FLUID THERAPY DURING PAEDIATRIC SURGERY*

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THE ABILITY of children to survive long surgical procedures depends largely on two factors—pulmonary ventilation and fluid balance. The essential features of adequate ventilation are generally recognized, but the principle of fluid therapy in children undergoing surgery is difficult to put into practical terms and is poorly understood.¹ The purpose of this study is an attempt to further the understanding of the problem of fluid therapy in children and to suggest some practical answers to the problems.

FUNDAMENTALS OF FLUID BALANCE

1. *Total Body Water*

Approximately 80 per cent of the total body weight of the newborn infant consists of fluid. This drops to 75 per cent in the young child and to 55 to 60 per cent in the adult.²

2. *Blood Volume*

The blood volume in infants is approximately 80 ml./kg. In adult males it is 65 to 70 ml./kg. and in females 55 to 65 ml./kg., varying inversely with the amount of body fat present.³

3. *Body Fluid Compartments*

The distribution of fluids within the body compartments in the infant differs markedly from that in the adult. In the infant the cellular compartment comprises 35 per cent of the body weight while the extra-cellular comprises about 40 per cent. In the adult, while the cellular compartment comprises 40 per cent of the body weight, the extra-cellular comprises only 13 per cent.

4. *Renal Function*

Although it is presumably in salt and water balance at birth, generally the newborn baby is precipitated toward dehydration by the lapse of some 24 to 48 hours before it begins ingestion of fluid. According to McCance⁴ the first three or four postnatal days are attended by loss in body weight of 6 to 10 per cent, probably attributable to dehydration. From birth until the third day, the plasma concentrations of urea, uric acid, and NPN generally increase.

According to Thomson,⁵ full-term infants excrete some 20 c.c. of water per day in the first two days, this figure rising to 225 c.c. by the twelfth day. The specific

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gravity of the body in the first two days has a wide range (1.008 to 1.020) with a mean of 1.012. It decreases thereafter for several weeks and levels off in the range of 1.006 to 1.011 with a mean of 1.008. This is felt to be related in part to fluid ingestion. At one month, the specific gravity during water deprivation may rise to 1.036, a figure which is comparable to that in the adult.

This seems to confirm that the kidney output is primarily a response to intake of fluid and electrolyte,^{6, 7} the kidney computing fluid requirements and excreting what is necessary to maintain a constant interior environment. Essential water and electrolyte are retained, and unessential water and electrolyte are promptly discarded.⁸

Because young infants are unable to concentrate urine above 1.020 they eliminate solid products with relatively large quantities of water. It is therefore of paramount importance to supply them with adequate amounts of water and electrolyte to cover losses and to provide for minimal requirements at all times.

5. Factors Regulating the Volume and Osmotic Pressure of the Body Fluids⁹

ADH. In the maintenance of life, the body has homeostatic mechanisms which help to maintain the volume and osmotic pressure of the body fluids within physiological limits. The cellular and extra-cellular osmotic pressure are controlled by the posterior pituitary antidiuretic hormone, ADH. The circulatory volume is controlled by the adrenal cortical hormone, aldosterone.

ADH regulates the osmotic pressure of cells and extra-cellular compartments by controlling the retention and excretion of water by the kidneys. The stimulus for the secretion of ADH is the relative osmotic pressure of the cells; that is, the osmotic pressure of the cells in relation to the osmotic pressure of the extra-cellular fluid. When the osmotic pressure of the extra-cellular fluid becomes relatively greater than the osmotic pressure of the cells, the pituitary gland is stimulated to secrete ADH. When the osmotic pressure of the extra-cellular fluid is relatively less than that of the cells, ADH secretion stops.

The location of this osmotic regulatory centre is in the hypothalamus, probably in its supraoptic and paraventricular nuclei. The site of action of ADH is probably in the distal tubules of the kidneys. The usual stimulus for the secretion of ADH is loss or lack of water.

However, ADH stimulation is said to occur in numerous ways. For example, it can occur as a result of fear or pain, as a result of acute stresses such as trauma, major operations, acute infections, as a result of the administration of analgesics such as morphine or demerol, or as a result of the administration of anaesthetic agents. In many of these conditions, the mechanism by which ADH secretion occurs as a result of acute stress is probably nothing else than a homeostatic compensation in a situation where water losses are increased or water intake has decreased.

Recent investigations suggest that urinary output is not suppressed by analgesic and anaesthetic agents, but by salt and fluid deficits.¹⁰⁻¹²

Aldosterone. The stimulus for the secretion of aldosterone is probably a decrease in cardiac output resulting in decreased intracranial blood flow. This stimulates

the intracranial "volume centre," which in turn carries two simultaneous effects: (a) Widespread vascular (arterial and venous) spasm occurs, probably owing to sympathetic stimulation and the release of norepinephrine. This affects many organs, and particularly the kidneys. As a result of this vasoconstrictor response the effective circulating blood volume tends to be restored toward normal. (b) The adrenal gland is stimulated to secrete aldosterone. Aldosterone reaches the kidneys and causes an increased tubular reabsorption of sodium and water, tending to increase blood volume.^{13, 14}

FLUID ALTERATIONS DURING A SURGICAL PROCEDURE

In 1960 Wilson and Adwan¹⁵ reported on a series of 100 consecutive elective gastric resections, in which only two patients received operative transfusions. The remainder were infused with large volumes of salt solutions. Shock and other morbidity was noted to be lower than in a comparable series in which some 40 per cent of the patients were transfused during surgery. The rationale for the substitution of salt solutions for transfusion was based on measurements of plasma volume and extra-cellular fluid.¹⁶ Extra-cellular fluid is characteristically lost during gastric resection. An over-all fluid loss of four litres is typical, of which three litres are extra-cellular fluid, one litre is blood, but only 450 c.c. are red cells.

In 1961 Shires, Williams, and Brown¹⁸ reported acute reductions of extra-cellular fluid stores during major surgery. This amounted to as much as 28 per cent of total extra-cellular reserves during cholecystectomy. Fluid losses were in direct proportion to the magnitude of surgery. Serum levels of sodium and potassium following extra-cellular losses remained within normal ranges, indicating that the electrolytes of the plasma are likewise lost to the "third space." It was felt that these extra-cellular deficits were due largely to internal redistribution. Shires *et al.* suggest that the deficits might be located within the splanchnic bed, the bowel lumen, in tissues adjacent to the wound, and intracellularly.

In 1963 F. D. Moore *et al.*¹⁷ studied the effects of experimental haemorrhage in man and concluded: (a) Refilling of the vascular compartments is an important homeostatic mechanism that drives fluid from the extra-cellular compartment to the vascular compartment in an attempt to maintain volume. However, it is a slow adjustment, requiring 36 to 48 hours to restore the blood volume to pre-bleeding levels. (b) Plasma protein levels are at first reduced following haemorrhage, but then are replaced from body stores. (c) Effects on the glomerular filtration rate are inconstant, but the urinary ratio of sodium to potassium concentration is consistently reduced, suggesting an aldosterone effect.

In 1964 Terry and Trudnowski⁹ studied 60 patients undergoing major and minor surgical procedures, treated with lactated Ringer's solution in large amounts (two litres for the first hour followed by one litre for each additional hour). They observed a significant reduction in intraoperative and postoperative hypotension. A continuous flow of urine was observed, and was assumed to be clinical evidence of adequate replacement. The need for whole blood transfusion was reduced by one third.

There appears to be enough evidence to warrant the assumption that during surgery the components of the blood—red cells, proteins, water, and electrolytes—are not lost in the same proportion in which they are contained in the vascular compartment, and that a much larger proportion of the surgical loss is water and electrolytes than proteins and red cells.

The common practice of replacing “estimated blood losses” with whole blood or whole blood and plasma (2:1) seems illogical in this light, and accounts for the inability to restore homeostasis despite blood transfusions in patients subjected to prolonged operations.

HOMEOSTATIC MECHANISMS DURING SURGERY

It has been a universal practice to deprive surgical patients of oral intake preoperatively for an average of eight hours. Insensible water losses for the preoperative period are greater than usual because of sweating associated with apprehension the night before surgery. Therefore, even before anaesthesia and surgery are started the patient has a mild degree of dehydration. This is enough to trigger the ADH-aldosterone-kidney mechanism in an attempt to preserve water by diminishing urine volume and by eliminating metabolic waste in a urine of high specific gravity.

At the time the above changes take place, pulse and pressure levels will usually be within normal limits because the homeostatic mechanisms have the capacity to adjust to fluid deprivation.

Analgesic drugs and anaesthetic agents are presumed to have an antidiuretic effect.¹⁸ Recently, however, evidence has been presented that urinary output can be maintained and even increased several-fold⁸ when the patient is infused with polyionic balanced solutions from the start of anaesthesia. A “delay period” has been noticed between the start of lactated Ringer’s infusion and diuresis. This is most likely due to the interval of time necessary to metabolize the ADH present in the plasma.

During operation, fluid losses inherent in surgical trauma initiate the need for conserving fluids to preserve volume. This purpose is accomplished by several homeostatic mechanisms such as vasoconstriction, transcapillary refilling of the vascular compartment, and distal tubular reabsorption of water. Because transcapillary refilling of the vascular compartment is a slow process and because surgical trauma depletes the extra-cellular compartment, hypovolaemia will occur associated with increased levels of aldosterone. The most immediate consequence is oliguria with high specific gravity and a reduction in the ratio of sodium to potassium concentration. If the fluid losses are large enough, the latter homeostatic mechanisms will be insufficient and the circulatory system will initiate a distribution of compromise. Vasoconstriction will become most marked in the extremities, skin, splanchnic area, and renal arteries in an effort to divert the remaining blood volume to vital organs. At this point tachycardia, hypotension, pale skin, oliguria are common findings.

The use of blood transfusion to prevent or reverse circulatory failure in this

circumstance has been only partially effective. A postoperative period characterized by fever, hypotension, tachycardia, ileus, oliguria, and even anuria is not uncommon following prolonged surgery.

OBJECTIVE OF THE STUDY

Assuming from the work of Wilson, Shires, Moore, Terry, and others that intra-operative urine flow and composition are primarily related to extra-cellular reserves, the present study was undertaken to determine to what extent the above principles could be applied to paediatric surgery. Would adequate hydration sustain urinary flow, and conversely would continuous urinary flow indicate optimal intravenous infusion rates?

METHOD

Fifteen patients undergoing prolonged surgery (more than two hours) and/or abdominal surgery were included in this study. Age ranged from 7 days to 16 years (Table I), ASA physical status from one to three. No cardiac patients were included. All patients were in water and electrolyte balance prior to surgery. Patients were premedicated with meperidine and atropine in doses based on age and weight. Patients under one year of age were given atropine only. Inhalation induction with nitrous-oxide-halothane-oxygen was used in patients under 14, and minimal sleep doses of thiopental sodium were used in the older patients. Anaesthesia was maintained with nitrous-oxide-oxygen (6:2 or 4:2) supplemented by halothane and/or muscle relaxant. Body temperature was monitored by means of an oesophageal thermometer, and a thermal blanket was used whenever necessary to maintain normal body temperature.

After induction, all patients were catheterized with a Foley catheter, and the bladder was emptied and the specific gravity of the urine obtained was measured with a densimeter or a T.S. meter when the amount of urine was less than 20 c.c. A capillary microhaematocrit was also determined at this time. All determinations were done in the operating room.

During the first hour of surgery, patients received an infusion of lactated Ringer's solution amounting to 30 per cent of the estimated blood volume. After each hour of surgery urine volume and specific gravity were recorded, and sufficient lactated Ringer's solution was infused to maintain the urinary output and specific gravity near the mean preoperative values.

Blood loss was estimated after weighing the surgical sponges, measuring the contents of the suction bottles, and estimating loss into the surgical drapes. Blood was transfused whenever the haematocrit fell below 30 per cent and/or the estimated blood loss was between 15 and 20 per cent of the patients' estimated blood volume.

During the postoperative period, plasma electrolyte determinations, urinary output, temperature, and time of reappearance of bowel sounds were recorded. Patients were observed for the possible development of tissue and/or pulmonary oedema.

TABLE I

Patient No.	Age	Procedure	Duration of anaesthesia (hours)	Urine output during surgery (ml.)	Lactated Ringer's solution (ml.)	Blood loss (ml.)	Blood transfused (ml.)	Weight (Kg.)	Body surface (m. ²)	Urine output 24 hours post-op. (ml.)
1	16 yrs.	Harrington	5	130	2350	2700	2000	43	1.45	1125
2	14	Harrington	5	250	5300	3600	3000	62	1.62	2700
3	12	Harrington	4	145	3250	2250	2100	40	1.29	2500
4	10	Craniotomy	4	360	1600	—	500	42	1.40	1250
5	10	Ureteropyeloplasty	3	105	1400	200	—	34.5	0.9	825
6	8	Ileal conduit	6	360	2500	500	400	19.7	0.8	1100
7	8	Closure recto vaginal fistula and anoplasty	3	160	750	350	300	25	0.9	1200
8	8	Spinal fusion	3	175	1400	1100	1000	26	1.1	1300
9	5	Heller Procedure	3	130	1000	200	—	25	0.9	1350
10	3	Correction petus excavatus (esophagostomy)	5	125	1000	475	500	15	0.6	850
11	2	Nephrostomy and nephrectomy	3	265	725	100	—	10	0.5	2500
12	15 mos.	Biopsy adrenal neuroblastoma	2	75	350	50	—	9.6	0.45	450
13	7	Torkildsen operation	2	40	225	100	60	10	0.42	520
14	2	Craniotomy	3	25	450	125	200	3.7	0.25	325
15	7 days	Exploratory laparotomy	1	55	110	30	—	3.3	0.20	260

TABLE II
PATIENT No. 2

Weight (Kg.)	57
Height (m.)	1.50
Haemoglobin (gm.)	14.2
Haematocrit (%)	41
Urine specific gravity	1.006
Body surface (m. ²)	1.62
Estimate blood volume (c.c.)	4200
Estimate urinary output (c.c./hr.)	60

Patients #2 and #11 have been selected as representative of the method followed in this study.

Patient #2

A 14-year-old female with idiopathic scoliosis undergoing a Harrington Procedure. History, physical examination, and laboratory data did not reveal any other abnormalities (Table II). Risk ASA #1. The sequence of events during surgery is illustrated in Figure 1.

Patient #11

A two-year-old under-developed male with congenital kidney anomalies, hydro-nephrotic syndrome, recurrent urinary infections, polydipsia (average daily intake

FIGURE 1. Patient #2. Sequence of events during surgery.

TABLE III
PATIENT No. 11

Weight (Kg.)	10
Height (m.)	0.83
Haemoglobin (gm.)	11.5
Haematocrit (%)	35
Urine specific gravity	1.005
Body surface (m. ²)	0.5
Estimate blood volume (c.c.)	800
Estimate urinary output (c.c./hr.)	60

1700 c.c.), polyuria (average daily output 1500 c.c.). Urine specific gravity was never above 1.015. IVP showed no contrast in the left kidney and a poor contrast right kidney. Risk ASA #3. (Table III.) Patient was scheduled for a left nephrectomy and a right nephrostomy. The sequence of events during surgery is illustrated in Figure 2.

COMMENT

From Table I and the two cases reported, the following comments may be made with respect to the type and amount of fluid infused during surgery.

Type of Fluid

All 15 patients were infused with lactated Ringer's solution instead of the often recommended 10 per cent dextrose in water.¹⁹ It appeared that a polyionic

POST OPERATIVE PERIOD

Na	K	Cl	CO ₂ content	pH	Hb	Hmt.	BUN
136	3.6	100	21.8	7.30	11.1	30	26.1

	1st day	2nd day
Urinary Output CC	2500	2000

FIGURE 2. Patient #11. Sequence of events during surgery.

balanced solution would be a more adequate fluid to be infused during surgery, since surgical losses seem to be made up of water and electrolyte in the proportions in which they are found in the plasma.

Amount of Fluid

The amount of fluid was not predetermined according to age or weight as has been proposed,²⁰ but was decided upon as surgery went along. Enough fluid was infused to maintain volume and specific gravity of the urine close to the "normal values" for each individual patient. By so doing it was felt that fluid needs were covered as they appeared, therefore taking into account individual patient variables, patient disease, surgical procedure, and the different phases of each surgical procedure.

Early diagnosis of oliguria permitted appropriate measures during the "golden hours" before the body homeostatic mechanisms had been compromised by the metabolic changes of anaesthesia and surgery.

RESULTS

1. None of the patients had oliguria during or after surgery. A continuous urinary output could be maintained up to predetermined values by increasing or decreasing the rate of infusion of the lactated Ringer's solution.

2. No significant pulse or blood-pressure changes were observed during the procedures.

3. Blood transfusions were given only to treat anaemia, not for hypovolaemia. Therefore, the amount of blood used was considerably reduced.

4. During abdominal surgery the bowel was moist, and the practice of covering the bowel with moist packs could be eliminated since the amount of fluid infused was more than enough to cover the evaporation losses from the bowel surfaces.

5. Plasma electrolytes were within normal limits postoperatively.

6. Postoperative urinary output was above estimated levels.

7. Bowel sounds were present within 24 hours after surgery in all the patients.

8. Temperature postoperatively was never above 100° except in Patient #3, who had a Torkildsen Procedure.

9. There were no signs of pulmonary oedema during or after surgery. One patient showed palpebral oedema at the end of surgery which resolved within eight hours postoperatively. This was probably due to positioning.

CONCLUSIONS

Monitoring of the urinary output and urine specific gravity during surgery on an hourly basis has provided a practical guide to fluid therapy in paediatric surgery. During surgery the infusion of a polyionic fluid with a plasma-like composition (lactated Ringer's solution) proved not to be detrimental to the patients; on the contrary, it resulted in a significant reduction in operative and postoperative morbidity.

Blood transfusion became of secondary importance because it was not used to restore blood volume but to replace proteins and red blood cells.

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