FLUID AND ELECTROLYTE PROBLEMS IN THE SURGERY OF THE AGED*

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FERENCE T is generally recognized that plasma electrolytes and the physiological regulation of electrolyte balance in the geriatric patient do not differ qualitatively from that of the younger patient. Quantitatively, however, the margin of safety and the ability to compensate under stress may be impaired in the aged.¹⁻⁴ An understanding of the common difficulties which may be anticipated in surgery of the aged patient is therefore important for successful management. In our experience, three major alterations should be given special consideration, since they are those which we have encountered most frequently in the pre- and postoperative intervals in aged patients.

The alterations which are common and the periods in which they are seen include: 1) in the preoperative period, contraction of blood volume and electrolyte alterations resulting from operative preparation; 2) in the postoperative period, the electrolyte changes which arise from the normal response to surgery; 3) abnormalities which result from pulmonary dysfunction following surgery. The purpose of this paper is: 1) to present the common alterations which may occur in the pre- and postoperative intervals; and 2) to propose that surgery need not be denied or compromised in the elderly patient.

PREOPERATIVE INTERVAL

Because of the greater incidence of pulmonary, cardiac, metabolic and renal disease in the aged, the initial workup must be more complete than in the young patient. The recognition of metabolic disturbances and reflected plasma chemical abnormalities have been

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Time	In	testinal Conten	nts		Plasma	
Min.	Sodium	Potassium mEq/L	Chloride	Sodium	Potassium mEq/L	Chlorid e
0			cc. of Isotonic	Mg SO ₄		
12 0	132	2.17	68.7	147	4.9	117

TABLE I—ELECTROLYTE CONCENTRATION IN AN ISOLATED ILEAL SEGMENT FOLLOWING THE INTRODUCTION OF ISOTONIC MAGNESIUM SULPHATE SOLUTION

elaborated elsewhere and therapy outlined for correction.⁵⁻⁹

A special problem facing the physician is one resulting from surgical preparation. It has recently become evident that patients who are undergoing standard bowel preparation for gastrointestinal surgery frequently display a hyperchloremic acidosis and negative potassium balance following the period of preparation with purgatives.¹⁰ The mechanisms of these changes are illustrated in Tables I and II. Basically, it has been shown that a poorly absorbed solution such as magnesium sulphate, when introduced into the intestinal lumen, will result in a secretion of electrolytes into the lumen. The concentration of electrolytes will vary for each intestinal segment and if the solution is hypertonic to the plasma, water will also shift into the lumen of the bowel.^{11, 12} This is illustrated in Table I, which shows the data from an experiment carried out by Bane.¹¹ In this experiment isotonic magnesium sulphate was placed in an isolated ileal segment of a dog and the shift of sodium, chloride and potassium measured over a two hour interval. As shown, the net shift of electrolytes resulted in a concentration of 132 mEq. of sodium, 68.7 mEq. of chloride and 2.17 mEq. of potassium per liter. Although it was apparent in these studies that concurrent electrolyte shifts were occurring both from the lumen to the plasma and from the plasma to the lumen of the bowel, the net shifts of sodium, chloride and potassium were always quantitatively greater from plasma to lumen when a poorly absorbable solution was introduced. In the jejunum, electrolyte shifts also occur, but differ as regards composition. Whereas sodium and chloride are secreted in about equimolar concentration in the jejunum and upper ileum, the concentration of chloride in lower bowel segments is less than the

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	Sodium	Potassium	Plasma Carbon Dioxide mEq/L	Chloride	pH	
Before bowel preparation	n 142	4.9	28	102	7.43	
After bowel preparation	140	5.3	24.2	116	7.33	

TABLE	II-PLASMA	ELECI	ROI	YTES	IN A	PATIEN	T BEFORE	AND	\mathbf{AT}	THE
	COMPLE'	TION O	F A	FIVE	DAY	BOWEL	PREPARA'	FION		

concentration of sodium. This is also illustrated in Table I, which shows that approximately half of the sodium and potassium may be shifted into the lumen with chloride and presumably a major fraction of cation is secreted with bicarbonate. It is noteworthy that the bicarbonate concentration of lower bowel content is higher than that found in the plasma, whereas chloride concentration of the lower ileum and large bowel is less than that normally found in the plasma.⁹ These concentrations are not unlike those which have been described in diarrhea contents or ileostomy drainage.⁶ Logically, it would be expected that such external losses of sodium and potassium in excess of chloride, if excessive and unreplaced, would result in a plasma hyperchloremia and acidosis. In the patients studied by us such was found to be the end result of vigorous bowel purgation. This is illustrated in a patient shown in Table II, whose plasma electrolytes and pH were essentially normal prior to a five day bowel preparation. At the end of the preparatory interval the patient displayed a plasma hyperchloremia and acidosis. Similar plasma findings have been demonstrated in eight of nine patients studied by us before and at the completion of bowel preparation for surgery.¹⁰ Cognizance of the potential existence and hazards of hyperchloremic acidosis preoperatively, compels its treatment which consists essentially of rehydration and alkali administration as bicarbonate or lactate. Usually this is satisfactorily accomplished the day prior to surgery, by oral or intravenous administration of 100-200 mEq. of sodium bicarbonate or lactate. A major consideration arising from these observations is that the patient should not be denied adequate bowel preparation, but rather, that the electrolyte derangements should be recognized and treated preoperatively.



Figure 1—Diagrammatic representation of the postoperative third space and plasma electrolyte changes.

PREOPERATIVE BLOOD VOLUME

It is not uncommon to find a contracted blood volume preoperatively in the aged patient.^{2, 13} In the majority of patients the contracted volume has been related to one of four factors: 1) gastrointestinal bleeding; 2) inadquate dietary intake; 3) chronic renal disease; or 4) malignancy. Since the contracted blood volume may not be revealed by any alteration in the hematocrit, it may easily escape detection. For this reason preoperative blood volumes in the aged patient are essential. Since the patient is usually "adjusted" to his contracted volume and sudden expansion has been known to result in congestive failure, daily replacement of 250 cc. of blood until correction is achieved affords a conservative and successful approach to this problem.

POSTOPERATIVE INTERVAL

The postoperative response to surgery is not significantly different from the response which has been described in younger members of the populace. Fundamental to therapy in the postoperative period is an understanding of the normal shifts of water and electrolytes which occur following major surgical procedures.^{6, 10, 14, 15} Essentially, these may be outlined as follows: In the immediate three to four days following surgery there is the formation of a "third space," and an associated extracellular expansion of water. This is illustrated in Figure 1. As shown, there is an initial shift of water and extracellular electrolytes to the third space, which may be into the pleural cavity, bowel, peritoneal cavity, pelvis or extremities. Retention of exogenously administered water and sodium is also commonly observed in the first few days postoperatively. The resultant retention of water and compensatory shifts of water from the intracellular to the extracellular compartment, result in dilution of the major extracellular components. The net result will be: 1) an expanded extracellular fluid; 2) hyponatremia and hypochloremia secondary to dilution* (Fig. 1); 3) a drop in hematocrit and protein which is also secondary to the hydremia. During the interval of these changes, urine volume may be low with a high specific gravity. During the resolution of the third space diuresis ensues, the excretion of sodium increases and losses of water and sodium may exceed intake. At the completion of resolution the return of plasma composition and the hematocrit to normal is the usual finding.

While these changes which have been observed postoperatively are not peculiar to the aged patient, certain features in this group of patients are noteworthy. 1) In the patient, with pre-existent cardiac dysfunction and a previously restricted sodium intake, the drop in plasma and sodium during the third space period may be more marked. 2) The administration of sodium must be carefully considered. 3) The expansion of the extracellular fluid may compromise circulatory dynamics as the result of pre-existent myocardial damage. Hence it is important to guard against overloading the circulation by unnecessary transfusions or excessive saline administration.

Hormonal Response: The adrenal response to surgery and the role of the adrenal hormones in the acute postoperative period has been recognized and it has been established that the "alarm reaction" following surgery is a major sequence.¹⁵ Release of anti-diuretic-hormone has been indicted in the antidiuretic response during the period of acute water retention.¹⁶ However, the importance of this factor and its mechanism of release in the postoperative extracellular dilution remains obscure.

^{*} Total extracellular sodium (as calculated from the plasma sodium concentration and extracellular sucrose space) may actually be greater in this postoperative interval.

	Sodium	Plasma Potassium mEq/L	Chloride	Blood Urea mgm%	Hematocrit %	Urine Spec. Grav.	Vol.
Dehydration (plus third space effect)	+	1	+	↑	+	↑ ↑	↓
Renal Shutdown (plus third space effect)	¥	↑ ↑	¥	↑↑	¥	¥	↓↓

 TABLE III—MAJOR DIAGNOSTIC FINDINGS IN RENAL SHUTDOWN AND POSTOPERATIVE DEHYDRATION

DEHYDRATION AND RENAL SHUTDOWN DURING THE POSTOPERATIVE PERIOD

The differential diagnosis between renal shutdown and dehydration is a common problem facing the clinician in evaluating early postoperative oliguria in the elderly patient who has had conservative fluid and electrolyte replacement and questionable renal function. This is particularly true in patients who have had a transfusion reaction, shock or severe trauma. Since the "third space effect" and oliguria will be superimposed on either of these situations the changes which occur are additive. Factors which may aid in establishing the correct diagnosis are listed in Table III. The specific gravity of the urine is of the utmost importance; if the specific gravity is low renal shutdown assumes more prominence, whereas an elevation of specific gravity may indicate dehydration. In renal shutdown the hematocrit and protein may drop while dehydration is accompanied by a stable or rising hematocrit. Plasma sodium and chloride concentrations remain unchanged or elevated if the patient is dehydrated and concentration decreases if renal shutdown exists. In renal shutdown the blood urea nitrogen and potassium rise precipitously, whereas dehydration causes only minor elevations of these plasma constituents. It is particularly important to differentiate between renal shutdown and dehydration, since excessive fluid therapy may be hazardous if marginal pulmonary or cardiac reserve exists in the patient.



Figure 2-Physiological compensations in respiratory acidosis (see text).

Respiratory Acidosis

The higher incidence of pulmonary disease and postoperative atelectasis is generally recognized in the older age group.¹⁻³ Therefore rigorous attention to adequate ventilation and the recognition of pulmonary acidosis and accompanying hypoxia are important. The physiological compensations occurring in respiratory acidosis have been well described and are diagrammed in Figure 2.17-20 They include: 1) an initial retention of carbon dioxide with elevation of carbonic acid and a drop in plasma pH; 2) a shift of chloride to the intracellular compartment, leaving behind an equal moiety of sodium in the extracellular fluid; 3) extracellular shifts of potassium and sodium; 4) increased renal tubular reabsorption of filtered bicarbonate; and 5) enhanced renal excretion of chloride and phosphate with ammonium and hydrogen ion with a consequent increase in the renal formation of sodium bicarbonate by virtue of ion exchange. As a result of these compensations the total CO₂ content of the plasma rises, chloride concentration falls, and there is little change in sodium and potassium concentration.* If compensations are complete, as is often the case, the extracellular pH will be normal or only slightly decreased.

^{*} Potassium concentration will rise only if there is severe renal impairment or superimposed anoxia.



Figure 3—The shaded area represents plasma pCO_2 in patients with metabolic alkalosis who had no deducible evidence of pulmonary disease. The circles represent patients with decreased pulmonary exchange. (Data from J. W. Poppell.)

Respiratory acidosis must be differentiated from metabolic alkalosis, which is not uncommon in the elderly patient who has had an inadequate potassium intake or has been subjected to gastric drainage of chloride.9 In both respiratory acidosis and metabolic alkalosis total carbon dioxide will be elevated; therefore, the differential diagnosis will evolve itself around the plasma pH, and clinical evidence of cyanosis or inadequate pulmonary ventilation. The finding that pulmonary compensations are minimal in metabolic alkalosis is of fundamental importance⁸ in differentiating between metabolic alkalosis and respiratory acidosis. This is illustrated in Figure 3, which summarizes the changes in plasma pCO₂ in the patients with decreased respiratory exchange, who were studied by Poppell.²¹ The shaded areas represent the respiratory compensations, as reflected by the plasma pCO₂ in patients without pulmonary disease who had metabolic alkalosis. The plotted points represent the calculated plasma pCO₂ of patients with respiratory disease and as illustrated, the majority of these patients had a plasma pCO₂ greater than the normal of 38-46 mm. Hg. Table IV

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			Plasma Concentration		
	<i>pCO</i> ₂	pH	Total Co ₂	Chloride	
Metabolic Alkalosis	+	↑	↑	¥	
Respiratory Acidosis	↑	$\pm - \downarrow$	↑	¥	

TABLE IV—PLASMA FINDINGS IN PATIENTS WITH METABOLIC ALKALOSIS AND RESPIRATORY ACIDOSIS

summarizes the plasma alterations which may be expected in respiratory acidosis and metabolic alkalosis. In both situations plasma carbon dioxide will be elevated and chloride decreased. In respiratory acidosis, however, the plasma pH will be low or normal and pCO₂ will be greatly increased.

Of major importance is the realization that respiratory acidosis of itself may not be a hazardous clinical entity but that its diagnosis is important because it is often a reflection of anoxia.*19 The treatment therefore, is not that of correction of the extracellular electrolyte abnormality, but rather energetic efforts to increase ventilation and establish adequate pulmonary exchange of oxygen. The treatment is dependent on the relief of precipitating factors. Ancillary measures include mechanical aids to ventilation, tracheal suction, early ambulation, bronchodilators, antibiotics, expectorants and other agents designed to facilitate removal of secretions.⁵ Bronchoscopy or tracheostomy may be life saving during the early postoperative interval. Although a discussion of narcotics is beyond the scope of this paper, the sparing use of these agents is strongly recommended. In the patients seen by us, the efforts to correct postoperative pulmonary difficulties have been discouraging, and mortality of the aged patient with pulmonary disease is greatly in excess of that observed from any other group of electrolyte disturbances we have encountered.

Summary

Three major considerations have been discussed in regard to electrolyte abnormalities in the aged patient undergoing surgery. These include: 1) The occurrence of hyperchloremic acidosis as the result of

^{*} Especially in a patient breathing room air.

preoperative bowel preparation in the elderly patient; 2) normal postoperative alterations in extracellular fluid and electrolytes; and 3) the high incidence of respiratory acidosis and anoxia. It has been stressed that the anoxia which frequently attends respiratory acidosis is common in the elderly patient and is enhanced by postoperative atelectasis. In our experience the morbidity and mortality from pulmonary disease is greater than from any other group of electrolyte disturbances in the postoperative aged patient.

REFERENCES

- Anglem, T. J. and Bradford, M. L. Major surgery in the aged, New Engl. J. Med. 249:1005-09, 1953.
- 2. Cole, W. H. Operability in the young and aged, Ann. Surg. 138:145-57, 1953.
- 3. Stewart, J. and Alfeno, G. S. Surgery of the elderly, J. Amer. med. Assoc. 154:643-46, 1954.
- Strohl, E. L. Preoperative and postoperative care of the elderly, *Geriatrics* 8:377-84, 1953.
- 5. Harrison, T. R. et al., editors. *Principles of internal medicine*. 2.ed. New York, Blakiston, 1954.
- Randall, H. T. Water and electrolyte balance in surgery, Surg. Clin. N. Amer. 32:445-69, April 1952.
- Randall, H. T. and Roberts, K. E. The significance and treatment of acidosis and alkalosis in surgical patients, *Surg. Clin. N. Amer.*, in press.
- 8. Roberts, K. E., Poppell, J. W., Vanamee, P., Beals, R. and Randall, H. T. Evaluation of respiratory compensations in metabolic alkalosis, *J. clin. Invest.*, in press.
- Roberts, K. E., Vanamee, P., Randall, H. T. and Walker, J. M. Common electrolyte abnormalities encountered in bowel surgery; mechanism of hypochloremic alkalosis, hypokalemic alkalosis and hyperchloremic acidosis, Surg. Clin. N. Amer. 35:1189-1200, Oct. 1955.
- 10. Schwartz, M. K. Unpublished observations.
- 11. Bane, H. N. Personal communication.
- 12. Visscher, M. B. Water and ion movements across intestinal and renal epithelium, *Trans. Fourth Conference on*

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Renal Function (Josiah Macy, Jr. Foundation), pp. 123-189, 1952.

- Beling, C. A., Bosch, D. T. and Carter, O. B., Jr. Blood volume in geriatric surgery, *Geriatrics* 7:179-83, 1952.
- Lyon, R. P., Stanton, J. R., Freis, E. D. and Smithwick, R. H. Blood and available fluid (thiocyanate) volume studies in surgical patients, Surg. Gynec. Obstet. 89:9-19, 1949.
- Moore, F. D. and Ball, M. R. The metabolic response to surgery. Springfield, Ill., C. C. Thomas, 1952.
- 16. Dudley, H. R., Boling, E. A., LeQuesne, L. P. and Moore, F. D. Studies on antidiuresis in surgery; effects of anesthesia, surgery and posterior pituitary antidiuretic hormone on water metabolism in man, Ann. Surg. 140:354-65, 1954.
- Davies, H. W., Haldane, J. J. R. and Kennaway, E. L. Experiments on the regulation of the blood's alkalinity, J. *Physiol.* 54:32-45, 1920.
- Giebisch, G., Berger, L. and Pitts, R. F. Extrarenal response to acute acid-base disturbance of respiratory origin, J. clin. Invest. 34:231-45, 1955.
- Peters, J. P. and Van Slyke, D. D. Quantitative clinical chemistry, v. 1. Interpretations. 2.ed. Baltimore, Williams & Wilkins Co., 1946.
- 20. Pitts, R. F. Mechanisms for stabilizing the alkaline reserves of the body, *Harvey Lect.* 48:172-209, 1952-1953.
- 21. Poppell, J. W., Vanamee, P., Roberts, K. E. and Randall, H. T. The effect of ventillatory insufficiency on respiratory compensation in metabolic acidosis and alkalosis. Submitted for publication.