Hyperparathyroidism and Hypoparathyroidism: Preoperative and Postoperative Care

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Careful analysis of the complex biochemical changes evoked by excessive or insufficient secretion of parathyroid hormone (parathormone) will allow for proper treatment of such patients undergoing operative procedures. The following discussion is designed to elucidate the major biochemical changes and to indicate the role that these alterations play in the care of the patient.

Hyperparathyroidism

The biochemical changes resulting from the excess elaboration of parathyroid hormone consequent to pathological changes of the parathyroid glands (primary hyperplasia, adenoma, carcinoma), are those attributable to the production of hypercalcemia. Secondary hyperplasia of the parathyroid glands is found in those diseased states wherein the plasma calcium is lowered (renal acidosis, sprue, osteomalacia), thereby stimulating the parathyroid glands to secrete additional hormone in order to return the plasma calcium to normal levels. As such, there is no hypercalcemia and no indication for operative intervention in secondary hyperparathyroidism, since measures designed to raise the plasma calcium will cure secondary hyperplasia (vitamin D, calcium lactate).

Symptoms and Signs

Primary hyperparathyroidism with hypercalcemia is treated surgically and expressed clinically as a syndrome involving development of renal calculi, duodenal ulcer, and band keratopathy. The symptoms of hypercalcemia are myriad. Headache, joint pains, dyspepsia, nausea, vomiting, polyuria, polydipsia, constipation and fatigue are the more common ones.

The biochemical changes (table 1) seen in hyperparathyroidism are secondary to the effects of hypercalcemia, for the most part, involving the kidney, but a reduction in neuromuscular transmission is observed as well. Obviously, the rise in plasma calcium secondary to excessive secretion of parathyroid hormone is due to the action of the hormone on bone. It has also been shown recently that parathyroid hormone will cause increased absorption of calcium from the gastrointestinal tract thereby tending to elevate the plasma calcium. Other actions of parathyroid hormone on calcium metabolism are effected through the kidneys and the lactating breast where the action of the hormone is designed to conserve calcium.

Renal Action of Parathyroid Hormone. The action of parathormone on the renal excretion of calcium and phosphorus is well known and the resultant hypercalcuria and hyperphosphaturia remain characteristic diagnostic signs of hyperparathyroidism. The precise mechanism by which the increased urinary output of phosphate is accomplished is a source of controversy. The work of Hiatt and Thompson, and recent confirmatory studies by Rich et al. in man have stressed the importance of tubular reabsorption of phosphate independent of altered glomerular filtration. The extent that tubular secretion participates in this process in man is unknown; and, in a recent review of the problem by Bartrter, it is concluded that although there is evidence for tubular secretion of phosphate in the chicken and alligator, this mechanism need not be postulated for man. In respect to the handling of calcium by the renal tubule, the studies of Talmage and Kleeman indicate that the parathyroid hormone enhances tubular reabsorption of calcium. The initial rise in the tubular maximal transport (Tm) for calcium, however, is even-
tually exceeded by a rising plasma calcium and an increase in the filtered load resulting in a net hypercalcuria.

The discovery of polyuria and impaired renal concentrating ability as prominent manifestations of hyperparathyroidism and other hypercalcemic states (sarcoidosis, vitamin D intoxication, milk-alkali syndrome and malignancies) has led to further studies on renal function. These associated abnormalities in different disease states have in common an elevation of urinary and plasma calcium, suggesting that the defect is the consequence of the excessive calcium and not of the specific disease. It is still possible however that excess of parathormone may have a deleterious effect on renal function independent of the hypercalcemia. Manitius et al. have demonstrated that the impairment of free water clearance ($T_{\text{H}_{2}O}$) is greater in degree in hypercalcemia produced by administration of parathyroid extract than in similar elevations induced by infusion of calcium salts alone. This is interesting in light of the evidence presented by Schneider, Reaven and Reaven where it was shown that renal calcification produced in mice by parathyroid extract differs from that produced by calcium gluconate.

The effect of hypercalcemia and hypercalcuria on renal function has been studied extensively in animals and man, both in experimental and in endogenously induced states. Shelling, Kajdi and Guth as well as Ellsworth and Nicholson demonstrated that excess loss of sodium chloride in the urine followed the injection of parathyroid extract. Wolf and Ball, comparing the effects of intravenous calcium in dogs, recorded an increased rate of potassium, sodium and chloride excretion. The data of Levitt derived from intravenous infusion of calcium salts and sodium versenate (sodium ethylene-diamine-tetracetic acid) in man and monkey, imply that the alteration of calcium in the plasma and urine effects an immediate and opposite change in the rate of reabsorption of sodium, chloride.

### Table 1. The Biochemical Effects of Hypercalcemia and Hypocalcemia

<table>
<thead>
<tr>
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<th>Hypercalcemia</th>
<th>Hypocalcemia</th>
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<tbody>
<tr>
<td><strong>Cardiac:</strong></td>
<td>Enhanced contractility and excitability, vagal stimulation</td>
<td>Impaired contractility</td>
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<tr>
<td><strong>ECG:</strong></td>
<td>Shortening of the Q-T interval. Absent, shortened or depressed ST segment.</td>
<td>Prolonged Q-T interval. Straightened and elongated ST segment.</td>
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<tr>
<td><strong>Digitalis:</strong></td>
<td>Digitalis synergism; enhances toxicity.</td>
<td>Lowers effectiveness of digitalis; reverses toxicity.</td>
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<tr>
<td><strong>Other:</strong></td>
<td>Bradycardia; premature ventricular contractions; ventricular fibrillation.</td>
<td>Impaired cardiac output; hypotension.</td>
</tr>
<tr>
<td><strong>Renal:</strong></td>
<td>Decreased tubular reabsorption of phosphate; increased phosphate clearance</td>
<td>Increase in tubular reabsorption of phosphate with decrease in phosphate clearance.</td>
</tr>
<tr>
<td><strong>Calcium:</strong></td>
<td>Increased tubular absorption of calcium with increased clearance of calcium.</td>
<td>Decreased tubular reabsorption with low or normal clearance of calcium.</td>
</tr>
<tr>
<td><strong>Other:</strong></td>
<td>Impaired acidification. Impaired concentrating ability.</td>
<td></td>
</tr>
<tr>
<td><strong>Neuromuscular:</strong></td>
<td>Decreased irritability; increased pressure, cerebrospinal fluid (rare).</td>
<td>Increased irritability; tetany and tetanic equivalents. EEG changes.</td>
</tr>
<tr>
<td></td>
<td>Stabilizes transmission.</td>
<td>Labilizes transmission. Increased cerebrospinal fluid pressure (common).</td>
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and potassium in the kidney. This observed diuresis has been confirmed repeatedly by numerous authors and has been found to be accompanied invariably by a defective urinary concentrating mechanism. Renal concentrating capacity as measured by maximum urine osmolarity ($U_{\text{max}}$) and free water clearance ($\text{C}_{\text{H}_{2}\text{O}}$) is severely impaired in hypercalcemic dogs and man, often out of proportion to other renal functional states. Morphological studies of hypercalcemic kidneys by Epstein, Rivera and Carone have demonstrated lesions involving the collecting ducts in the outer medulla. These lesions were not found to be specific for calcium and bore a similarity to the renal changes associated with hypokalemic nephropathy. Employing parathyroid hormone, these authors have succeeded in inducing similar lesions (i.e., small foci of epithelial degeneration, calcification of epithelial cells in the basement membrane, and necrotic calcified intratubular casts with obstruction and dilation) of the distal nephron within 24 hours.

The mechanism by which the structural and functional changes occur under parathyroid hormone influence is unclear. According to current theories, impaired concentrating facility could result from (1) an alteration in the permeability to water and sodium of the distal tubular membrane and collecting ducts, (2) insufficient medullary solute concentration, or, (3) unresponsiveness or resistance to antidiuretic hormone (ADH). Manitius et al. concluded from their observations that there was an impaired reabsorption of sodium by the renal tubules and a diminished ability to create and maintain a high concentration of sodium in the interstitial fluids of the medulla and papilla. They also suggested that the permeability of the collecting ducts to the active diffusion of water might be defective. Barter in his studies on hypercalcemic subjects could not substantiate a failure of sodium conservation. *In vitro*, however, calcium does inhibit sodium and water transport across frog skin. Calcium also can suppress the action of vasopressin in slices of necturus kidney. The possibility that the accompanying diuresis and elevated osmotic load could conceivably result in hyposthenuria seems unlikely since experimental data indicate that the depression of $\text{C}_{\text{H}_{2}\text{O}}$ is far more than that which a massive solute diuresis could create. At present, therefore, we are unable to determine the exact mechanism by which parathyroid hormone impairs concentrating mechanisms in the kidney or increases total urinary solute. In respect to the latter, Wallach and Carter believe that a portion of the generalized cationic diuresis may be explained by heterionic exchange and transfer of these ions from the hydration shell of bone crystal.

The administration of parathyroid hormone has also been shown to effect urinary acidification mechanisms. This is to be expected since (1) one of the major anions with which parathyroid hormone is concerned is phosphate, which is also the major urinary buffer, and (2) urine is acidified by the distal tubule which is the area where morphologically calcium is deposited in states of excessive parathyroid hormone. Wallach and Carter demonstrated that the augmented excretion of sodium, chloride, potassium and magnesium was accompanied by an increase in titratable acid, ammonia and pH in the urine of dogs treated with calcium gluconate. This diminution in renal tubular acidification capacity has been confirmed in a hyperparathyroid patient by Wrong and Davies. Fourmann, McConkey and Smith studied the excretion of hydrogen as measured by the changes in the pH and in excretion of ammonia and titratable acid after ingestion of ammonium chloride in four patients with hyperparathyroidism. These patients failed to lower urinary pH and apparently this defect was present even after parathyroidectomy. Nordin has confirmed this and observed that the ingestion of commercial parathyroid extract was invariably followed by a rise in bicarbonate excretion and a rise in urine pH. The real possibility that these changes might represent alterations secondary to the hemodynamic effects (i.e., increased glomerular filtration rate) of crude extract has been somewhat clarified by the recent use of purified hormone by Rasmussen and Fchet. These authors observed that the injection of purified hormone in hypoparathyroid, euparathyroid, and pseudohypoparathyroid patients resulted in increased urinary pH. They postulated that the mechanism may be explained entirely by phosphate secretion, resulting in increased sodium excretion, a de-
creased exchange of sodium for hydrogen ion, and an elevation of pH. Although this is speculative, their finding of an impairment in hydrogen secretion is intriguing in view of the tendency toward development of acidosis (low CO₂ combining power) often manifested by hyperparathyroid patients.

**Myocardial Effects of Hypercalcemia.** The cardiovascular effects of hypercalcemia assume a somewhat less but still important role in the hyperparathyroid subject. Clinically, this is obvious, since the cardiac status is germane to the problem of successful surgical intervention. Equally relevant are the effects of hypercalcemia on the function of the heart.

Since the historical work of Ringer 21 it has been known that calcium is one of the three cations necessary for the normal function of the perfused heart; calcium increasing the contractility and prolonging systole, potassium having the reverse effects. Equally well known now is the effect of hypercalcemia in man and experimental animals. The infusion of calcium in man results in changes resembling vagal stimulation and consist of bradycardia, sinus arrhythmia, shifting pacemaker and variable degrees of heart block. In high concentrations the ventricular musculature is stimulated and premature ventricular contractions, ventricular tachycardia and ventricular fibrillation may ensue. The electrocardiographic changes ascribed to hypercalcemia are a relatively abnormal and shortened Q-T interval and an absent or shortened ST segment which is thought to represent an early onset of repolarization. Usually there are few or no changes in the T wave, although it may occasionally show a decrease in amplitude.

The action of ionized calcium on myocardial function is but one facet of the significant influence that this ion exerts on biological phenomena. It has become increasingly common to view the effect of calcium in terms of the electrochemical alteration associated with the cellular event of excitation; namely, bioelectric potential, ionic distribution and membrane permeability. According to the present hypothesis, the cardiac cell is considered a three phase system consisting of an intracellular phase, extracellular phase and the intervening membrane. The relative potential difference between the interior and the exterior of the membrane, the resting membrane potential ($E_m$), is a consequence of the permeability characteristics of the membrane and the ionic composition of the bathing fluids. The close approximation between the actually measured resting membrane potential and that calculated from the ratio of intracellular ($K_i$) to extracellular potassium ($K_o$) by application of the Nernst equation

$$E_m = \frac{RT}{F} \log \frac{K_i}{K_o}$$

indicates that potassium is the main determinant by virtue of its very high membrane permeability. 23

Calcium is considered a membrane stabilizer. It has, in common with numerous other drugs in this category (local anesthetics, antihistamines, prostigmine), the ability to alter excitability while having little or no effect on resting membrane potential. 24, 25 Calcium has been demonstrated to protect against depolarizing stimuli, presumably by affecting membrane permeability to potassium and sodium. Removal of calcium has a reverse effect. Evidence that calcium acts by virtue of reducing permeability across the cell membranes has been confirmed by direct studies of radiopotassium flux. 26 Indirect measurement of sodium and potassium permeability using the voltage clamp technique on the giant squid axon have also revealed that excess calcium produces a reduction of their movement. 27 Studies on the myocardial transport of sodium and potassium during depolarization again reveal that hypercalcemia augments the capacity of the myocardial tissue to alter its permeability. 28

Although the precise mechanism by which calcium affects membrane permeability is unknown, the similarity between hyperpolarization and elevated calcium, and the rapidity with which calcium acts, indicate a membrane surface phenomenon. In this regard Shanes 29 has introduced the concept of calcification of fiber surfaces. Radio-calcium studies have revealed that calcium is bound to fiber surface sites and that the positive ionotropic effects of hypercalcemia reflect a stoichiometric competition for sodium and potassium. 30 From these studies it has been postulated that there is a competition between calcium and potas-
sium and sodium for the sites of transport through the membrane and that the level of bound membrane calcium (Ca\textsubscript{m}) is crucial to their permeability.\textsuperscript{21}

Calcium has also been demonstrated to affect myocardial contraction directly. The importance of environmental ionic calcium in the activation of myosine ATPase is well known. Heilbrunn.\textsuperscript{22} has also stressed the fact that ionized calcium is the most potent of all physiological ions injected into muscle in eliciting contraction (in comparison to sodium, potassium, magnesium and ATP). Since there is also evidence which suggests that the actual calcium flux into and out of myoplasm occurs during depolarization, these findings may indicate that the release of bound membrane calcium within the cell may be the process by which depolarization is linked to contraction in the myocardium.\textsuperscript{31}

The similarity of action of cardiac glycosides and calcium on myocardial excitability and contractility has been alluded to in the past.\textsuperscript{23} It has been suggested that this resemblance indicates that calcium and the glycosides affect the same parameter of myocardial excitation, that of membrane permeability. Indeed, both are considered stabilizers, pharmacologically. More recent evidence such as the inability of digoxin to act in the absence of calcium is not totally inconsistent with the fact that digitals affects membrane bound calcium (Ca\textsubscript{m}), vide supra.\textsuperscript{33}

Preoperative and Postoperative Management in Hypercalcemia

The best care that can be given the hypercalcemic patient approaching operation consists in the careful restoration, either orally or intravenously, of the losses of water, sodium and potassium measured by analysis of urine and plasma. Hypercalcemic crisis (parathyroid coma) has been described by numerous investigators. This is a condition wherein renal failure, cardiac arrhythmias, congestive failure, obstipation, mental deterioration and coma ensue, as the plasma calcium rises to high levels (18–24 mg./100 ml.). The benefits that accrue from the use of sodium versenate or the artificial kidney in lowering plasma calcium levels are only temporary, for the calcium will return to previously high levels within one hour after cessation of such therapy. The treatment for such a crisis, therefore, is immediate cervical exploration and resection of the hyperfunctioning gland.

While digitalis has been emphasized as being similar to calcium in the effect on cardiac excitability, this has not been generally recognized in treating patients with hypercalcemia and congestive heart failure. A detailed discussion of the interplay among calcium, magnesium, sodium and potassium in the preoperative hyperparathyroid patient is beyond the scope of this paper. In addition, patients coming to operation for parathyroidectomy often are given a number of other drugs which may modify their response to digitalis. All of this must be borne in mind both in pre- and postoperative care.

While the administration of calcium intravenously to an already digitalized patient may not be as dangerous a procedure as heretofore postulated,\textsuperscript{34} the reverse situation is felt to carry an entirely different significance. One should likewise not assume that giving digitalis to a patient made hypercalcemic by calcium salt given intravenously is the same as digitalizing the chronically hypercalcemic patient.\textsuperscript{22} This has been borne out repeatedly in clinical situations, although experiments designed to show the difference between acute and chronic hypercalcemia as regards digitalis sensitivity have never been published.

Digitalization of the hypercalcemic patient, therefore, should not be undertaken except in the most extreme circumstances. In the already digitalized patient, the digitalis requirements may be somewhat decreased. In a patient not previously digitalized and in congestive heart failure, one should never give digitalis intravenously since rapid death from toxicity may ensue, even with a minute dose. Oral or intramuscular doses of digitalis or its derivatives must be used cautiously, and the patient should be checked carefully before each dose for development of arrhythmias.

Postoperative care can only be governed by the clinical state of the patient following operation. If tetany appears, it will usually be within twelve to twenty-four hours (sometimes within six hours) following removal of a hyper-secreting parathyroid gland. Tetany is the result of insufficient secretion of parathormone.
from the remaining glands due to suppression by the hyperfunctioning gland(s) now removed. Preoperative administration of vitamin D and calcium is not only hazardous and contraindicated, but may mask the effects of removal of the parathyroid adenoma, and lead to confusion. Generally, one should allow the patient to approach tetany, for this proves that an adenoma has not been overlooked by the surgeon. The treatment of the tetany is simple and consists in the immediate administration of calcium salts orally or intravenously, and the institution of calciferol or other vitamin D therapy until the patient stabilizes his plasma calcium at a low normal or normal level. Permanent hypoparathyroidism is rare but obviously depends on the skill of the surgeon and how much functioning parathyroid tissue has been left behind. Oftentimes, the patient who develops a cardiac arrhythmia as a result of hypercalcemia will revert to normal sinus rhythm merely from the lowering of plasma calcium. If there was extensive osseous disease preoperatively, it is generally felt that the plasma calcium will fall more rapidly and take a longer time to rise postoperatively, due to the “hungry bone” phenomenon as described by Fuller Albright. Recalcification of these lesions is often apparent within one month following parathyroid removal, but may take as long as one year for complete restitution.

**Hypoparathyroidism**

The problems of the hypoparathyroid patient approaching surgery are obviously quite different from those of the hypercalcemic patient, and are usually manifested by tetany.

The principle causes of tetany are hypercalcemia and alkalosis. Of the nonparathyroidal causes of hypocalcemia, rickets, osteomalacia, steatorrhea and renal insufficiency are the most common and can easily be diagnosed by their associated clinical pictures.

**Etiology, Symptoms and Signs**

Primary parathyroid deficiency is extremely rare, usually occurring in patients under the age of 16 and often persisting throughout adult life. In most instances, clinical evidence of parathyroid deficiency is secondary to thyroidecetomy, appearing within 12–24 hours postoperatively but occasionally seen as long as one week following a thyroid operation. During the past decade increased knowledge and experience in surgical techniques have decreased the incidence of permanent parathyroid insufficiency secondary to operation. Transient deficiency is not unusual and may be attributed to trauma, edema and hemorrhage with temporary interference of the blood supply to the remaining parathyroid glands. There have been no documented cases of hypoparathyroidism secondary to the administration of radioactive iodine in the treatment of thyrotoxicosis.

The pronounced disturbance of calcium and phosphate metabolism in hypoparathyroidism is reflected by plasma calcium levels as low as 5 mg. per cent and plasma inorganic phosphate levels as high as 9–12 mg. per cent while the alkaline phosphatase is normal. The decrease in calcium facilitates the transmission of impulses across the myoneural junction which is responsible for much of the clinical picture.

The most striking symptom is increased neuromuscular excitability resulting from the decreased plasma ionized calcium. Most patients complain of tonic equivalents or frank tetany as manifested by carpo-pedal spasm in which the stiff hollow hand with rigid fingers is marked by flexion at the metacarpal-phalan-geal joints, the wrist and the arm at the elbow joints; the legs and feet are extended. Tonic and clonic convulsions, laryngeal stridor and spasm may prove fatal while numbness, muscle cramps, dysphagia, dysarthria, and cardiac irregularities oftentimes are seen. Spasm may involve the smooth muscle of the eye, the gastrointestinal tract, bladder, and blood vessels. Approximately 40 per cent hypoparathyroid patients are seen because of epileptic seizures, and the electroencephalographic findings in these patients suggest that there are occasionally underlying factors unrelated to the plasma calcium level that play an important part in lowering the threshold for convulsive seizures. Mental changes are frequent and include anxiety, depression, increased irritability and psychoses. Acute symptoms of tetany may be precipitated by infection, undue fatigue, overbreathing, menstruation and emotional upsets, as well as by increased phosphate content of the diet. In some cases the symptoms may
be mild, variable or even vague. Patients have manifested fatigue, muscular weakness, palpitations, numbness and tingling of the extremities and other signs of latent tetany for as long as 30 years before a diagnosis of chronic hypoparathyroidism was established.

Neuromuscular excitability can easily be demonstrated by contraction of the facial muscles in response to a light finger tap over the facial nerve in front of the ear (Chvostek’s sign). This sign is almost always positive in untreated hypoparathyroidism; however, it appears occasionally in normal individuals. Trousseau’s sign is obtained by occluding the circulation to the arm with a blood pressure cuff, the pressure raised slightly above the systolic level, and observing for development of carpo-pedal spasm.

Physical examination in chronic hypoparathyroidism may reveal extensive atrophic ectodermal changes. The hair may be sparse, prematurely gray and occasionally absent in the axillary and pubic regions. The skin is usually rough, dry and scaly, and there may be papules, vesicles or bullae present. Generalized or patchy erythema may also be found. The nails are deformed and brittle and show transverse ridging. Cataracts are frequently present, and their extent is directly related to the duration and severity of the hypocalcemia. Early lens changes can usually be found with the aid of a slit lamp. Papilledema has been observed in a few cases. The ECG will usually show a prolongation of the Q–T interval, and the density of bone in the roentgenogram may appear normal or increased. Abnormalities of dentition such as deformed or absent roots may be helpful in determining the age of onset of the disease. In primary hypoparathyroidism bilateral symmetrical calcification of the basal ganglia is often seen on skull films. Other areas such as the cerebellum and choroid plexus are occasionally calcified.

Preoperative and Postoperative Management of Hypoparathyroidism

It is imperative that a thorough study of the hypoparathyroid subject coming to operation be carried out. Those cases of hypoparathyroidism resulting from extensive thyroid surgery of any nature should be investigated as to residual thyroid function, since one of the causes of persistent and intractable tetany is hypothyroidism. Oftentimes restoration of the circulating thyroxin level by any one of the thyroid extracts commercially available will help in the management of the chronically hypoparathyroid subject. This is a fact not readily appreciated and, if remembered, can lead to gratifying results in the treatment of chronic hypoparathyroidism, as well as acute hypoparathyroidism. The evaluation should also include a neurological examination designed to elicit the degree of neuromuscular excitability present in each patient. Careful cardiac evaluation including an electrocardiographic examination for arrhythmias is also important. Fundoscopic examination for papilledema is mandatory. Since patients with hypoparathyroidism, without other abnormalities, may have increased cerebrospinal fluid pressure, lumbar puncture for spinal anesthesia should be done with utmost caution, if this is the case.

It is obvious that the hypoparathyroid patient will be a safer candidate for surgery if the plasma calcium is brought within normal limits. Therefore, the object of therapy in chronic hypoparathyroidism is to reduce the plasma phosphate and to raise the plasma calcium level to within normal levels. This can best be accomplished by a combination of dietary and drug therapy. The diet should include as much calcium as can be tolerated but there is very little to be gained by reducing the amount of phosphate in the diet unless it is consumed to excess. If additional calcium by mouth is needed, it can be given in the form of calcium chloride, 20 per cent solution, (1 tablespoon three times a day in orange juice or cola drink) or as calcium lactate, 2 to 3 g. per day. Since dihydrodrotachysterol is the most rapidly acting vitamin D derivative, it is the drug of choice if operation is planned within a short time. One can expect the plasma calcium level to rise in response to dihydrodrotachysterol within three to five days after initiation of therapy. The initial dose of dihydrodrotachysterol should be within the range of 250,000 units (6.25 mg.) for the first five days, and then gradually lowered to a maintenance dose of from 50,000 to 100,000 I.U. Following operation other vitamin D preparations can be utilized, since the cost of dihy-
drotachysterol is exorbitant and not required for the long term care of the hypoparathyroid subject. It is necessary that tetany be adequately treated before operation is attempted, since apprehension may cause the patient to hyperventilate before induction of anesthesia and blow off carbon dioxide (CO₂). The resultant respiratory alkalosis enhances the neuromuscular excitability that accompanies a chronically lowered plasma calcium level. Most of the agents used during anesthesia which block neuromuscular transmission, such as curare, are well tolerated by the hypoparathyroid subject, and no special care need be taken in this regard. Preanesthetic medication, such as atropine, is not contraindicated in these patients.

The management of the acutely hypoparathyroid subject either pre- or postoperatively is concerned with the immediate correction of hypocalcemia and can best be accomplished by the slow (1 g. every 30 minutes until 4 g. are given) intravenous infusion of calcium gluconate or calcium chloride. The effect, however, is transitory lasting only a few hours, and additional calcium may be required. Caution should be exercised with patients on digitalis, since rapid infusion of calcium in these cases may cause cardiac arrest, although this is extraordinarily rare phenomenon (vide supra). Parathyroid extract may be given with the calcium offering a more prolonged action on the plasma calcium level. It is rarely used except in critical situations, since it must be given daily and may be associated with local anaphylactoid reactions (although anaphylactoid reactions have not been seen by the authors, they are described, though inadequately documented).

The anesthesiologist should be aware of the fact that tetany can develop in a very short period of time under the influence of varying stimuli. The development of alkalosis from whatever cause during operation is to be avoided under all circumstances, since this will lead to rapid appearance of tetany or its equivalents. It is not unwise in a patient difficult to regulate to give a slow drip of calcium gluconate during operation, to prevent development of tetany at the time.

Summary and Conclusions

Careful measurement of the complex biochemical changes in various states of parathyroid dysfunction allows for better proper pre- and postoperative care. The effects of hypercalcemia are for the most part exerted on the kidney and heart, although there is decreased neuromuscular excitability as well. Careful restoration of losses of various electrolytes and water is paramount, and often restores abnormal renal function to almost normal levels. Digitalis in chronically hypercalcemic states is not only hazardous but usually not indicated if early operation is planned. The use of hypocalcemic agents such as sodium verenate and of hemodialysis are usually of little or no help in the management of hypercalcemia. Prompt operation is the only known cure for hyperparathyroidism.

The problem in treating the acutely hypocalcemic patient with tetany consists mainly in the intravenous use of calcium salts. Other agents such as vitamin D or Benemid (probenecid) and dietary regulation are reserved for the patient with chronic tetany. It has been stressed that it is hazardous to plan to operate on a patient with hypocalcemia, as inappropriate preanesthesia medication may affect the tetany in a deleterious manner. Postoperative tetany following removal of hyperfunctioning parathyroid tissue, inadvertent removal, or trauma to the glands is usually transient and as such constitutes no major problem.

This work was supported in part by Grant A-3967 (N.I.H.) and funds from the Massachusetts Chapter of the Arthritis and Rheumatism Foundation.

References


