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We may return unduly long letters to the author for shortening so that we can offer readers as wide a selection as possible. We receive so many letters each week that we have to omit some of them. Letters must be signed personally by all their authors. We cannot acknowledge their receipt unless a stamped addressed envelope or an international reply coupon is enclosed.

Oral temperature and hypothermia

SIR,—Dr James Carne (10 March, p 690) should not be surprised that in a majority of his elderly patients attending surgery during the recent cold winter weather oral temperatures were 35°C or below. He was not observing hypothermia in these patients but the effect of a cold external environment on the temperature of the tissues inside the mouth. Oral temperature readings are recognised to be notoriously misleading when taken in either low or high ambient temperature conditions.

In a recent series of investigations on 16 young adults aged 20-40 years and 16 elderly people 70-85 years of age sitting comfortably (wearing normal indoor winter clothing) in a temperature-controlled room, five-minute oral temperature readings measured by a thermistor decreased progressively, in both groups, approaching 35°C in the elderly at the end

of two hours in 12°C ambient air conditions. Similarly, lagged (but not servo-controlled) ear temperatures measured by thermistor in the external auditory canal decreased significantly at 12°C, but there was no significant change in urine temperature taken at the beginning and end of the experiment.

The diagnosis of hypothermia must be based on measurement of the deep body temperature. This can be carried out by recording urine temperature, as has been shown in domiciliary studies in the elderly.¹

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¹ Fox, R H, et al, *British Medical Journal*, 1973, 1, 200.

Is hyperthermia a teratogen?

SIR,—We wish to make several comments regarding the recent article (9 December, p 1586) entitled "Is hyperthermia a teratogen?" There are different causes of raised body temperature, and physiological responses evoked by fever are quite different from responses to hyperthermia (such as that induced in a sauna bath). Most investigators in thermoregulation consider that fever results when

deep body temperature matches a raised thermoregulatory "set point," induced by pyrogen. In hyperthermia, the set point is lower than body temperature.¹ The significance of the physiological differences between fever and hyperthermia has recently been discussed elsewhere.²

The article in question did not differentiate between fever and environmental hyperthermia,

nor did it consider the possibility that raised body temperature in these two different physiological states might produce different pregnancy-related problems (ranging from abortion to learning disabilities).

The article also left an impression that lesions either develop exclusively as a consequence of "hyperthermia" or are exclusively due to some other cause. We suggest, and have reported elsewhere,³ that fever may exacerbate developmental problems caused by another agent. These effects might then be expressed by increased severity of a syndrome characteristic of the primary teratogenic agent. Preliminary data from our laboratory indicate that in rabbits exposure to rubella vaccine and prostaglandin-induced fever produces fewer young, reduced body weight, and heart and dental defects. Controls had normal litter-size and weight and no defects.

For the following reasons it seems that comparing the incidence of microphthalmia or anencephaly, or both, in young born in Finland (where saunas are taken frequently) with the incidence of these defects in other countries cannot be used to either support or deny the proposition that hyperthermia is a teratogen. Firstly, the short period of maternal hyperthermia developed in a sauna may not be severe enough to cause easily detected birth defects in healthy humans. The 1.6-2°C increase in rectal temperature for less than two hours due to sauna exposure⁴ is near the minimum reported to cause defects in animals.^{5,6} Secondly, a high frequency of sauna bathing might, conversely, prevent implantation or cause early miscarriage, thereby drastically reducing the number of malformed fetuses brought to term. Thirdly, recording the incidence of manifestation of a syndrome attributed to febrile illness and then expecting to find a high incidence of that syndrome in a population of predominantly healthy individuals having relatively short exposures (as compared with defect-related fever of two days-one week⁶) to environmental hyperthermia does not seem valid.

We feel that although definitive evidence for the teratogenicity of fever or hyperthermia, or

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

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¹ Bligh, J, *Temperature Regulation in Mammals and Other Vertebrates*. Amsterdam, North Holland Publishing, 1973.

² Kluger, M J, *New England Journal of Medicine*, 1978, **21**, 117.

³ Suarez, W A, and Hunter, W S, *The Physiologist*, 1978, **21**, 117.

⁴ Sohar, E, et al, *Israel Journal of Medical Science*, 1976, **12**, 1275.

⁵ Edwards, M J, *Teratology*, 1969, **2**, 313.

⁶ Smith, D W, Clarren, S K, and Harvey, M A S, *Journal of Pediatrics*, 1978, **92**, 878.

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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¹ Barter, F C, and Schwartz, W B, *American Journal of Medicine*, 1967, **42**, 790.

² Rosenow, E C, Segar, E W, and Zehr, J E, *Mayo Clinic Proceedings*, 1972, **47**, 169.

³ Mendoza, S A, *Pediatric Clinics of North America*, 1976, **23**, 681.

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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¹ Aronson, A S, and Svenningsen, N W, *Archives of Disease in Childhood*, 1974, **49**, 654.

² Monson, J P, and Richards, P, *British Medical Journal*, 1978, **1**, 24.

³ Delin, K, Aurell, M, and Ewald, J, *British Medical Journal*, 1978, **1**, 757.

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