A recent review of all the cases of hypoparathyroidism seen in the State University of Iowa Hospitals\(^1\) revealed an apparent increase in incidence of the surgical form of this disorder in children and adolescents during the past decade. For this reason it seemed worthwhile to scrutinize more closely the cases appearing in this age group. It is the purpose of this report to summarize the cases of hypoparathyroidism seen in children and adolescents at this hospital, to discuss their management and prophylaxis, and to compare the disorder in this age group with that seen in adults.

The reason for the increased incidence of secondary hypoparathyroidism in the younger age groups is largely twofold. First, carcinoma of the thyroid has been seen more frequently in this hospital during the past decade.\(^2\) The forms of therapy employed have been primary surgical ablation of the neoplasm or facilitation of I-131 therapy by surgical removal of all normal thyroid tissue. Damage to, or removal of, parathyroids during the course of these surgical procedures partially accounts for the increased incidence of hypoparathyroidism.

Secondly, the incidence of secondary hypoparathyroidism may have increased because near total thyroidectomy is now being used in the surgical treatment of thyrotoxicosis in children. This has followed from the observed higher recurrence rate in young thyrotoxic patients as compared with adults,\(^3\) and the observation that the single most important factor in avoiding recurrent hyperthyroidism is the amount of thyroid tissue removed.\(^4\) Although administration of radioactive iodine is rapidly replacing surgery as the preferred treatment of thyrotoxicosis in adults and the age limit for I-131 treatment is being lowered progressively as satisfactory long-term results accumulate, surgery remains a common therapeutic measure in children. The hazards of treating thyrotoxicosis with I-131 are unknown for children with their long life expectancy. Medical treatment has not obviated the need for surgery in some children.

**PATIENT MATERIAL**

Eight cases of hypoparathyroidism in patients less than 17 years of age at the time of diagnosis have been seen at the State Uni-
versity of Iowa Hospitals. The first patient was seen in 1946. The longest follow-up period was 7 years and the shortest 3 months. All but one are currently under observation. One patient had primary hypoparathyroidism, and seven cases were secondary to thyroid surgery. Among the latter, three followed subtotal thyroidectomy done for thyrotoxicosis and four followed total thyroidectomy performed for carcinoma. The patient with primary parathyroid deficiency will be presented first.

CASE REPORTS

Case 1 (55-1705)

A 9-month-old white male was admitted to the Department of Pediatrics with a 4-day history of multiple short episodes of rigidity and clonic movements of the extremities, breath-holding and cyanosis. The past history was remarkable only because of a 3-day episode of twitching of the face and extremities noted at the age of 1 week, which disappeared after calcium was administered for a few days. The infant appeared normal except for a temperature of 39°C (102°F), laryngeal stridor and a positive Chvostek's sign. Primary hypoparathyroidism was suspected at the time of first examination. The diagnosis was confirmed by the finding of a concentration of calcium in the serum of 5.0 mg/100 ml and phosphorus 9.8 mg/100 ml. A negative lumbar puncture, normal roentgenograms of skull and chest, concentration of urea nitrogen in the blood of 11 mg/100 ml and pH of the blood 7.36 gave additional indirect support for the diagnosis. An Ellsworth-Howard test revealed a response to exogenous parathyroid hormone, thereby classifying the case as idiopathic, rather than pseudohypoparathyroidism. Symptomatic improvement followed parenteral administration of calcium. Definitive therapy consisted of oral administration of vitamin D₂, aluminum hydroxide and calcium gluconate. The dosage schedule and course are

Fig. 1. Case 1. Nine-month-old white male with primary idiopathic hypoparathyroidism. This case illustrates rapid control of the disorder in spite of handicaps imposed by the age of the patient and a diet (milk) high in content of phosphorus.
shown in Figure 1. During the lag period before normal concentrations of phosphorus were present in serum the patient was asymptomatic. Later, hypercalcemia and hypophosphatemia indicated overtreatment which was corrected by stepwise reduction in the medication.

Parathyroid insufficiency in an infant is most unusual. Steinberg and Waldron collected 52 cases of primary hypoparathyroidism in a review of the literature. None of the patients was less than 1 year of age at the onset of symptoms and the youngest patient was 3½ years of age at the time the diagnosis was made. In the patient herein reported it is a matter for speculation whether the short episode of twitching of face and extremities at the age of 1 week was evidence of tetany of the newborn or an incipient manifestation of hypoparathyroidism. However, there is little doubt that the convulsions at the age of 9 months were from parathyroid insufficiency. The early age of onset suggests agenesis or developmental failure of the parathyroid glands. The early diagnosis and treatment should obviate the tragic complications often associated with longstanding uncontrolled hypoparathyroidism, provided adrenal insufficiency does not appear.

The early persistence of hyperphosphatemia was attributed to the high phosphorus content of the milk formula which constituted the major portion of the infant’s diet. When solids low in content of phosphorus were substituted for part of this formula there was further improvement in the concentration of calcium and phosphorus in the serum. The patient was asymptomatic during the period of hyperphosphatemia after initiation of therapy but could not be considered well controlled from the standpoint of chemical studies of the serum. This underscores the need for complementing clinical observations with laboratory information. The absence of signs and symptoms of increased neuromuscular irritability does not necessarily imply that the patient is being adequately treated.

**Case 2 (53-1793)**

A 13-year-old white girl was admitted to the hospital with a 7-month history of hyperthyroidism. Propylthiouracil therapy was administered. After she became euthyroid, Lugol’s solution was given and 10 days later bilateral subtotal thyroidectomy was performed. It was estimated that 6 gm of thyroid tissue were left. All four parathyroid glands were identified and preserved. The patient developed hoarseness, paresthesias and occasional carpopedal spasm 7 weeks postoperatively. The diagnosis of hypoparathyroidism was confirmed by the findings of concentrations of calcium and phosphorus in the serum of 7.4 and 8.4 mg/100 ml, respectively, and a negative Sulkowitch test. Treatment was started with vitamin D₃, 50,000 units daily, calcium, 3 gm/day orally and aluminum hydroxide. Restriction of dietary phosphorus was not emphasized. Symptomatic control was excellent, but for 5 months the concentration of calcium in the serum varied from 8.4 to 6.5 mg/100 ml and that of phosphorus from 7.8 to 7.0 mg/100 ml. Thereafter, the values gradually reverted toward normal over a 2-year period, during which time no change in medication or dosage was made. All therapy was then withdrawn. The patient has been followed for 8 additional months, during which time she has been asymptomatic. She has had low normal concentrations of calcium and phosphorus in the serum, minimal calciuria and an occasional positive Chvostek’s sign. Recently, a calcium infusion test was performed. The post-infusion rise in concentration of phosphorus in the serum was disproportionately less than the rise in excretion of phosphorus in the urine on the day of the test as compared to the excretion of phosphorus on the control day. The results are considered inconclusive in quantitating the degree of parathyroid dysfunction since they do not fulfill the criteria of normal or subnormal parathyroid function as set forth by Howard.

This case of secondary hypoparathyroidism was delayed in onset. It developed in spite of the fact that the parathyroid glands were identified and left undisturbed at the time of surgery; this would tend to indicate interference with blood supply and/or postoperative edema and fibrosis as etiologic agents in the production of the disorder. Initial poor control of the hypopara-
thyroidism was related to inadequate dosage of vitamin D₉ and also to the inclusion of too much phosphorus in the diet. Spontaneous remission apparently occurred after a somewhat protracted period. She is still being followed because the inconclusive results of the calcium infusion test throw some doubt on the functional status of the parathyroid glands.

Case 3 (46-5629)
A 16-year-old white girl was first seen 9 months after a thyroidectomy performed elsewhere for thyrotoxicosis. She was asymptomatic post-operatively for 2 months, at which time she began to experience paresthesias, leg cramps and carpopedal spasms, which became progressively more severe. A convulsion occurring 3 months after the operation prompted a visit to her physician. Hypoparathyroidism was suspected and vitamin D₉, ParaThorMone® and calcium therapy orally were begun in unknown doses. Symptoms promptly disappeared and she was later referred for definitive management. At the time of admission to the hospital, the physical examination was unremarkable except for a positive Trousseau's sign. The concentration of calcium in the serum was 7.7 mg/100 ml and that of phosphorus 6.9 mg/100 ml. One milliliter of AT-10 was administered three times weekly and calcium was given orally (plus large intake of milk). The concentration of calcium in the serum varied from 7.0 to 8.0 mg/100 ml and the concentration of phosphorus from 9.5 to 6.0 mg/100 ml for 4 years. She was asymptomatic during this time and had only occasionally positive Trousseau's and/or Chvostek's signs. During the next 3 years the concentration of phosphorus ranged between 5.6 and 4.2 mg/100 ml, and the Chvostek's and Trousseau's signs remained negative. This improvement was coincident with a decrease in intake of milk. Follow-up has not been possible for the past 4 years.

This case again emphasizes how misleading clinical evidence can be regarding control of hypoparathyroidism, and the need for tests of calcium excretion in the urine and occasional determinations of concentrations of calcium and phosphorus in serum as yardsticks for the adequacy of therapy. Decrease in the concentration of phosphorus in the serum and disappearance of the Chvostek's and Trousseau's signs during the last 3 years this patient was followed may be attributed to the decrease in amount of phosphorus ingested.

Case 4 (56-5111)
A 12-year-old white girl was admitted to the hospital with thyrotoxicosis of 1 year's duration, partially controlled by propylthiouracil. Bilateral subtotal thyroidectomy was performed after additional preparation. Parathyroid tissue was identified and preserved at the time of surgery. The surgical specimen showed parathyroid tissue within the thymus. Severe tetany developed 24 hours after operation and was controlled by intravenous administration of calcium. At this time the concentrations of calcium and phosphorus in the serum were 8.8 and 5.2 mg/100 ml, respectively; Chvostek's sign was positive and the urinary Sulkowitch test for calcium was negative. Definitive therapy consisted of administration of 50,000 units of vitamin D₂ and 12 gm/day of calcium orally. She remained asymptomatic with normal concentrations of calcium and phosphorus in the serum for 6 months. All therapy was then withdrawn to test the assumption that the parathyroid deficiency was temporary in nature. However, 1 month later she developed paresthesias and tetany relieved by intravenous administration of calcium gluconate, and the above therapy was resumed. We plan a second period of withdrawal of therapy in the near future to see if hypocalcemia manifestations recur, and to do a calcium infusion test should doubt as to the parathyroid function status remain.

Again of interest in this case was the development of tetany when the surgeon thought parathyroid tissue had been left. The ectopic parathyroid tissue within the thymus is a clinical reminder of the common embryologic derivation of the lower pair of parathyroid glands and thymus from the third branchial pouch.

Case 5 (52-9211)
A 9-year-old white female was admitted to the hospital with a slowly enlarging mass in the right side of the neck of several months' dura-
tion. Metastatic carcinoma of the thyroid had been proven by biopsy of a cervical node. During a total thyroidectomy and thyrectomy, three parathyroid glands were identified and removed with the en bloc excision of the extensive thyroid malignancy. The left superior parathyroid gland was identified and preserved. The patient developed laboratory evidence of hypoparathyroidism 8 days after this procedure, with a concentration of calcium of 8.2 mg/100 ml and phosphorus of 6.8 mg/100 ml. Oral administration of calcium, 11 gm/day (administered in milk), AT-10, 1.25 ml daily and aluminum hydroxide were begun. At the time of the first admission to this hospital 3 weeks after the operation, myxedema was apparent. Administration of thyroid extract was started and the oral administration of calcium and AT-10 continued, as shown in Figure 2. Therapeutic 125I was also given. During the succeeding 10 months, there were no signs or symptoms of hypoparathyroidism and the described treatment program was continued. Primary interest in the thyroid malignancy during this period interferes to some extent with documentation of the control of the hypoparathyroid state. Later, a radical dissection of the left side of the neck was done. The patient was discharged while receiving thyroid extract only, to determine if parathyroid insufficiency persisted. She was asymptomatic until thyroid extract was discontinued 2 weeks prior to the next visit, when she began to experience paraesthesias and episodes of carpal spasm which responded to oral administration of calcium. At this time the concentration of calcium in the serum was 5.4 mg/100 ml and that of phosphorus 9.4 mg/100 ml and Chvostek’s and Trousseau’s signs were present. Therapy was resumed, substituting vitamin D2, 100,000 units daily for AT-10. All signs and symptoms of increased neuromuscular irritability and hypo-

![Graph of calcium and phosphorus concentrations over time with annotations for RT Radical Neck Dissection, Paresthesia, Carpal Spasm, Tinnitus, and Cramps in Arms & Legs Tinnitus.](image)

**Fig. 2.** Case 5. Nine-year-old white female with carcinoma of the thyroid treated initially by total thyroidectomy. For 2 years the signs and symptoms of hypoparathyroidism were mild enough not to demand treatment. Fortunately, adequate hypoparathyroid therapy was begun before cataracts and other complications of chronic hypocalcemia developed.
thyroidism disappeared. Within 1 month the patient resumed her normal intake of milk and discontinued the parathyroid substitution therapy on her own volition. Again she was asymptomatic until discontinuing thyroid extract prior to the next return visit, when paresthesias and tinnitus returned. At this time Chvostek's and Trousseau's signs were again positive. The concentration of calcium in serum was 5.4 mg/100 ml and that of phosphorus 8.2 mg/100 ml, indicating parathyroid insufficiency; in addition, there was clinical and laboratory evidence of myxedema. Since resumption of treatment, the patient has been seen on many occasions, each time discontinuing thyroid extract 2 weeks prior to admission but continuing the calcium and vitamin D₃ therapy. She has remained asymptomatic during this interim except for signs and symptoms of low metabolism during the 2 weeks of abstinence from thyroid therapy preceding each return visit. She has maintained satisfactory concentrations of calcium and phosphorus in serum and has shown no evidence of increased neuromuscular irritability. During this 2-year period she has taken the prescribed drugs faithfully and has performed urine Sulkowitch tests daily.

This case is interesting from many aspects. Evidence of panathyroid insufficiency was present during the first 10 months despite treatment, probably because of excessive intake of milk and inadequate doses of AT-10. It has been well controlled the last 2 years with low dietary intake of phosphorus and high doses of vitamin D₃. AT-10 is as effective as vitamin D₃ in the treatment of hypoparathyroidism, but the latter is favored because it is by far the cheaper of the two drugs. Expensive drugs are conducive to many lapses in control. During the early days of therapy when AT-10 was the sole available preparation, there were many patients who could not afford control of a parathyroid disorder.

An apparent relationship between the control of thyroid and parathyroid deficiency was demonstrated on two different occasions. When thyroid extract alone was given, no signs or symptoms of increased neuromuscular irritability were manifested, but when the thyroid therapy was withdrawn, subjective and objective evidence of increased neuromuscular irritability appeared coincident with laboratory evidence of thyroid deficiency. This seeming relationship was investigated recently by withdrawing thyroid and parathyroid substitution therapy concurrently. While in this state, the concentrations of calcium and phosphorus in serum were followed closely. A calcium infusion test showed a rise in post-infusion concentration of phosphorus in the serum which was disproportionately less than the percentile increase in the urinary excretion of phosphorus on the control day. These results confirmed the presence of hypoparathyroidism according to Howard's criteria. Tri-iodo-thyronine was given for its rapid action in restoring normal metabolic activity. No appreciable improvement in the concentrations of calcium and phosphorus in the serum was noted during the short period observed. This suggests that euthyroidism may protect the patient from experiencing the subjective manifestations of increased neuromuscular irritability when in a hypoparathyroid state, but that it apparently does not directly alter concentrations of calcium or phosphorus in the serum.

Case 6 (53-14989)

A 16-year-old white female was referred for treatment of carcinoma of the thyroid which had been proven by biopsy of one of several slowly enlarging cervical masses present for 4 years. Physical examination revealed an enlarged thyroid with palpable bilateral cervical nodes. A roentgenogram of the chest was normal. No parathyroid tissue was identified at the time of surgery. Postoperatively, therapy was begun "prophylactically" since it was assumed that the parathyroid glands had been included in the specimen. Fair control of the concentration of calcium in the serum was maintained initially but the concentration of phosphorus varied from 4.7 to 6.0 mg/100 ml, probably due to the large intake of milk inadvisably recommended. The patient discontinued therapy on her own volition 2 months postoperatively. She returned in 4 months complaining of paresthesias. A positive Chvostek's sign was elicited, and the concentrations of cal-

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Calcium and phosphorus in the serum were 6.6 and 5.6 mg/100 ml, respectively. Therapy was re-instituted with vitamin D₂, 50,000 units twice weekly, AT-10, 1 ml daily and calcium, 8 gm/day orally. Because of myxedema, administration of thyroid extract was started. Four months later the concentration of calcium in the serum had risen to 7.9 mg/100 ml, that of phosphorus had fallen to 4.1 mg/100 ml and the patient was asymptomatic. Over the succeeding 8 months the concentration of calcium varied from 9.0 to 9.4 mg/100 ml, and the concentration of phosphorus from 5.4 to 5.2 mg/100 ml. Then, all parathyroid substitution therapy was withdrawn to see if the hypoparathyroidism were still present. Recurrence of the signs and symptoms of parathyroid insufficiency prompted resumption of therapy with vitamin D₂, 50,000 units every other day, and AT-10, 1.0 ml twice weekly. Recently, an intravenous calcium infusion test was performed while the patient was receiving full therapy for hypoparathyroidism. The test resulted in a decrease in the post-infusion concentrations of both calcium and phosphorus, but no change in urinary excretion of phosphorus. These results do not fall into any of Howard's categories of parathyroid function. At the time of writing, the patient is euparathyroid with continued hypoparathyroid hypoparathyroidism.

This case emphasizes the technical difficulty of identifying and preserving parathyroid tissue coincident to ablation of a locally invasive thyroid malignancy. Secondly, it illustrates again that milk and other foods having a high content of phosphorus are to be avoided. Thirdly, it demonstrates the use of periods of trial without therapy in determining persistence of parathyroid deficiency. No arbitrary time limits can be set for this evaluation, although in our experience patients with hypoparathyroidism lasting longer than 1 year do not regain normal parathyroid function.

Case 7 (54-10627)

A 10-year-old white female had a history of a gradually enlarging mass in the right side of the neck associated with hoarseness of 3 weeks' duration. The past history was interesting in respect to thymic enlargement with stridor noted at 7 days of age, which was treated by three doses of local x-ray therapy. Physical examination revealed the right thyroid lobe to be enlarged and nodular, and laboratory studies indicated a euthyroid state. Total thyroidectomy and radical dissection of the right side of the neck were done. Histopathologic study showed carcinoma of the thyroid with involvement of lymph nodes; no parathyroid tissue was identified grossly or microscopically. Hypoparathyroidism developed in the early postoperative period. As indicated in Figure 3, early therapy was unnecessarily vigorous, resulting in repeated vomiting, drowsiness and hypercalcemia 3 weeks postoperatively. All parathyroid substitution therapy was then discontinued. Two months later signs of hypoparathyroidism had recurred, as evidenced by an intermittently positive Trousseau's sign as well as concentrations of calcium and phosphorus in serum of 7.6 and 6.6 mg/100 ml, respectively. Administration of calcium orally, as well as inadequate dosages of vitamin D₂, were resumed. Since this failed to correct the clinical or laboratory evidences of parathyroid deficiency, small doses of AT-10 and large doses of vitamin D₂ were added to the treatment program. The concentrations of calcium and phosphorus thereafter approached normal levels. One year later hypercalcemia and hyperphosphatemia were seen to coexist, promoting discontinuance of administration of AT-10. Recently a calcium infusion test was performed. The findings were those typically seen in hypoparathyroid patients, namely, an increase in the post-infusion concentrations of both calcium and phosphorus in the serum, as well as in urinary excretion of phosphorus, on the day of the test. The daily dosage of vitamin D₂ has been increased to 100,000 units; 10 gm/day calcium orally has been continued and a limited intake of phosphorus encouraged. The hypoparathyroidism is now adequately controlled.

Of interest in this case, though not germane to the subject under discussion, is the association of thymic irradiation and the subsequent development of thyroid cancer. There are many recent references in the literature to this association, the explanations ranging from a truly causal relationship to one purely of coincidence.
The difficulty of identifying and preserving parathyroid tissue when total thyroidectomy is done is again illustrated. Thirdly, this case is an example of overly vigorous initial treatment. Although rare, there is an occasional patient in whom it is difficult to find the proper dosage for long-range control. A final point of interest is the coexistence of hyperphosphatemia and hypercalcemia observed in this patient. This has not been seen in any of our other patients, and occurred here with a high intake of phosphorus coincident with adequate parathyroid substitution therapy.

**Case 8 (54-7162)**

A diagnosis of thyroid carcinoma was made elsewhere in a 13-year-old white girl on the basis of biopsy of a cervical node. A hard mass in the right lobe of the thyroid and a few bilateral small cervical nodes were found. Roentgenogram of the chest was interpreted as normal. Total thyroidectomy and radical dissection of the right side of the neck were performed. One parathyroid gland was preserved. Microscopic examination of the surgical specimen revealed adenocarcinoma of the thyroid with metastases to lymph nodes. Hypoparathyroidism was suspected on the third day after surgery when the patient complained of paresthesias of the extremities. This impression was supported by a persistently positive Chvostek’s sign, a concentration of calcium in the serum of 8.4 mg/100 ml and a concentration of phosphorus of 5.0 mg/100 ml. The paresthesias were relieved by intravenous administration of calcium and the positive Chvostek’s sign disappeared quickly after onset of treatment with
vitamin D₂, 50,000 units daily, and calcium lactate powder, 4 teaspoons daily. Six weeks later, treatment was discontinued. The patient has been asymptomatic and the concentrations of calcium and phosphorus in the serum have remained normal during the 1-year follow-up period.

This case emphasizes the benignity of temporary surgical hypoparathyroidism when suspected early on clinical grounds, confirmed by finding abnormal concentrations of calcium and phosphorus in the serum, and then properly treated. Such a patient suffers little disability or expense, but should be followed to rule out the persistence of masked or borderline parathyroid deficiency.

**DISCUSSION**

Hypoparathyroidism in children has essentially the same course as that seen in adults. At this institution, only 2 cases of primary hypoparathyroidism have been diagnosed; 1 was in a series of 45 cases of hypoparathyroidism in adults and 1 in this series of 8 cases in children.

In the cases studied at the State University of Iowa, the ratio of males to females was not significantly different in the two age groups, 1:7 in the pediatric age group and 1:5.4 in the adults. In the cases of secondary hypoparathyroidism, the ratio of benign to malignant thyroid disease was 4:3 in children as compared with 10:1 adults.

The day of onset of secondary hypoparathyroidism was comparable in the two age groups, occurring most often during the first 2 postoperative days. The onset was delayed several weeks postoperatively in two cases in the series herein reported.

As in adults, paresthesias and a positive Chvostek’s sign are the most frequent clinical manifestations. Trousseau’s sign is often positive in both age groups, but carpopedal spasm is infrequent in children. Demonstration of a low concentration of calcium and high concentration of phosphorus in the serum is the ultimate diagnostic criterion of hypoparathyroidism regardless of age, serving to confirm the diagnosis as well as to determine the baseline on which to establish the type and vigor of therapy.

The principles of management of hypoparathyroidism are the same in all age groups, and consist of administration of supplementary calcium orally, a diet low in content of phosphorus, and vitamin D₂ and/or AT-10. Management is difficult when there is a high intake of phosphorus which is inevitable when milk, dairy products and fish are included in the diet. Aluminum hydroxide combines with phosphorus and reduces its absorption from the gastrointestinal tract, although it is no substitute for restricting food high in content of phosphorus. If AT-10 is used, it should be given in daily doses of 1.0 to 2.0 ml. Vitamin D₂, 50,000 to 200,000 units daily, is effective and less expensive. Individual differences make it necessary to follow each patient carefully in order to arrive at the proper dosage, and occasional alterations in the treatment schedule may be required thereafter. Except for the infant, all of the children in the series herein reported required doses of drugs similar to those used in adults.

The aims of management are likewise similar: To maintain normal concentrations of calcium and phosphorus in serum and to ameliorate signs and symptoms of increased neuromuscular irritability. Success of treatment varies with the familiarity of the physician with the disorder and the degree of co-operation on the part of the patient.

For successful long-term management the patient and/or his family must understand the disorder, the aims of treatment, and the consequences of poor control. Co-operation is then achieved which is not otherwise obtained. Dosage schedules must be clearly outlined and the signs and symptoms of increased neuromuscular irritability, as well as those of hypercalcemia, must be enumerated. The importance of periodic examinations and occasional analyses of the blood must be stressed. The details of performing the Sulkowitch test, as well as the significance of the results, must be made clear to the patient. The inevitable complications of long-term hypocalcemia, in-
cluding lenticular cataracts, ectodermal changes and central nervous system dysfunction, should be explained. If properly presented, the possibility of occurrence of these complications can serve as motivation for greater co-operation with the physician.

In regard to secondary hypoparathyroidism thought to be "temporary" in nature, one is not justified in assuming that there is no longer need for continued scrutiny in such a patient simply because he is asymptomatic and maintaining acceptable concentrations of calcium and phosphorus in the serum a few months after all hypoparathyroid therapy has been discontinued. The tendency to take an either-or attitude regarding temporary versus permanent hypoparathyroidism is unfortunate, and not justified by the course of some of these patients. There are all degrees of severity, with exacerbation of symptoms at times sufficiently remote from the thyroidectomy that some patients have been treated for epilepsy, brain tumor and other erroneous diagnoses ultimately attributable to parathyroid insufficiency.\textsuperscript{15-17} One may speculate that such a patient could become symptomatic under the stress of ingesting a diet high in content of phosphorus and low in content of calcium and vitamin D, a change in endocrine milieu, or a combination of such circumstances. There is evidence to suggest that patients may even develop cataracts due to hypocalcemia that causes no signs or symptoms of increased neuromuscular irritability.\textsuperscript{1} It might be desirable for this reason to perform studies of parathyroid function, such as the calcium infusion test, on patients who have had "temporary" postoperative tetany, and to follow those patients who demonstrate abnormalities in parathyroid activity and yet are able to maintain concentrations of calcium and phosphorus in the serum within the ranges of normal. Of value in this respect would be tests capable of quantitating degrees of parathyroid lack although useful in demonstrating normal as well as frankly hypo- and hyperparathyroid states. In other words, it is of least value where the information regarding parathyroid function is needed most.

The risk of development of hypoparathyroidism after thyroid surgery should be one of the many considerations weighed in the planning of therapy, and in the technique of surgery for hyperthyroidism, nontoxic goiter and even carcinoma of the thyroid. This is especially true in the younger patient who might be faced with a lifetime of potential disability should surgery be elected and hypoparathyroidism result. Surgery may play a lesser role in the treatment of thyrotoxicosis in children as safer and more effective anti-thyroid drugs become available, and as more experience with therapeutic administration of radioactive iodine is gained. However, surgery will remain the primary method of therapy in carcinoma of the thyroid in the foreseeable future; the objectives: Ablation of the neoplasm or removal of all normally functioning thyroid tissue in preparation for effective therapy with radioactive iodine. If the object is removal of all normal thyroid in preparation for possible I-131 therapy rather than complete surgical ablation of the neoplasm, parathyroid tissue should be left even at the risk of including a few follicles of thyroid. It is not suggested that parathyroid preservation be placed ahead of the surgical cure of thyroid neoplasm. However, it is the surgeon's responsibility to pay careful attention to technical considerations relevant to a minimum of postoperative hypoparathyroidism. Like diabetes, hypoparathyroidism may be managed easily, but is not always well managed.

**SUMMARY**

Eight cases of hypoparathyroidism in patients less than 17 years of age have been presented and discussed. One case was primary in type and seven were secondary to thyroidectomies performed for thyrotoxico-
sis or carcinoma of the thyroid gland. The similarity of symptomatology, time of onset, treatment and long-term management between cases of hypoparathyroidism in the younger age group and in adults has been emphasized.

Prophylaxis against the development of hypoparathyroidism following thyroid surgery is the responsibility of the surgeon. When present, the disorder must be recognized early and treated promptly with adequate dosages of appropriate drugs. Conscientious long-term clinical and laboratory follow-up examinations are necessary to avoid the serious complications of chronic hypocalcemia. Tests to quantitate more accurately degrees of parathyroid function are needed in secondary hypoparathyroidism which is thought to be temporary in nature. Children and adolescents with hypoparathyroidism deserve special care in diagnosis, treatment and long-term management.

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