determined attempts to the most superficial gestures. In about a quarter there appeared to be no intention to die, but a hope of achieving some other aim, notably a change in attitude of another person in close relationship to the patient. In 63% of the patients the attempt appeared to be impulsive and unpremeditated. Half the cases were diagnosed as having depressive disorders. While a few cases show no psychiatric illness requiring treatment, all attempts at suicide require psychiatric investigation.

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ELECTROCARDIOGRAPHIC CHANGES IN IDIOPATHIC HYPERCALCAEMIA OF INFANCY

BY

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In 1952 Lightwood described the occurrence in infancy of a syndrome in which failure to thrive and constipation were found in association with a raised serum calcium level. This condition, to which the name idiopathic hypercalcaemia has been given, has since received considerable attention in the literature. To the symptoms already mentioned may be added thirst and polyuria, and also the association of mental deficiency, osteosclerosis, and cardiac murmurs in a group separately designated the "severe" type (Fanconi and Girardet, 1952; Schlesinger *et al.*, 1952). In the severe type the prognosis is grave, in contradistinction to the almost uniformly benign course of the majority. A connexion has been postulated between the development of this disorder and the intake of vitamin D (Lowe *et al.*, 1954), although this is still by no means certain.

The results of electrocardiographic investigation of infants with idiopathic hypercalcaemia have been given in some published reports, but there is no consistency in the abnormalities described (Fanconi and Girardet, 1952; Schlesinger *et al.*, 1952; Lowe *et al.*, 1954; Dawson *et al.*, 1954). In the belief that further information on this point would be desirable, electrocardiographic studies were undertaken on all patients coming under observation while suffering from this disorder.

Clinical Material

The material consisted of 24 patients aged from 4 to 92 weeks, admitted to one medical unit at the Royal Hospital for Sick Children, Glasgow, during 1955 to 1957. All conformed to the clinical picture described, and only one case was of the "severe" type (Case 9).

All cases showed at some point a serum calcium beyond the range encountered in healthy infants in this hospital—namely, 10.1 to 11.5 mg./100 ml. The highest values of serum calcium found in individual patients varied from 12 to 18.6 mg./100 ml.

Every patient had a history of prophylactic administration of vitamin D. The estimated total daily intake of vitamin D at the onset of symptoms and the highest serum calcium values in each patient are recorded in the Table.

Details of Cases

Case No.	Age at Admission (Weeks)	Daily Intake of Vitamin D at Onset of Symptoms (International Units)	Highest Recorded Serum Calcium (mg./100 ml.)	Changes in Contour of ST-T Complex	Remarks
1	32	1,120	16.6	Absent	
2	29	880	18.6	Present	
3	60	700	13.4	"	
4	33	1,716	15.3	"	
5	35	1,316	18.0	"	
6	48	1,120	15.7	"	
7	38	8,840	14.0	Absent	
8	20	1,450	13.9	"	
9	92	2,800	15.3	"	"Severe" type
10	37	500	12.8	"	Hyperchloraemic renal acidosis
11	21	2,200	13.0	Present	
12	4	700	12.9	Absent	Toxoplasmosis
13	35	2,700	15.8	Present	
14	20	1,500	12.0	Absent	
15	14	890	13.7	"	
16	36	1,050	16.2	Present	
17	61	3,950	14.2	"	Systolic murmur
18	33	1,866	13.8	"	
19	36	1,050	14.6	"	
20	26	1,680	14.5	"	Osteogenesis imperfecta
21	43	2,036	15.6	"	
22	4	336	12.3	Absent	
23	35	700	12.3	Present	
24	34	700	12.2	Absent	

One patient (Case 10) had hyperchloraemic renal acidosis as well as hypercalcaemia; one (Case 12) had toxoplasmosis; and another (Case 20) had osteogenesis imperfecta. With the exception of the patient with "severe" hypercalcaemia and one other (Case 17) each of whom had a systolic murmur, none had clinical evidence of heart disease.

Electrocardiographic studies were confined to the three standard limb leads and were carried out on the same day as that on which the serum calcium was estimated, but an electrocardiogram was not always obtained at the time of the highest serum calcium value.

The sensitivity of the electrocardiograph was standardized before each examination so that a current of one millivolt produced a deflection of one centimetre.

The number of electrocardiographic examinations of individual patients varied from one to seven. After one electrocardiogram was obtained, second and subsequent examinations were carried out only if the initial record was abnormal. In Cases 5 and 16 tracings were obtained at weekly intervals until the disease was thought to be cured on clinical and biochemical grounds.

Altogether 53 electrocardiograms were found to be of a quality adequate for critical scrutiny.

Results

Of the 24 patients in this series, 14 showed a distinct abnormality in the electrocardiogram at some time

during the course of the disorder. The changes, confined to the ST-T complex, were best seen in the standard leads I and II. The ST segment was elevated and sometimes merged with the upward deflection of an unusually broad and prominent T wave. The summit of this was flattened and formed a plateau, which in a few instances was slightly notched (Fig. 1). As a rule, the higher the serum calcium the more pronounced were these changes (Fig. 2), but there were numerous important exceptions to this observation. Not

FIG. 1.—Electrocardiograms in idiopathic hypercalcaemia showing changes in ST-T contour. A: Case 2 (serum calcium 18.6 mg./100 ml.). B: Case 16 (serum calcium 16.2 mg./100 ml.). C: Case 17 (serum calcium 10.4 mg./100 ml.). D: Case 18 (serum calcium 13 mg./100 ml.). E: Case 20 (serum calcium 13.4 mg./100 ml.).

FIG. 2.—Idiopathic hypercalcaemia (Case 5). Standard limb leads (I and II) showing presence and regression of ST-T changes. A: ST elevation and plateau T wave. QTc 0.39 (serum calcium 18 mg./100 ml.). B: One week later. ST elevation persists. QTc 0.405 (serum calcium 11.3 mg./100 ml.). C: Three weeks later. Normal ST-T complex. QTc 0.37 (serum calcium 10.9 mg./100 ml.).

infrequently the change in contour persisted even after the serum calcium had fallen to a normal level (Fig. 1, C). Arguing against a direct relationship between the serum calcium and ST-T changes, it is noteworthy that in eight patients the ST-T complex was abnormal when the serum calcium was within the normal range, in four it was normal when the serum calcium was highly elevated, and in three it returned to normal while the serum calcium was still elevated (Fig. 3).

FIG. 3.—Distribution of 53 electrocardiographic observations in idiopathic hypercalcaemia, related to the serum calcium. The black areas represent electrocardiograms with ST-T changes.

According to Katz (1946) and other authors the electrocardiographic change characteristic of hypercalcaemia is a shortening of the electrical systole (QT). When the length of the electrical systole is corrected for the heart rate (QTc), the normal range for infancy and childhood can be taken as 0.38 ± 0.04 (Lyon and Kaplan, 1954). In this series the raised serum calcium

was not accompanied by shortening of the QTc (Fig. 4). Indeed, in 36 of the 53 observations the QTc was above the mean of the normal range, and in 20 of these there were changes in the contour of the ST-T complex.

There did not appear to be any correlation between the length of the cardiac cycle, the P-R interval, or the QRS complex and the level of the serum calcium. None of the records showed any irregularity of cardiac rhythm apart from sinus arrhythmia.

Discussion

The continued ability of the myocardium to contract depends on the presence of the calcium ion. While calcium lack prevents the muscle from responding normally to the pacemaker, calcium in excess produces vagal stimulation, irregular rhythms, and, finally, systolic arrest (Goodman and Gilman, 1955). Accepted changes in the electrocardiogram as a result of hypercalcaemia are a shortening of the electrical systole (QT), and also perhaps a depression of the T wave (Kellogg and Kerr, 1936). These abnormalities are said to disappear as the hypercalcaemia subsides. In the present series such changes in the electrocardiogram were not found. The electrical systole was not shortened, nor was there in any instance depression of the T wave. Although changes in the contour of the ST-T complex were on the whole more obvious in those children with the highest serum calcium values, the contour was sometimes normal when the serum calcium was highly elevated, and abnormal when it was within the normal range. It is obvious that the electrocardiographic changes found in this series (elevation of the ST segment and a prominent, broad, flattened T wave) are not those usually attributed to the effect of hypercalcaemia, nor indeed do they appear to be directly related to the level of the serum calcium. One is led, therefore, to search for some other factor associated with idiopathic hypercalcaemia which could be responsible for this electrocardiographic picture.

Ingestion of vitamin D either as cod-liver oil (D_3) or as one of the synthetic preparations (D_2) is known to produce certain abnormalities in the electrocardiogram apart from those which could be directly attributed to an associated hypercalcaemia. In a study of the electrocardiogram of rabbits fed on cod-liver oil, Agduhr and Stenström (1930) found a plateau T wave in some instances, and reported that changes in the T wave occurred simultaneously with lengthening of the Q-T interval. Elevation of the ST segment, prolongation of electrical systole (QT) and alterations in the summit of the T wave were often observed by these authors in other species of animals similarly treated. About the same time Jundell and Stenström (1931) studied electrocardiograms from infants treated with cod-liver oil and Vigantol (D_2). In addition to delayed auriculo-ventricular conduction and widened QRS complexes they found abnormal T waves. In contrast to this,

FIG. 4.—Correlation of serum calcium with QTc value of corresponding electrocardiograms.

Anning *et al.* (1948) found no constant changes in the electrocardiogram which could be attributed to the administration of calciferol (D_2), although they admitted that the P-R interval was sometimes lengthened and the electrical systole (QTc) shortened.

Electrocardiographic changes similar to those described in this series have been recorded by Jelke (1946) and by Skatvedt (1947) in patients suffering from vitamin-D poisoning. Jelke's patient, a girl of 17 months, had received 6.25 million I.U. of vitamin D_2 for the treatment of rickets. The T wave was prominent, broad, and flat-topped, the ST segment was raised, but the electrical systole (QT) was shortened. At the time these observations were made the serum calcium was 15.9 mg./100 ml. The ST segment remained raised even after the serum calcium had fallen to normal. Skatvedt's patient, a girl aged 1 year, had been given in error 6.5 million I.U. of vitamin D_2 over a period of nine months. On admission to hospital the serum calcium was 19.8 mg./100 ml. There was elevation of the ST segment and a marked platform (*sic*) T wave which was still abnormal three months later although the child was well and the serum calcium was normal in amount. The QTc (calculated from the published electrocardiogram) was within normal limits.

I have recently studied a case of vitamin-D poisoning. A girl aged 2½ years was given 5.4 million I.U. of vitamin D_2 over a period of five weeks immediately before admission to hospital. On admission the serum calcium was 19.6 mg./100 ml., and the electrocardiogram showed elevation of the ST segment in lead II and a broad plateau T wave in leads I and II (Fig. 5). These abnormalities became less pronounced as the hypercalcaemia subsided, but had not entirely disappeared when the serum calcium returned to normal. The QTc was not at any time below the normal range.

FIG. 5.—Vitamin D poisoning. E.C.G. standard leads. A: On admission. Plateau T wave (leads I and II). QTc 0.40 (serum calcium 19.6 mg./100 ml.). B: 3 days later. Plateau T wave (leads I and II). ST elevation (lead II). QTc 0.35 (serum calcium 15.6 mg./100 ml.). C: 6 days later. Slight regression of changes in ST-T contour. QTc 0.355 (serum calcium 12.5 mg./100 ml.).

It would appear that the changes found in the electrocardiogram in this series of infants with idiopathic hypercalcaemia are more likely to be related to the intake of vitamin D than directly to the hypercalcaemia. Whether the synthetic vitamin D_2 is more prone to produce these changes than the natural vitamin D_3 is uncertain. Nevertheless, these electrocardiographic findings lend further support to the idea that the administration of vitamin D is associated with the production of idiopathic hypercalcaemia. However, an explanation is still required for the observation that in these cases the electrical systole is not shortened in the presence of hypercalcaemia. This may in part be

due to the width of the T wave, or perhaps to the same noxious influence on the myocardium as that causing the changes in the contour of the ST-T complex.

At this point it is pertinent to consider whether the electrocardiographic changes in this series and in hypervitaminosis D are evidence of an organic myocardial lesion or not. Sagall *et al.* (1943) produced similar and reversible changes in the ST-T complex by the administration of quinidine, but Dressler *et al.* (1951) considered that a notched or plateau T wave was suggestive of myocardial damage and found that notching of T waves in the standard limb leads was often seen in patients with coronary arteriosclerosis.

Organic changes in the myocardium attributed to the consumption of cod-liver oil have been reported in two infants who died at the age of 16 days and 4 months (Malmberg, 1928). In each case there was atrophy of muscle cells and connective-tissue change. Ross and Williams (1939) found calcium deposition in the media of arteries and in the myocardium of an infant who had suffered from poisoning by irradiated ergosterol (vitamin D_2). A more extreme example of this kind (Bauer and Freyberg, 1946) concerned a 29-year-old woman who had consumed huge quantities of vitamin D in the year before her death. In the coronary arteries there was thickening of the intima and calcification of the media, while in the myocardium fatty change and areas of infarction were found. The electrocardiogram of this patient showed slight prolongation of electrical systole as well as lengthening of the P-R interval, changes thought to be due to myocardial damage. These authors concluded that the occurrence of pathological calcification seemed to depend more on fluctuations of serum calcium concentration than on the absolute serum level. It seems possible that the incidence of such changes might be more readily related to the diffusible calcium than to the total serum calcium, by analogy with the relative importance of these values in determining the onset of calciferol poisoning during treatment with this substance (Anning *et al.*, 1948).

Only one patient included in this series has died (Case 20). This child had osteogenesis imperfecta, and death occurred at the age of 14 months, after an apparent recovery from idiopathic hypercalcaemia five months previously. The last electrocardiogram which was obtained at that time was normal, although the previous record had shown a distinctly abnormal ST-T contour. At necropsy nephrocalcinosis and longitudinal sinus thrombosis were found, but a routine histological section of myocardium did not show any calcification or abnormality of coronary vessels. In those patients who initially had electrocardiographic changes and whose disease was followed to clinical recovery, the electrocardiogram returned to normal. It is nevertheless not certain that this implies a complete resolution of myocardial or coronary artery changes if such were present. The possibility therefore remains that patients who in infancy have suffered from idiopathic hypercalcaemia may carry residual coronary artery disease with them into later life.

Summary and Conclusions

Electrocardiographic studies have been made in 24 patients suffering from idiopathic hypercalcaemia, and abnormalities were found in 14. These consisted of elevation of the ST segment and a prominent, broad, and plateau-like T wave.

The electrocardiographic changes are not those usually associated with hypercalcaemia. The pattern does, however, closely resemble that found in some patients suffering from vitamin-D poisoning. Such electrocardiographic changes may possibly result from lesions of the myocardium or coronary arteries.

The possibility exists that residual coronary artery damage from idiopathic hypercalcaemia in early childhood may persist into later life.

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CONTROL OF SURGICAL INCONTINENCE BY MEANS OF THE PORTSMOUTH VALVED CATHETER

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Surgical treatment of primary or secondary cancer of the bladder and/or urethra has become common practice in contemporary gynaecology. This usually results in incontinence from either the bladder or its substitute. Total extirpation of the urethra is followed by such a distressing degree of incontinence that the patient gladly accepts further surgery. Uretero-colic anastomosis so often results in an increasing degree of kidney damage that other operations have been devised in order to offer greater kidney protection. Most favoured among these operations has of recent years been the ileal graft draining through an abdominal opening. Whether it be an ileostomy or a suprapubic or perineal cystostomy, present systems of urine collection and disposal are, in my opinion, unsatisfactory. The most difficult problem

is presented by the perineal cystostomy. Five years ago I had to perform a total cystectomy in two such cases in order to relieve the patients' misery. What a drastic remedy, to have to remove an otherwise healthy organ.

For these reasons a new method of control, by means of a valved catheter, was conceived. In all these cases there is a soft-tissue track between the bladder or substitute and the body surface. If an effective seal can be obtained, continence is achieved. Methods of constricting the track or distending it were considered and discarded. The only remaining remedy seemed to be by compression from either end. This would quickly

FIG. 1.—Portsmouth valved catheter. Therapeutic series: 3, 2, 1, and 0.5 cm.

result in shortening of the track and recurrence of incontinence. A system was therefore then devised whereby the track was compressed by degrees over several days until an effective and lasting seal was obtained.

Description and Fitting.—The valve consists simply of two inflatable bulbs applied to a 24 French catheter. They are of 30-ml. capacity and are filled independently. In the fitting series (Fig. 1), the bulbs are 3, 2, 1, and 0.5 cm. apart. It is usually necessary to fit the 3-cm. valve in the first instance, but if an effective seal is not at once obtained it should be replaced by a 2-cm. valve. Within 24 hours the patient complains of pain or dampness of the surrounding skin. As soon as this occurs the catheter should be replaced by one in which the bulbs are closer together. On this occasion an effective seal is usually obtained for three or four days. Though the track has now been shortened to about 2 cm., seepage of urine again occurs, and a 1-cm. catheter should be fitted. It has never yet been necessary to use the final 0.5-cm. valve in the series. During this time the patient quickly learns how simple it is to manage the prosthesis.

Management.—(1) *Suprapubic or Perineal Cystostomies* (Fig. 2): When first fitted the catheter should be released two-hourly. Within a few days bladder capacity is greatly increased, and soon these patients behave as normal, but they are all instructed