fit enough.

DR. NABARRO: It is very interesting that she was so sensitive to her sodium level. It suggests that her initial coma was due to hypernatraemia rather than the hypopituitary type I suggested.

DR. T. M. C. PARSONS (6): Could I ask Dr. Nabarro about hypoglycaemic coma in patients who do have anorexia nervosa? I have the impression that this occurred in patients who continued with their disease into middle life, or can one expect to see it in the typical young girl of this sort?

DR. NABARRO: I would have thought it was very unusual in a young girl. It is pretty uncommon and I have seen it only in older patients, as you suggest.

MEMBER OF AUDIENCE: Would Dr. Nabarro comment on the terminal hemiplegia?

DR. NABARRO: I think it is difficult to know what happened then. I understand her optic discs and visual fields were normal. This worries me. The cases of atypical pinealoma I have seen have had visual disturbances, usually because of involvement of the optic radiation. This termination with fits and hemiplegia would be compatible with an infiltrating lesion in this area.

MEMBER OF AUDIENCE: What would Dr. Nabarro have thought of the actual presenting level of sodium of 185 mEq/l. if he had seen this patient then?

DR. NABARRO: I would have thought she had some hypothalamic disturbance and would have wanted a skull *x*-ray examination because I would have suspected a craniopharyngioma. I would have given intravenous dextrose and hydrocortisone, as was done, but I would have been giving pitressin at an earlier stage. A sodium of this level means diabetes insipidus and you will not get the sodium down until you do give some pitressin.

PLASMA OSMOLALITY

DR. E. J. ROSS (7): Why did her plasma osmolality not fall, though her plasma sodium fell from 180 to 140?

DR. DALY: The figures were checked several times. Also, in fact she had individual doses of pitressin soon after admission. At this stage we were giving it virtually continuously. Do you have an answer?

DR. ROSS: She may not have recovered as well as you expected because she was hyperosmolar all the time. Did the urea level go up?

DR. DALY: No, it fell after admission to normal levels.

MEMBER OF AUDIENCE: Dr. Nabarro, what was the clinical time-scale of the cases in the group of hypothalamic lesions you mentioned?

DR. NABARRO: The histories were all probably a bit longer than this girl's. But the difficulty is to know when her trouble really started. What was her growth pattern?

DR. DALY: At 14 she was 5 ft 5 in. (163 cm) and on admission she was 5 ft 7½ in. (168 cm).

DR. NABARRO: That dates her onset later than 14. Most cases of atypical pinealoma have a rather longer history than this.

MEMBER OF AUDIENCE: What about the E.S.R. in relation to the diagnosis?

DR. NABARRO: The initial E.S.R. was high because of the pneumonia and it dropped to about 30, which was not unreasonable in the circumstances.

PROFESSOR SIR CHARLES STUART-HARRIS (8): Could I ask about her appetite. Her original complaint was loss of appetite; it improved and she gained weight; and then she lost it again. It seems that this did not precede the onset of thirst.

DR. NABARRO: This is difficult; I thought perhaps she really did have anorexia nervosa at one time and this is why she lost her appetite at first. And later when the other process developed her appetite went away again.

DR. DALY: Thank you, Dr. Nabarro. I am going to ask Dr. Timperley to give us the answer.

Necropsy Findings

DR. W. R. TIMPERLEY (9): The slide (fig. 3) shows a sagital section of the brain. In the region of the hypothalamus is a greyish mass of tumour between the mamillary bodies and the optic chiasma, reaching up into the lateral wall of the third ventricle and into the lamina terminalis. The tumour is partly cystic and low-power histology (fig. 4) shows it to be composed of two cell types—one with a large vesicular nucleus and a moderate amount of cytoplasm, the other resembling lymphocytes. The high-power magnification shows these more clearly

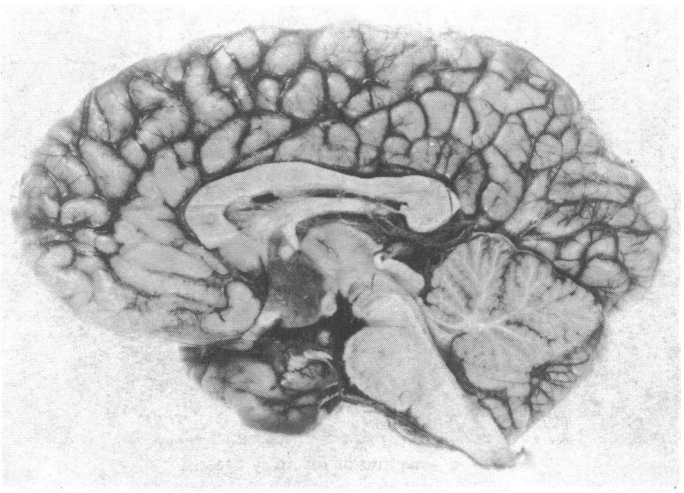

FIG. 3—Sagital Section of the Brain.

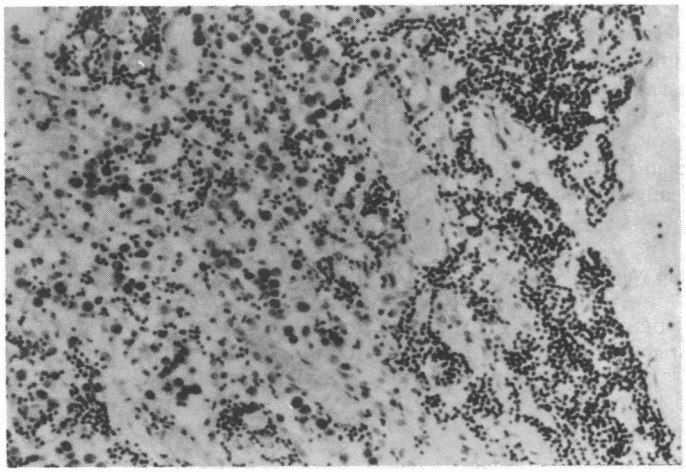

FIG. 4—Low-power histology of tumour.

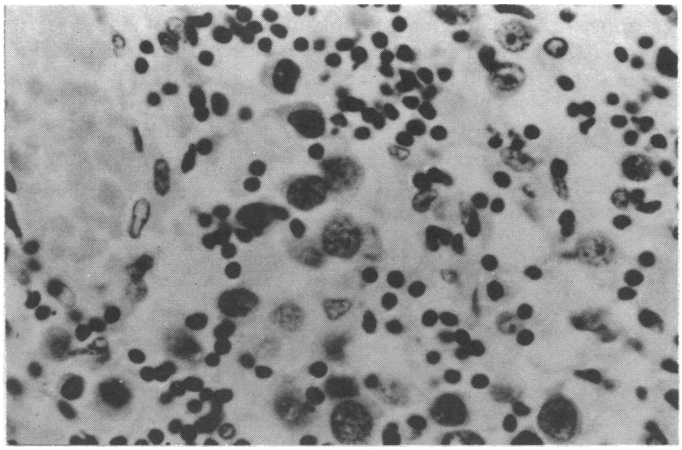

FIG. 5—High-power magnification of tumour.

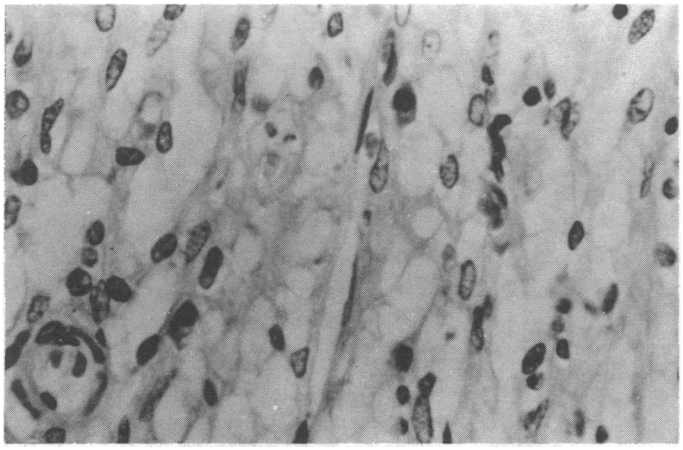

FIG. 6—Section of Pituitary Stalk.

(fig. 5)—this small cell is probably a lymphocyte, although some argue that it could be a neural blast cell. There are many mitotic figures, suggesting that it was actively growing. This is, of course, the typical histological picture of the tumour Dr. Nabarro finally decided on, the atypical pinealoma, seminoma, dysgerminoma, or atypical teratoma.

This section of the pituitary stalk (fig. 6) shows a rather vacuolated appearance and there is a slight infiltration of plasma cells, suggesting some atrophy of the stalk. The anterior pituitary appeared normal; the blood supply was presumably intact. The thyroid was slightly atrophied; many of the follicules were small; and there was some fibrosis. The ovaries apparently contained no corpora lutea but there were some cystic follicles so there must be some FSH activity. There were corpora albicantia, compatible with formation two years previously, after that they become fibrotic and shrunken. The uterus showed inactive endometrial glands with no evidence of cyclical activity. The adrenals weighed 8·5 g (normal 9-12 g) and the cortical thickness was slightly reduced. The vertebral bone was somewhat osteoporotic, probably owing to her low food intake.

Discussion

DR. DALY: I would like to congratulate Dr. Nabarro for being right on the button.

DR. BULL: Could I ask about the pineal gland itself. It is about 4 cm away from the site of the tumour, so how does the tissue get there?

DR. TIMPERLEY: We sectioned the pineal gland itself all the way through because there is an argument about this tumour—is it pineal or is it part of a teratomatous process. Occasionally one finds a small focus of neoplastic cells in the pineal gland. In this case we did not find any tumour cells within the pineal gland, which is not uncommon. These tumours arise in a variety of sites—including the hypothalamic floor, and tectum of the midbrain. So far no-one has described any pineal cells normally in these sites.

The tumour does, however, resemble the type of tumour which arises within the pineal gland and also resembles the pineal gland during embryogenesis. Several people have found hydroxyorthomethyl-o-transferase in atypical pinealomas.[2] It is involved in the production of melatonin.

This hypernatraemia is also interesting. There has been at least one case of ectopic pinealoma with marked hypernatraemia in the absence of diabetes insipidus.[3] I wondered what has happened to the story about the pineal gland producing a hormone, adrenoglomerulotrophin, involved in aldosterone production, and whether this could be a hormonally active tumour. In the mammal melatonin plays some part in the day/night regulation of gonadotrophin and thyroid hormone output. If you inject melatonin into animals you get a reduced gonadotrophin output, reduced thyroid cell height, and reduced TSH output. So if this were a hormonally active tumour arising from pineal cells, one might expect some of these hormonal changes to be due to the tumour itself.

TABLE II—*Histochemical Characteristic of Cells from Pinealoma, Testicular Seminoma, and Normal Pineal*

Enzyme	Pinealoma		Seminoma		Normal Pineal Gland	
	Large cells	Small cells	Large cells	Small cells	Pineocytes	Glia
Dehydrogenases						
6 Phosphogluconate	+	+	+	+	+	+
G-6-Phosphate	+	+	+	+	+	+
Glycerophosphate	+	+	+	+	+	+
Lactic Acid	+	+	+	+	+	+
Isocitric	+	+	+	+	+	+
Succinic	+	+	+	+	+	+
Malic	+	+	+	+	+	+
Glutamic	+	+	+	+	+	+
β-Hydroxy-butyric	+	+	+	+	+	+
DPNH diaphorase	+	+	+	+	+	+
TPNH diaphorase	+	+	+	+	+	+
Cytochrome oxidase	+	+	+	+	+	+
Acid Phosphatase	+	+	+	+	+	+
Esterase	+	+	+	+	+	+
Leucine Aminopeptidase	0	0	0	0	0	0
Alkaline Phosphatase	+	0	+	0	0	0

The other story is that the tumour may be a teratoma and arise from germinomatous cells which tend to occur in the midline—mediastinum, ovaries, testis, and so on. Many of these tumours have teratomatous elements such as cartilage, muscle, or thyroid—but this one did not. Table II shows a histochemical comparison between cells from a pinealoma, seminoma of the testis, and normal pineal gland. Both tumours contained alkaline phosphatase in the large cells. None was present in cells of the normal pineal gland. This is not a common enzyme and is usually associated with specific cell function. It is probably not a chance finding. So the argument remains unresolved.

DR. DALY: It simply remains for me to thank Dr. Nabarro for his masterly analysis and correct conclusion, and the other participants.

APPOINTMENTS OF SPEAKERS

(1) DR. J. J. DALY, M.D., M.R.C.P., Consultant Physician, United Sheffield Hospitals
(2) DR. J. D. N. NABARRO, M.D., F.R.C.P., Consultant Physician, Middlesex Hospital, London
(3) DR. T. POWELL, M.B., F.F.R., Consultant Radiologist, United Sheffield Hospitals
(4) DR. A. M. COOKE, D.M., F.R.C.P., Honorary Consulting Physician, Radcliffe Infirmary, Oxford
(5) DR. J. W. D. BULL, M.D., F.R.C.P., Consultant Radiologist, National Hospital for Nervous Diseases, London
(6) DR. T. M. C. PARSONS, M.A., M.B., Assistant Lecturer, Bland Sutton Institute, Middlesex Hospital, London
(7) DR. E. J. ROSS, M.B., F.R.C.P., Reader in Medicine, University College Hospital, London
(8) PROFESSOR SIR CHARLES STUART-HARRIS, M.B., F.R.C.P., Professor of Medicine, University of Sheffield
(9) DR. W. R. TIMPERLEY, D.M., M.R.C.PATH., Consultant in Neuropathology, United Sheffield Hospitals

This conference was recorded and edited by Dr. W. F. Whimster.

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Occasional Survey

Fibreoptic Endoscopy and the Barium Meal—Results and Implications

P. B. COTTON

British Medical Journal, 1973, 2, 161–165

Introduction

Although gastroscopy has been practised in a few centres for many years, the recent introduction of fibreoptic instruments has greatly enhanced the range and ease of examinations, and is transforming the clinical practice of gastroenterology. With the correct instruments and training, it is not difficult to examine and take specimens for biopsy from the entire oesophagus, stomach, duodenum (and colon) of conscious relaxed patients, on an outpatient basis. While only three to four years ago these new instruments could be regarded as expensive tools of mainly research interest, their diagnostic yield has led to rapidly increasing application throughout the country. In St. Thomas's Hospital the demand in the past two years for routine examination has increased from five to 25 a week. Our early experience of fibreoptic endoscopy[1] was obtained using instruments which have rapidly been superseded. The instrument field is now more stable.[2] Routine upper gastrointestinal endoscopy must involve examination of the oesophagus, stomach, and duodenum, at least as far as the duodenal bulb. To ensure an adequate survey in all patients, it is necessary to have available both a forward viewing fully flexible panendoscope (ACMI Model 7089P or Olympus Model GIFD) and a lateral viewing duodenoscope (Olympus Model JFB). Although Salmon *et al.*[3] have compared the use of forward and lateral viewing optical systems in the duodenal bulb, no clinical series is available concerning the overall results of properly equipped routine oesophagogastroduodenoscopy.

This paper reviews our recent experience of upper gastrointestinal fibreoptic endoscopy in relation to radiology to try to identify the cases in which endoscopy is of particular diagnostic value. It also discusses the practical problems of providing widespread endoscopy services.

St. Thomas's Hospital, London SE1 7EH
P. B. COTTON, M.D., M.R.C.P., Senior Medical Registrar

Patients and Methods

During the year October 1971 to September 1972, 956 patients underwent upper gastrointestinal fibreoptic endoscopy in our unit on a total of 1,020 occasions. Those endoscoped for cannulation of the papilla of Vater are excluded from this total. Patients' ages ranged from 3 months to 86 years. The only contraindication to examination was the possibility of transmitting infection (Australia antigenaemia or active tuberculosis), since endoscopes cannot be sterilized.

With modern forward-viewing instruments endoscopy need not be preceded by a standard barium-meal examination. Nevertheless, this is still the most usual routine sequence, and radiological reports provide the main indications for endoscopy (Table I). Since part of the purpose of the study was to compare radiology with endoscopy only patients who had had a barium-meal examination within one month of endoscopy were included. Results of endoscopy in patients with acute bleeding and in