both, is still lacking, there is enough evidence to require further investigation of the subject.

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Syndrome of inappropriate antidiuretic hormone secretion

SIR,—I was interested to read the case report by Drs C Haslett and N J Douglas (23-30 December, p 1753). Despite its title of "Inappropriate antidiuretic hormone secretion associated with neurohypophyseal choristoma," the information with which we are provided is insufficient for diagnosis of the syndrome of inappropriate ADH secretion.

The most important omission is a description of the patient's fluid balance—a consideration which must be central to any discussion of the cause of hyponatraemia. It is also important to know about the urinary osmolality and electrolytes before water deprivation. The initially low plasma osmolality is entirely predictable from the figures we are given for plasma electrolytes (given the absence of circulating "hidden osmoles") and is of little use taken on its own.

Assuming that the patient's fluid balance was compatible with the syndrome of inappropriate ADH secretion, then the biochemical data with which we are provided merely indicates an inability to excrete a water load. This kind of defect can also be found in hypopituitarism and hypothyroidism. One wonders whether the alternative explanations way had been excluded.

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*.*We sent a copy of this letter to the authors, whose reply is printed below.—ED, *BMJ*.

SIR,—We agree with most of the points made by Dr Jenkins, but in our original case report it was necessary to omit some details in the interest of brevity (600 word limit on *BMJ* short case reports). It was not possible to discuss each of the many criteria^{1,2} necessary for the diagnosis of the syndrome of inappropriate ADH secretion or to exclude individually the numerous causes of the syndrome.³

As regards the patient's fluid balance, there was no clinical evidence of salt and water depletion or overload and no evidence of underperfusion. The patient was not on any diuretic agent which might have interfered with free water formation. We did not measure urine osmolality on the night of admission. This was done the next morning after a period of therapeutic fluid deprivation as we felt that her state of altered consciousness was at least in part related to the low plasma sodium.

However, we are aware that prolonged fluid deprivation might cause volume-mediated ADH release with concentrated urine and hypotonic plasma; but there was no evidence of volume depletion on admission and the effect of a few hours of fluid restriction would be negligible.

We cannot exclude abnormality of pituitary, thyroid, or adrenal function on biochemical evidence because the patient's condition suddenly deteriorated 18 hours after admission while she was still being investigated; and when she subsequently returned to our unit for terminal care it was felt that she should not be further investigated but allowed to die in dignity. However, it seems extremely unlikely that hypoadrenalism was a factor as the patient had a normal blood pressure, both lying and sitting; the plasma potassium concentration was low; and the pituitary and adrenals were normal at necropsy. Similarly, hypothyroidism seems unlikely as she was not clinically hypothyroid and, indeed, had a pulse rate of 120 per minute on admission.

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Bleeding massive gastric ulcer on diflunisal (Dolobid)

SIR,—I was interested to read Dr B B Scott's letter (17 February, p 489), in which he described the case of a patient suffering from a massive bleeding gastric ulcer while on diflunisal (Dolobid). The following report is of a similar experience with a patient taking diflunisal.

This patient was a 79-year-old widow who suffered from longstanding back pain due to severe osteoarthritis of her lumbar spine. In 1968 she had been admitted to hospital having vomited about half a litre of fresh blood. There was no previous history of indigestion, but at the time she was taking aspirin, phenacetin, and codeine tablets for her backache. A barium meal showed a small sliding hiatus hernia with reflux, but no evidence of ulceration. She was given two units of blood and made an uneventful recovery, with no subsequent indigestion.

However, she continued to suffer from backache, for which various analgesics were prescribed over the years. These included paracetamol, phenylbutazone, ibuprofen, ketoprofen, and pentazocine, none of which caused symptoms of gastric irritation. In April 1978, with a worsening of her back pain, she was started on diflunisal 250 mg twice daily with food. After five weeks at this level, and with no symptoms related to her gastrointestinal tract, the dosage was increased to 500 mg twice daily. She was also taking Navidrex K (cyclopenthiiazide and potassium chloride), one tablet each morning, for hypostatic oedema of her legs; but she had received this preparation over the previous four years without any problems. Two weeks after starting the higher dose of diflunisal, she vomited over three-quarters of a litre of fresh blood and was admitted to hospital. She then admitted that she had started to suffer from epigastric pain, nausea, and anorexia almost immediately after the dose of diflunisal had been increased, but continued with the drug.

On admission to hospital she was pale but not shocked, and her haemoglobin concentration was 7.9 g/dl. The diflunisal was stopped, and she was given three units of blood. Over the course of the next 10 days her haemoglobin rose to 11.4 g/dl, her faecal occult blood tests became negative, and her indigestion disappeared. A barium meal showed thickened gastric rugae on the greater curve of the stomach, but no actual ulceration was seen. Her subsequent progress has been uneventful, although her backache persists.

As with the case described by Dr Scott, all the indications here are that the diflunisal was the cause of her haematemesis. The makers of Dolobid recommend caution when this product is prescribed for patients with a previous history of gastrointestinal haemorrhage, and this case should serve to underline this point.

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Assessment of renal concentrating ability

SIR,—We were interested to read the paper by Dr J R Curtis and B A Donovan (3 February, p 304) on the assessment of renal concentrating ability and would like to make the following comments.

The assessment of concentrating ability in the ambulant patient by the measurement of osmolality on aliquots of all urine specimens passed in a normal 24-hour period is an attractive possibility but is time consuming by comparison with the shorter intranasal desmopressin tests previously described¹⁻³ and is, as the authors state, not certain of success. In addition, we were surprised at the dosage of 4 µg desmopressin in the intramuscular test since we have previously demonstrated achievement of maximum osmolality with half this dosage intramuscularly in a shorter overnight test.²

We feel that the short intranasal desmopressin test provides the most convenient method of assessing renal concentrating ability. When investigating polyuria it must obviously be preceded by some form of water deprivation test. In this context control observations of urine osmolality throughout 24 hours may be unhelpful if excessive water drinking forms part of the differential diagnosis.

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SIR,—We have reported on the use of the deamino-D-arginine vasopressin (desmopressin) test (25 March 1978, p 757). The intranasal desmopressin test appeared to be as good as the pitressin test for measurement of the maximal urinary concentration. We would like to add a few comments.

We suspected that technical problems might arise with the intranasal administration of the drug. The correct administration is, of course, a very crucial point in the procedure. We have therefore tested intravenous administration of an equivalent dose—that is 4 µg, as it is held that 10% of the 40 µg given intranasally is