control may enable safe anticoagulant treatment to continue by reducing the dose of the oral anticoagulant for the duration of the new treatment (for example, in an influenza epidemic where prophylactic antibiotics must be given to bronchitics). If bleeding does complicate treatment with oral anticoagulants doctors may be unaware of how to deal with the problem. Vitamin K (fat soluble) given intravenously in a dose of 5-10 mg will reverse the effect of this form of anticoagulant treatment in three to eight hours, but both the delay and the rebound resistance to further treatment can be substantial problems: the resistance may persist for up to two weeks. An alternative—which can stop severe bleeding quickly—is to raise the concentrations of the affected coagulation factors by fresh-frozen plasma or factors II, IX, and X concentrate (obtainable commercially or through regional transfusion service centres).

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Water intoxication and oxytocin

Oxytocin infusions may cause water intoxication—an unusual but not rare occurrence. The first report,[1] in 1962, was of a patient who had received large doses of oxytocin to evacuate the uterus in a missed abortion. A review[2] in 1975 found 23 reported cases, but the condition is probably much more common than this suggests: small series in single hospital units have since been described from New York,[3] Leeds,[4] and Birmingham.[5] The problem is most likely to arise as a result of prolonged attempts to empty the uterus in missed abortion or midtrimester termination of pregnancy, but it has also been described after oxytocin infusion in patients with hydatidiform mole,[4] incomplete abortion,[6] and postpartum haemorrhage[7]; in induction of labour[8]; and even in the augmentation of spontaneous labour.[5]

The water intoxication causes a profound hyponatraemia, and this was originally attributed to haemodilution with infused water.[9] Since, however, the fall in the plasma sodium concentration is in some cases greater than could be explained on the basis of dilution alone other possibilities include an increased excretion of sodium in the urine[10] and a shift of sodium from extracellular to intracellular fluid.[4] Irrespective of the mechanisms, there seems to be a clear relation between symptoms and plasma sodium concentration. The critical range associated with the risk of fits is probably 120-125 mmol(mEq)/l.[4]

Though most patients had been given high-dose oxytocin infusions, the quantity of the infused fluid is more important than the oxytocin concentration. Even very low doses of oxytocin (2-5 mU/min) are antidiuretic, and this effect becomes apparent within 10-15 minutes of the start of the infusion.[11] Irrespective of the oxytocin concentration, however, in virtually all reported cases of oxytocin-induced hyponatraemia the patients have received more than 3·5 litres of infused fluid, usually 5% dextrose in water. In the only reported exception the patient also received very large quantities of buccal oxytocin.[12] Another factor contributing to hyponatraemia is the antidiuretic effect of the pethidine and morphine[13] commonly used for analgesia with oxytocin infusions.

Water intoxication usually presents with fits and loss of consciousness, but in some cases there may be preceding signs such as a rise in the venous pressure, bounding pulse, and tachycardia.[11] Alterations in levels of consciousness are difficult to spot in labour but have been retrospectively noted in some patients.[5] The diagnosis depends on recognition that the condition may be present. As it is unusual, other conditions such as eclampsia may initially be suspected; but the patient is usually normotensive and will not have proteinuria, though she may have oedema. The diagnosis is confirmed by measurement of the electrolyte concentrations in the blood.

Treatment consists of stopping the fits and ensuring adequate respiration, if necessary by means of artificial ventilation. The infusion of oxytocin and dextrose in water must be stopped. Isotonic saline should be slowly infused to return the serum sodium concentration towards normal, and this will be followed by a physiological diuresis, which may then be assisted by frusemide or other rapidly acting diuretics. Alternatively, hypertonic saline may be considered.[14]

The clinician's prime objective should be to prevent the condition. The advent of prostaglandins has reduced the need for prolonged oxytocin infusion. Risks may also be minimised by the judicious use of automatic infusion systems delivering relatively small quantities of fluid. Possibly an isotonic solution, such as Ringer's lactate, would be preferable to dextrose in water as a vehicle for oxytocin. Most important of all, however, is simple clinical accountancy. No patient should receive more than three litres of fluid containing oxytocin, and, in every case, a careful fluid balance record is essential.

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