PAPERS AND SHORT REPORTS

Iatrogenic hyponatraemia of the newborn due to maternal fluid overload: a prospective study

WILLIAM O TARNOW-MORDI, JONATHAN C L SHAW, DAVID LIU, DAVID A GARDNER, FREDERICK V FLYNN

Abstract

Over five weeks 136 out of 246 deliveries were studied. Maternal plasma sodium concentrations were normal at admission. At delivery no significant difference was found between maternal and infant cord plasma sodium concentrations. Twenty-four of the 41 mothers who had received only oral fluids during labour had infants whose cord plasma sodium concentrations were normal. Of the 95 mothers who had been given intravenous fluids, however, only 14 had infants with normal plasma sodium concentrations, 31 had a concentration of 130 mmol (mEq)/l or less and nine of these had a concentration of 125 mmol/l or less. There was a highly significant inverse relation between cord plasma sodium concentration and rate of fluid administration, suggesting that hyponatraemia was due to intravenous treatment with predominantly sodium-free solutions. Endogenous anti-diuretic activity probably increases during labour, and synthetic oxytocin in large doses has been shown to have an anti-diuretic effect. The dose used in this study did not appear to have such an effect. Glucose solutions are often used as a vehicle for oxytocin; 83% of all fluid intake in this study was 5% or 10% glucose in water.

Fluid balance in labour should be supervised closely, and oxytocin should be given in a more concentrated solution.

Introduction

During the last 20 years many reports have described severe water intoxication in women receiving infusions of glucose in water, with or without oxytocin, during pregnancy or the puerperium. Since 1962 cases have been described following treatment for missed, inevitable, and incomplete abortion,¹⁻⁸ postpartum haemorrhage,⁹⁻¹⁰ and termination of pregnancy,¹¹⁻¹² and during induction¹³ and augmentation¹⁴ of labour. Water intoxication and hyponatraemia with resultant cerebral oedema, coma, and convulsions have serious consequences. Three mothers have died,¹⁵⁻¹⁷ one suffered severe brain damage¹⁸ and another a visual deficit and dysphasia.¹² Since 1965 water intoxication and hyponatraemia have also been reported in the infants of such mothers, resulting in neonatal convulsions and respiratory problems.¹⁹ No long-term damage has been documented in these babies. Despite the publicity, iatrogenic hyponatraemia causing convulsions in both mother and infant is still occurring.²⁰⁻²²

We undertook this study after observing unexplained hyponatraemia in infants admitted to the neonatal unit at University College Hospital and unexpectedly large weight losses during the first 48 hours of life in otherwise healthy term infants in the postnatal wards. These findings suggested that a degree of water overloading in the infants was more common than we had suspected. The aim of the study was to define the incidence and major determinants of hyponatraemia in a population of newborn babies.

Patients and methods

The study covered the five weeks from 16 January to 22 February 1980. All mothers admitted to the delivery suite were eligible, and as many were entered into the study as time and circumstances on a busy labour ward permitted. Although no formal randomisation took place, the composition of the study group, when analysed according to features of labour and method of delivery, did not differ significantly from that of the other mothers admitted during the period of the study ($0.05 < p < 0.1$). Conclusions drawn from the 136 infants studied are therefore applicable to the group as a whole.

Samples of maternal blood were taken on admission and as soon as practicable after delivery, and samples of cord blood were collected at

Departments of Paediatrics, Obstetrics, and Chemical Pathology, University College Hospital, London WC1E 6AU

WILLIAM O TARNOW-MORDI, MRCP, DCH, senior house officer, neonatal unit (present appointment: paediatric registrar, Royal Berkshire Hospital, Reading)

JONATHAN C L SHAW, FRCP, DCH, consultant neonatologist

DAVID LIU, MPHIL, MRCP, lecturer in obstetrics

D A GARDNER, MRCPATH, consultant chemical pathologist

F V FLYNN, FRCP, FRCPATH, professor of chemical pathology

delivery. The plasma was centrifuged and separated in the labour ward and analysed in the department of chemical pathology. Sodium, urea, and total protein concentrations were measured using a Technicon SMAC analyser and osmolality with an advanced model 3D osmometer; in our laboratory the standard procedures used have between-batch coefficients of variation of 0.6%, 2.0%, 1.1%, and 0.9%. Details of the duration and type of labour and of the maternal fluid intake were recorded by midwives and attending medical students.

Tests of statistical significance were performed using the Student's *t* test, paired *t* test, and χ^2 test with Yates's correction.

Results

For the purpose of analysis the measurements have been divided into five groups according to certain aspects of management of the mother during labour (table I). The maternal plasma sodium concentration on admission (mean $\pm$ SD) was 137 ± 2.0 mmol(mEq)/l, and was not significantly different in the five groups of mothers. Since there was no significant difference on paired *t* testing between maternal plasma sodium concentration taken within 15 minutes of delivery and the umbilical cord plasma sodium concentration, only the cord plasma concentrations will be presented here.

Table I shows the distribution of umbilical cord plasma sodium concentrations of 136 infants at birth. Twenty-four of the 41 infants whose mothers received only oral fluids had a cord plasma sodium concentration from 136 to 145 mmol/l. The remaining 95, whose mothers all received intravenous infusions, included only 14 with a sodium concentration in the range 136–145 mmol/l. Thirty-seven mothers received epidural analgesia and intravenous fluids with

oxytocin; 16 had infants with a plasma sodium concentration of 130 mmol/l or less, six of these being 125 mmol/l or less.

Table II shows the relation between mean cord plasma concentrations of sodium and urea, mean rate of maternal fluid intake, and mean duration of labour. A least squares regression analysis of the ungrouped data from all deliveries for which details of fluid treatment were available (*n*=110) showed for both plasma sodium and urea concentrations a highly significant inverse linear relation with the rate of maternal fluid administration during labour (*p* < 0.001). Corresponding analyses of the correlations between the rate of fluid intake and the cord-blood plasma protein and osmolality measurements showed the same inverse relation for each, but the significance level was less impressive (*p* < 0.01). It is worth noting from table II that the mean rate of fluid intake in mothers who received epidural analgesia and intravenous oxytocin was 195 ml/h, equivalent to a daily infusion of 4.7 litres.

Solutions of 5% or 10% glucose in water accounted for 83% of the total volume of fluid given to all the mothers studied. Oxytocin, when used, was generally given in 5% glucose solution at an initial concentration of 4 U/l. The mothers who received intravenous infusions containing oxytocin but who were given no epidural analgesia had a mean rate of oxytocin infusion during the first and second stages of labour of 2.9 mU/min. Mothers receiving intravenous oxytocin with epidural analgesia had a mean rate of oxytocin infusion during the first and second stages of 4.4 mU/min. The rate of oxytocin administration was not significantly related to the umbilical cord plasma sodium concentration found subsequently in either group. The highest mean infusion rate of oxytocin during the first and second stages of labour was 17.5 mU/min, and occurred in one of the mothers given intravenous oxytocin and epidural analgesia.

TABLE I—Plasma sodium concentrations in cord blood of 136 infants subdivided according to management of the mother during labour

Type of management during labour	Plasma sodium in cord blood (mmol/l)				
	121–125	126–130	131–135	136–140	141–145
Oral fluids only (<i>n</i> =41)	0	2	15	20	4
Intravenous fluids only (<i>n</i> =19)	0	2	11	5	1
Intravenous fluids + oxytocin (<i>n</i> =24)	1	5	14	4	0
Intravenous fluids + epidural analgesia (<i>n</i> =15)	2	5	6	2	0
Intravenous fluids + oxytocin + epidural analgesia (<i>n</i> =37)	6	10	19	2	0
Total	9	24	65	33	5
%	6.6	17.6	47.8	24.3	3.7

TABLE II—Comparison of mean cord plasma sodium and urea concentrations, mean rates of maternal fluid intake, and mean duration of labour, in the different maternal groups. Results are mean $\pm$ SEM

Type of management during labour	Plasma sodium in cord blood (mmol/l)	Plasma urea in cord blood (mmol/l)	Rate of maternal fluid intake (ml/hour)	Duration of labour (hours)
Oral fluids only (<i>n</i> =41)	136.1 $\pm$ 0.5	3.11 $\pm$ 0.10	14 $\pm$ 4	7.3 $\pm$ 0.7
Intravenous fluids only (<i>n</i> =19)	133.4 $\pm$ 1.0	3.05 $\pm$ 0.19	98 $\pm$ 21	11.3 $\pm$ 1.5
Intravenous fluids + oxytocin (<i>n</i> =24)	132.4 $\pm$ 0.8	3.03 $\pm$ 0.18	157 $\pm$ 19	8.9 $\pm$ 1.2
Intravenous fluids + epidural analgesia (<i>n</i> =15)	130.9 $\pm$ 1.0	2.99 $\pm$ 0.22	128 $\pm$ 19	11.1 $\pm$ 1.0
Intravenous fluids + oxytocin + epidural analgesia (<i>n</i> =37)	130.4 $\pm$ 0.6	2.97 $\pm$ 0.16	195 $\pm$ 20	13.8 $\pm$ 1.2

Conversion: SI to traditional units—Urea: 1 mmol/l $\approx$ 6 mg/100 ml.

TABLE III—Features relating to the nine infants born with plasma sodium of 125 mmol/l or less

Infant No	Plasma sodium in cord blood (mmol/l)	Length of first and second stages of labour (h min)	Volume of maternal fluid intake in labour (litres)	Type of maternal fluid management	Type of fluid given during first and second stages of labour	Mean rate of fluid intake (ml/h)	Mean rate of oxytocin infusion (mU/min)	Birth weight (grams)	Maximum recorded weight loss during first week (%)
1	125	14 05	4.0	Intravenous fluids + oxytocin	5% glucose + Hartmann's	284	3.8	3510	4.3
2	125	13 00	2.0	Intravenous fluids + epidural analgesia	5% glucose	154		3580	5.0
3	125	12 57	2.0	Intravenous fluids + epidural analgesia	5% glucose + Hartmann's	158		3180	6.3
4	124	7 06	1.2	Intravenous fluids + oxytocin + epidural analgesia	5% glucose	169	1.4	3300	6.1
5	125	2 50	1.7	" " " "	5% glucose	593	12.4	3280	4.9
6	122	27 01	4.6	" " " "	5% and 10% glucose	172		4100	10.7
7	125	8 00	2.0	" " " "	5% glucose	250		2880	6.9
8	122	8 24	2.2	" " " "	5% glucose	264	2.5	3060	11.1
9	123	19 00	5.0	" " " "	5% and 10% glucose + Hartmann's	263		3475	6.7
Mean	124	12 30	2.7			256	5.0		6.9

The maximum recorded weight loss (mean $\pm$ SEM) during the first week of life for infants of mothers given oral fluids only, was $3.6 \pm 0.2\%$; of those given intravenous fluids only, $5.5 \pm 0.4\%$; those given intravenous oxytocin infusions, $5.4 \pm 0.4\%$; those given intravenous fluids with epidural analgesia, $5.1 \pm 0.6\%$; and those given intravenous oxytocin infusion with epidural analgesia, $6.4 \pm 0.5\%$.

Table III gives details relating to the nine infants with a cord plasma sodium concentration of 125 mmol/l or less. Mean duration of labour was 12 h 30 min, and mean rate of maternal fluid intake in labour 256 ml/h, equivalent to an infusion rate of 6.1 l/day. Of the total maternal fluid intake in labour in this group, 5% glucose in water accounted for 83%, 10% glucose in water 6%, and Hartmann's solution 11%. Complete data regarding the rate of oxytocin administration were available in only four cases, and among these the mean rate of oxytocin infusion was 5.0 mU/min. Maximum recorded weight loss in the first week of life for these infants (mean $\pm$ SEM) was $6.9 \pm 0.8\%$. Significantly more of them (7 out of 9) had a maximum weight loss of 5% or more when compared with the infants whose mothers received oral fluids only and for whom weights were recorded (4 out of 20) ($p < 0.001$).

Discussion

This study shows a high incidence of hyponatraemia in both mothers and infants at the time of delivery. In animal studies²³ it has been shown that the plasma sodium concentration of the fetus is about 3 mmol/l lower than that of the mother when expressed per litre of plasma, but Battaglia *et al* (1960)²⁴ found no difference in man. We were unable to establish whether such small differences exist; many mothers were not in a steady state as they had infusions running, and maternal samples were often not taken at the moment of delivery. The lack of significance of the paired *t* test of maternal versus cord blood plasma sodium does not indicate that there is no difference but rather that cord plasma concentrations adequately represented maternal concentrations at delivery for the purpose of this study.

Since maternal plasma sodium concentrations in all groups were essentially normal on admission events during labour must have caused the hyponatraemia. Hyponatraemia could be due to the retention of water in the body in excess of sodium or to loss of sodium from the extracellular fluid compartment without a corresponding loss of water. Negative sodium balance probably played only a minor part because mothers who received only oral fluids had a rather low sodium intake, but most had normal or near normal plasma sodium concentrations at delivery. By contrast 81 of the 95 mothers who received intravenous fluids had infants with cord plasma sodium concentrations below the normal range—some dangerously so. The highly significant inverse relation between the rate of fluid administration and the plasma sodium concentration suggests that hyponatraemia resulted from water overload due to intravenous treatment with predominantly sodium-free solutions. The corresponding relations in the case of plasma urea and total protein concentrations, and osmolality provide confirmatory evidence for haemodilution. A normal adult can maintain water balance on 2500 ml/day or 100 ml/h,²⁵ but 68% of mothers in this study who received a similar volume gave birth to infants with plasma sodium concentrations below the normal range even though extrarenal losses would have increased as a consequence of the muscular effort of labour. These mothers must, therefore, have an impaired ability to excrete a water load during labour. This is consistent with the reported values for plasma antidiuretic hormone (ADH) concentration in labour,²⁶⁻²⁸ which, though rather variable, resemble quite closely the values found after overnight dehydration.²⁹⁻³⁰ There seem to be no data on the response of plasma ADH concentrations to water loading in labour, but the observed inverse relationship between cord plasma sodium concentrations and rate of maternal fluid intake suggests that plasma ADH does not respond normally to a water load in labour, presumably due to the overriding influences of pain, fear, exercise, and drugs which stimulate ADH release. Though detectable concentrations of endogenous oxytocin can be measured in labour³¹ little is known of the rate of secretion

so the role of endogenous oxytocin cannot be evaluated. According to Abdul Karim and Assali (1961)³² synthetic oxytocin given at 45 mU/min has an antidiuretic effect equivalent to vasopressin, and antidiuretic effects have been noted when the infusion rate exceeded 16 mU/min.³³ The doses of oxytocin given to augment labour in this group did not seem to exert an antidiuretic effect since in the two groups who received oxytocin alone and epidural analgesia alone there was no significant difference in the cord sodium concentrations or rate of maternal fluid intake. The data point to water overload as the principal cause of hyponatraemia.

The low plasma sodium concentration cannot, however, be accounted for entirely by the volume of water administered. This may result from the sodium diuresis of acute water intoxication shown by Wrong (1957)³⁴ to begin two hours after an intravenous fluid load and reach a peak after six to 10 hours. Another contributory factor in those receiving hypertonic glucose infusions could be the transfer of water from cells in response to the temporary raising of plasma osmolality. Movement of sodium into cells as a result of diminished activity of the sodium pump is a hypothetical possibility; we are, however, unaware of any evidence that it occurs other than in grave or chronic illness.

Glucose solutions are given to treat maternal ketoacidosis which, it is said, interferes with uterine action. This may have been necessary in the severe ketosis that used to be seen in prolonged labours, but we have found no data on the effect of ketosis on uterine action or on the value of glucose infusions in the treatment of the mild ketosis that is so common in normal labour today. Perhaps it is time the practice was reviewed. Glucose solutions are also given as a vehicle for oxytocin. The manufacturers of oxytocin (Syntocinon) describe its use in a concentration of 1.0 IU/l of 5% glucose.³⁵ At the maximum rate of infusion they recommend of 12 mU/min the patient would receive fluid at a rate of 17.3 litres/day. They rightly describe the dangers of water intoxication but do not suggest increasing the concentration of oxytocin to reduce the volume of fluid given. By administering more concentrated solutions via syringe pumps or microdrop infusion sets, this problem could be overcome. Finally, intravenous solutions are given to correct maternal hypotension encountered during epidural analgesia. For this purpose it is logical to use 0.9% saline or Hartmann's solution, but in our experience 5% glucose is sometimes mistakenly given. In the present study 83% of the solutions used were 5% or 10% glucose in water. If sodium-containing solutions had been used there would have been less hyponatraemia but this would have led to new dangers of hypertension and overload of the circulation. The remedy is less water not more sodium.

The consequences of water intoxication to the mother may be serious, and the reports of maternal deaths and permanent neurological disablement¹²⁻¹⁵⁻¹⁸ have already been mentioned. As the plasma sodium falls there is progressive cerebral oedema, with signs ranging from convulsions, coma, and psychosis to subtler manifestations such as headache, vomiting, lethargy, confusion, and mood changes.³⁶ Water intoxication may underlie the un-cooperative behaviour of some patients in labour and may account for the withdrawn or psychotic state of some mothers in the puerperium.³⁷ Convulsions or coma with hyponatraemia occurring in association with oxytocin infusion should be reported to the Committee on Safety of Medicines. A further consequence of water intoxication may be interference with normal myometrial activity in labour. There are no studies on the parturient myometrium, but hypo-osmolar solutions which cause cell swelling³⁸ have a profound effect in vitro on the spontaneous activity of vascular smooth muscle from the rat leading to depolarisation and tetany.³⁹

In the infant convulsions, apnoea, cyanotic spells, respiratory distress, and feeding difficulty, have all been attributed to hyponatraemia due to intrapartum water overload. Though the data are not conclusive, the excess weight loss after birth reported here may be another consequence of water overload.

We conclude that the fluid requirements of mothers in labour are, like those of postoperative patients, much less than those of normal adults, and fluid treatment should be modified accordingly. All intravenous fluids should be prescribed by a doctor and an input and output record kept by the midwives. The fluid requirements of most mothers would probably be met by an intake of about 50 ml/h (1200 ml/day). If this were given as 10% glucose in 0.18% saline it would provide 69 g glucose/m²/day, an amount shown to be sufficient to suppress ketonuria in starving subjects.⁴⁰ The aim of fluid treatment in labour should be to maintain strict water and electrolyte balance, and we suggest that if intravenous treatment is given maternal plasma electrolytes should be measured in labour.

This study was presented to the meeting of the Obstetric Anaesthetists' Association, Charing Cross Hospital, London, 1980. We are grateful to the midwives and medical students who patiently collected the data and to Mrs G Harris and Miss M Malacos for expert secretarial help. We thank Professors Fairweather and Brant for their co-operation.

References

- Liggins GC. Acute water retention associated with continuous slow infusion of oxytocin. *Journal of Obstetrics and Gynaecology of the British Commonwealth* 1962;**69**:277-81.
- Leventhal JM, Reid DE. Oxytocin induced water intoxication with grand mal convulsion. *Am J Obstet Gynecol* 1968;**102**:310-1.
- Pedlow PRB. Water intoxication induced with oxytocin. *Journal of Obstetrics and Gynaecology of the British Commonwealth* 1970;**77**:1113-4.
- Pittman JG. Water intoxication due to oxytocin. *N Engl J Med* 1963;**268**:481-2.
- Whalley PJ, Pritchard JA. Oxytocin and water intoxication. *JAMA* 1963;**186**:601-3.
- Silva P, Allan MS. Water intoxication due to high doses of synthetic oxytocin. *Obstet Gynecol* 1966;**27**:517.
- Self J. Water intoxication induced by oxytocin administration. *Am J Med Sci* 1966;**252**:573-4.
- Josey WE, Pinto AP, Plant RF. Oxytocin induced water intoxication. *Am J Obstet Gynecol* 1969;**104**:926.
- Bilek W, Dorr P. Water intoxication and grand mal seizures due to oxytocin. *Can Med Assoc J* 1970;**103**:379-82.
- Turcot MJP. Water intoxication induced by oxytocin. *Union Med Can* 1971;**100**:1997-2000.
- Ahmad AJ, Clark EH, Jacobs HS. Water intoxication associated with oxytocin infusion. *Postgrad Med J* 1975;**51**:249-52.
- Morgan DB, Kirwan NA, Hancock KW, Robinson D, Howe JG, Ahmed S. Water intoxication and oxytocin infusion. *Br J Obstet Gynaecol* 1977;**84**:6-12.
- Burt RL, Oliver KL, Whitener DL. Water intoxication complicating elective induction of labour at term. Report of a patient who survived. *Obstet Gynecol* 1969;**34**:212-7.
- Storch AS. Acute water retention associated with continuous slow infusion of oxytocin. *Obstet Gynecol* 1971;**37**:109-11.
- Lilien AA. Oxytocin induced water intoxication; report of a maternal death. *Obstet Gynecol* 1968;**32**:171-3.

- Gupta DR, Cohen NH. Oxytocin, "Salting out," and water intoxication. *JAMA* 1972;**220**:681-3.
- Ware S. Transplacental hyponatraemia due to oxytocin. *Br Med J* 1978;**ii**:362.
- Laurens NH, Birnbaum SJ. Water intoxication associated with oxytocin administration during saline induced abortion. *Am J Obstet Gynecol* 1975;**121**:2-6.
- Altstatt LB. Transplacental hyponatraemia in the newborn infant. *J Pediatr* 1965;**66**:985-7.
- Schwarz RH, Jones RWA. Transplacental hyponatraemia due to oxytocin. *Br Med J* 1978;**ii**:152-3.
- McKenna P, Shaw RW. Hyponatraemic fits in oxytocin augmented labors. *Int J Gynaecol Obstet* 1979;**17**:250-2.
- Burnell RH, Dahlenburg GW. Iatrogenic hyponatraemia in mother and infant. *Med J Aust* 1979;**2**:254.
- Weedon AP, Stacey TE, Ward RHT, Boyd RDH. Bidirectional sodium fluxes across the placenta of conscious sheep. *Am J Physiol* 1978;**235**:F536-41.
- Battaglia F, Prystowsky H, Smisson C, Hellegers A, Bruns P. Fetal blood studies. XIII. The effects of the administration of fluids intravenously to mothers upon the concentrations of water and electrolytes in plasma of human fetuses. *Pediatrics* 1960;**25**:2-10.
- Ruch TC, Patton HD, Scher AM. *Physiology and biophysics*. 20th ed. Philadelphia: W B Saunders, 1973:474.
- Hoppenstein JM, Mittenberger FW, Moran WH. The increase in blood levels of vasopressin in infants during birth and surgical procedures. *Surgery Gynecol Obstet* 1968;**127**:966-74.
- Czernichow P, Pattin A. Vasopressin in the newborn: Plasma levels in cord and maternal blood. *Annales d'Endocrinologie* 1978;**39**:225-6.
- Rees L, Forsling ML, Brook CGD. Vasopressin concentrations in the neonatal period. *Clin Endocrinol (Oxf)* 1980;**12**:357-62.
- Robertson GL, Mahr EA, Athar S, Sinha T. Development and clinical application of a new method for the radio immunoassay of AVP in human plasma. *J Clin Invest* 1973;**52**:2340-52.
- Skowsky WR, Rosenbloom AA, Fisher DA. Radio immunoassay measurement of arginine vasopressin in serum: development and application. *J Clin Endocrinol Metab* 1974;**38**:278-87.
- Chard T, Hudson CN, Edwards CRW, Boyd NRH. Release of oxytocin and vasopressin by the human fetus during labour. *Nature* 1971;**234**:352-3.
- Abdul-Karim R, Assali NS. Renal function in human pregnancy V. Effects of oxytocin on renal haemodynamics and water and electrolyte excretion. *J Lab Clin Med* 1961;**57**:522-32.
- Singhi S, Singh M. Transplacental asymptomatic hyponatraemia following oxytocin infusion during labour. *Indian J Med Res* 1979;**70**:55-7.
- Wrong O. The relationship between water retention and electrolyte excretion following administration of antidiuretic hormone. *Clin Sci* 1957;**15**:401-8.
- Sandoz Products. *Data sheet compendium*. Datapharm Publications, 1980: 958-9.
- Arieff AI, Llach F, Massry SG. Neurological manifestations and morbidity of hyponatraemia. Correlation with brain water and electrolytes. *Medicine* 1976;**55**:121-9.
- Burnell GM, Foster TA. Psychosis with low sodium syndrome. *Am J Psychiatry* 1972;**128**:1313-4.
- Johnsson O. Changes in cell volume of isolated vascular smooth muscle in response to reduced osmolarity. *Acta Physiol Scand* 1969;**77**:201-11.
- Johnsson O. Changes in the activity of isolated vascular smooth muscle in response to reduced osmolarity. *Acta Physiol Scand* 1969;**77**:191-200.
- Talbot NB, Richie RH, Crawford JD. *Metabolic homeostasis: a syllabus for those concerned with the care of patients*. Cambridge, Massachusetts: Harvard University Press, 1959. (Commonwealth Fund.)

(Accepted 2 July 1981)

WATER AGRIMONY is called in some countries Water Hemp, Bastard Hemp, and Bastard Agrimony, Eupatorium, and Hepatorium, because it strengthens the liver. The root continues a long time, having many long slender strings. The stalk grows up about two feet high, sometimes higher. They are of a dark purple colour. The branches are many, growing at distances the one from the other, the one from the one side of the stalk, the other from the opposite point. The leaves are fringed, and much indented at the edges. The flowers grow at the top of the branches, of a brown yellow colour, spotted with black spots, having a substance within the midst of them like that of a Daisy: If you rub them between your fingers, they smell like rosin or cedar when it is burnt. The seeds are long, and easily stick to any woollen thing they touch.

They delight not in heat, and therefore they are not so frequently found in the Southern parts of England as in the northern, where they grow frequently: You may look for them in cold grounds, by ponds and ditches' sides, and also by running waters; sometimes you

shall find them grow in the midst of waters. They all flower in July or August, and the seed is ripe presently after.

It is a plant of Jupiter, as well as the other Agrimony, only this belongs to the celestial sign Cancer. It heals and dries, cuts and cleanses thick and tough humours of the breast, and for this I hold it inferior to but few herbs that grow. It helps the cachexia or evil disposition of the body, the dropsy and yellow-jaundice. It opens obstructions of the liver, molifies the hardness of the spleen, being applied outwardly. It breaks imposthumes away inwardly: It is an excellent remedy for the third day ague. It provokes urine and the terms; it kills worms, and cleanses the body of sharp humours, which are the cause of itch and scabs; the herb being burnt, the smoke thereof drives away flies, wasps, &c. It strengthens the lungs exceedingly. Country people give it to their cattle when they are troubled with the cough, or broken-winded. (Nicholas Culpeper (1616-54) *The Complete Herbal*, 1850.)