

Cardiac hypertrophy as a result of long-term thyroxine therapy and thyrotoxicosis

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Abstract

Objectives—To define the effects of long-term thyroxine treatment upon heart rate, blood pressure, left ventricular systolic function, and left ventricular size, as well as indices of autonomic function, and to compare findings with those in patients with thyrotoxicosis before and during treatment.

Design—Cross sectional study of patients prescribed thyroxine long term (n = 11), patients with thyrotoxicosis studied at presentation (n = 23), compared with controls (n = 25); longitudinal study of patients with thyrotoxicosis studied at presentation and serially after beginning antithyroid drug treatment (n = 23).

Methods—24 h ambulatory monitoring of pulse and blood pressure, echocardiography, forearm plethysmography, and autonomic function tests.

Results—Long-term thyroxine treatment in doses that reduced serum thyrotrophin to below normal had no effect on blood pressure, heart rate, left ventricular systolic function or stroke volume index, but was associated with an 18.4% increase in left ventricular mass index (mean (SEM) 101.9 (3.09) g/m² v controls 86.1 (4.61), P < 0.01). Thyroxine treatment, like thyrotoxicosis, had no effect on tests of autonomic function. Untreated thyrotoxicosis resulted in pronounced changes in systolic and diastolic blood pressure and an increase in heart rate during waking and sleep. Patients with thyrotoxicosis at presentation had an increase in left ventricular systolic function (ejection fraction 70.5 (1.66)% v 65.4 (1.79), P < 0.01; fractional shortening 40.4 (1.54)% v 35.6 (1.46), P < 0.01), increased stroke volume index (45.9 (2.4) ml/m² v 36.6 (1.7), P < 0.001), and an increase in forearm blood flow, and decrease in vascular resistance. They had a similar degree of left ventricular hypertrophy to that associated with thyroxine treatment (99.3 (4.03) g/m²); all changes were corrected within 2 months by antithyroid drugs.

Conclusions—The development of left ventricular hypertrophy in patients receiving thyroxine in the absence of significant changes in heart rate, blood pressure, and left ventricular systolic function is consistent with a direct trophic effect of thyroid hormone on the myocardium. The presence of left ventricular hypertrophy determines that further studies are

essential to assess cardiovascular risk in patients taking thyroxine long term.

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Keywords: thyroxine; thyrotoxicosis; left ventricular function; left ventricular hypertrophy

The clinical effects of thyroid disease on the cardiovascular system have been recognised for many years, one of the first descriptions of hyperthyroidism reporting the association of toxic goitre and palpitation.¹ It has been shown that untreated thyrotoxicosis results in increases in heart rate, systolic blood pressure, and ventricular systolic and diastolic function;²⁻⁴ changes which are largely corrected by antithyroid treatment.^{2,5} Furthermore, thyrotoxicosis has been reported to cause left ventricular hypertrophy in humans and the cat,^{6,7} while restoration of euthyroidism has been shown to reduce left ventricular mass.⁶ The similarity between the cardiovascular features of thyrotoxicosis and those of a hyperadrenergic state has led to debate regarding the relative contributions of direct effects of excess thyroid hormones on the heart and indirect effects mediated by a change in adrenergic sensitivity.⁸

While it is clear from the studies described above that overt hyperthyroidism is associated with marked changes in cardiovascular function, the cardiovascular effects of thyroxine replacement treatment are much less well defined. Thyroxine treatment, in doses which suppress serum thyrotrophin to below normal, has been reported to be associated with an increase in nocturnal heart rate⁹ and shortening of the systolic time interval of the pre-ejection period,¹⁰ while a recent study has demonstrated a change in left ventricular systolic function and dimensions in patients given thyroxine.¹¹ Thyroxine treatment is widely prescribed, surveys indicating that approximately 1% of the general population¹² and 4% of those aged 60 years and above are receiving long-term thyroxine treatment.¹³ Up to 21% of those in the general population prescribed thyroxine are taking a dose sufficient to suppress serum thyrotrophin to below the lower limits of normal, indicating a degree of hyperthyroidism,¹⁴ and further indicating that a large population is at risk of any cardiovascular complication of this thyroid hormone excess. These facts determine that it is important to define further the cardiovascular consequences of thyroxine treatment, especially in doses which suppress serum thyrotrophin to below normal.

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We have employed a series of non-invasive methods to define changes in heart rate, blood pressure, left ventricular function and size, as well as peripheral vascular resistance and indices of autonomic function in patients receiving long-term thyroxine treatment. Results were compared with those from patients with thyrotoxicosis investigated at presentation and serially after beginning antithyroid treatment, and with normal controls.

Patients and methods

PATIENTS

The thyroxine treated group comprised 11 women (mean (range) age 44.5 (35–65) years, mean body mass index 24.4 kg/m²) who were all receiving long-term thyroxine treatment (mean (range) dose 210 (100–300) µg/day) for a mean (range) of 9.6 (3–21) years because of a past history of thyroidectomy for differentiated thyroid cancer. All patients had received constant doses of thyroxine with the intention of maintaining serum thyrotrophin below the normal range. None had evidence of recurrent or metastatic disease at any time since total thyroidectomy. Findings from this group were compared with those from 23 women with overt thyrotoxicosis (mean (range) age 38.8 (22–65) years, mean body mass index 22.3 kg/m²) who were investigated at presentation and serially at 1 and 2 months after beginning antithyroid treatment with carbimazole. Findings were also compared with those from 25 normal healthy women (mean (range) age 42.9 (21–65) years, mean body mass index 23.7 kg/m²). Exclusion factors for patient and control groups included a previous or present history of cardiovascular disease, the presence of atrial fibrillation, symptoms or signs suggestive of cardiac failure and treatment with any cardiovascular drug. All patients and controls gave written consent to the studies which were approved by the South Birmingham Health Authority Research Ethics Committee.

ASSESSMENT OF THYROID STATUS

Thyroid function was determined in patients and controls by measurement of serum free thyroxine, free tri-iodothyronine (Amerlex M radioimmunoassays, Kodak Clinical Diagnostics, Amersham, UK) and thyrotrophin using a sensitive immunometric method (IDS Gamma-BCT, Boldon, UK). Normal ranges were free thyroxine 9–24 pmol/l, free tri-iodothyronine 2.0–9.0 pmol/l, and thyrotrophin 0.4–4.5 mU/l, determined as described previously.¹³ The limit of sensitivity of the thyrotrophin assay was 0.05 mU/l.

AMBULATORY MONITORING OF PULSE AND BLOOD PRESSURE AND ECHOCARDIOGRAPHIC STUDIES

An automatic cuff sphygmomanometer (TM 2420; A&D, Tokyo, Japan) was used to determine 24 h ambulatory heart rate and blood pressure.¹⁵ M mode and cross sectional echocardiography (Hewlett-Packard Sonos 500, 2.5 MHz transducer) were performed by the same operator for all participants to measure

cardiac dimensions, as described previously;¹⁶ measurements included left ventricular end systolic and end diastolic dimensions, left ventricular diastolic posterior wall thickness, interventricular diastolic septal thickness, and right ventricular diastolic diameter. Fractional shortening and ejection fractions were determined from echocardiographic data as indices of systolic cardiac function, and left ventricular mass index (left ventricular mass/body surface area) was calculated using the method of Devereux.¹⁷ Stroke volume index (stroke volume/body surface area) was calculated using the formula of Teicholz *et al.*^{18,19}

MEASUREMENT OF BLOOD FLOW AND VASCULAR RESISTANCE

Forearm blood flow was measured by plethysmography, using methods established in our own unit,²⁰ and forearm resistance calculated as a function of mean blood pressure and blood flow as described.²¹

TESTS OF AUTONOMIC FUNCTION

Responses of heart rate and blood pressure to 60° upright tilt with footplate for 40 min, isometric forearm exercise, a cold pressor test, and Valsalva's manoeuvre were determined²² to provide measures of autonomic activity. Beat to beat blood pressure was measured during autonomic testing using a Finapres finger cuff (Ohmeda 2300 Finapres, BOC Health Care, Denver, Colorado, USA) positioned at heart level.

STATISTICAL ANALYSIS

Results from patients receiving thyroxine and from those with thyrotoxicosis were compared with those from normal controls using the Mann-Whitney *U* test. Linear regression analysis was used to relate heart rate and blood pressure results to measures of thyroid function.

Results

TESTS OF THYROID FUNCTION

The serum free thyroxine concentration was significantly raised in women treated with thyroxine and to a greater extent in those with untreated thyrotoxicosis compared with that in controls (thyroxine treatment mean (SEM) 28.0 (2.6) pmol/l ($P < 0.005$), thyrotoxicosis 51.6 (2.2) ($P < 0.0001$), and controls 15.0 (0.4)), while serum free tri-iodothyronine was significantly raised only in those with thyrotoxicosis (thyroxine treatment 6.6 (0.5) pmol/l, thyrotoxicosis 24.6 (2.1) ($P < 0.005$), and controls 5.6 (0.2)). Treatment of thyrotoxicosis with antithyroid drugs resulted in a decrease in serum free thyroxine and free tri-iodothyronine concentrations by 1 month after starting treatment and all had free thyroid hormone concentrations within the normal range by 2 months after antithyroid treatment (free thyroxine at 1 month 25.6 (5.9) pmol/l ($P < 0.05$ *v* controls) and at 2 months 17.2 (3.1); free tri-iodothyronine at 1 month 15.1 (2.6) pmol/l ($P < 0.05$ *v* controls) and at 2 months 6.7 (1.0)). Serum thyrotrophin values were below

Table 1 Systolic and diastolic blood pressure (BP), and heart rate in patients receiving long-term thyroxine treatment, patients with thyrotoxicosis before and after antithyroid drug treatment for 1 or 2 months, and controls

	Thyroxine treatment (n = 11)	Thyrotoxicosis (n = 23)			Controls (n = 25)
		At presentation	At 1 month	At 2 months	
24 h systolic BP (mm Hg)	116 (3)	128 (3)†	121 (3)	115 (3)	113 (2)
24 h diastolic BP (mm Hg)	79 (2)	68 (2)*	69 (1)	72 (2)	73 (2)
Mean 24 h BP (mm Hg)	91 (2)	88 (2)*	86 (2)	86 (2)	86 (2)
24 h heart rate (beats/min)	74 (2)	94 (3)†	81 (3)	74 (3)	76 (2)
Waking systolic BP (mm Hg)	121 (4)	135 (3)†	126 (3)*	120 (3)	116 (3)
Waking diastolic BP (mm Hg)	82 (2)	71 (2)	72 (1)	74 (2)	75 (2)
Mean waking BP (mm Hg)	95 (3)	92 (2)	90 (2)	89 (2)	89 (2)
Waking heart rate (beats/min)	77 (2)	98 (3)†	83 (3)	77 (3)	79 (2)
Sleeping systolic BP (mm Hg)	98 (3)	107 (3)*	103 (4)	99 (3)	98 (2)
Sleeping diastolic BP (mm Hg)	69 (2)	56 (2)*	60 (2)	63 (2)	63 (2)
Mean sleeping BP (mm Hg)	78 (2)	73 (2)	74 (2)	75 (2)	75 (2)
Sleeping heart rate (beats/min)	65 (2)	84 (2)†	74 (2)*	64 (2)	64 (2)

Values are means (SEM). *P < 0.01; †P < 0.001 v controls.

the limit of assay sensitivity (< 0.05 mU/l) in five patients receiving long-term thyroxine treatment, while the remainder in the group had detectable serum thyrotrophin concentrations below the normal range (0.05–0.5 mU/l). All patients with overt thyrotoxicosis had a serum thyrotrophin measurement below the limit of assay sensitivity at presentation, while all controls had a normal serum thyrotrophin value (mean 2.38 mU/l).

24 H AMBULATORY MONITORING OF PULSE AND BLOOD PRESSURE

There were no significant differences in mean 24 h systolic and diastolic blood pressures in patients receiving long-term thyroxine treatment compared with those in controls. In contrast, mean systolic blood pressure was significantly increased in patients with untreated thyrotoxicosis compared with that in controls, while mean diastolic blood pressure was reduced (table 1). These differences in systolic and diastolic blood pressure in patients with thyrotoxicosis were abolished by 2 months after starting antithyroid drug treatment (table 1). Linear regression analysis of blood pressure results in patients with thyrotoxicosis at presentation showed a significant relation between 24 h systolic blood pressure and mean heart rate with circulating concentrations of free thyroxine (P < 0.05); a similar relation was not observed in the thyroxine treated group. Mean heart rate was not different from controls in those receiving thyroxine, in contrast to a significant increase in patients with thyrotoxicosis which was no longer evident 1 month after antithyroid treatment (table 1).

Analysis of waking and sleeping blood pressure and heart rate measurements showed that similar findings for systolic and diastolic blood pressure and heart rate were evident when waking and sleeping values were analysed separately (table 1). Despite differences in systolic and diastolic blood pressure and heart rate in patients with thyrotoxicosis at presentation, there was no change in diurnal variation in blood pressure or heart rate in either patients with thyrotoxicosis or those treated with thyroxine compared with that in controls (data not shown).

ECHOCARDIOGRAPHIC DETERMINATION OF LEFT VENTRICULAR SYSTOLIC FUNCTION AND CARDIAC DIMENSIONS

Analysis of results of echocardiography (table 2) showed no significant difference in left ventricular systolic and diastolic dimensions in the group receiving long-term thyroxine treatment. There was, however, a significant increase in the interventricular septal thickness and calculated left ventricular mass index in patients treated with thyroxine. The left ventricular mass index was similarly increased in patients with thyrotoxicosis at presentation compared with that in controls; a difference no longer evident by 1 month after antithyroid treatment. There was no significant difference in the mean fractional shortening or systolic ejection fraction in patients treated with thyroxine, although these measures of systolic function were increased in patients with thyrotoxicosis before antithyroid treatment (table 2). These abnormalities in thyrotoxicosis were again corrected within 2 months by antithyroid drug treatment.

Table 2 Echocardiographic data in patients receiving long-term thyroxine treatment, patients with thyrotoxicosis before and after antithyroid drug treatment for 1 or 2 months, and controls

	Thyroxine treatment (n = 11)	Thyrotoxicosis (n = 23)			Controls (n = 25)
		At presentation	At 1 month	At 2 months	
LV end systolic dimension (cm)	2.88 (0.18)	2.86 (0.09)	3.10 (0.08)*	3.09 (0.09)	2.86 (0.08)
LV end diastolic dimension (cm)	4.55 (0.06)	4.77 (0.02)*	4.79 (0.10)*	4.76 (0.10)	4.50 (0.03)
LV posterior wall diastolic dimension	0.94 (0.11)	0.86 (0.09)	0.82 (0.03)	0.79 (0.02)	0.88 (0.08)
RV diastolic dimension (cm)	2.11 (0.23)	1.93 (0.16)	1.71 (0.28)	2.08 (0.18)	1.89 (0.19)
IVS diastolic dimension (cm)	1.03 (0.06)*	0.88 (0.03)	0.75 (0.04)	0.74 (0.03)*	0.84 (0.03)
Fractional shortening (%)	36.3 (1.8)	40.4 (1.5)*	35.9 (1.3)	36.5 (1.2)	35.6 (1.5)
Ejection fraction (%)	65.8 (2.2)	70.5 (1.7)*	63.4 (2.0)	65.7 (1.5)	65.4 (1.8)
LV mass index (g/m ²)	101.9 (3.1)*	99.3 (4.0)*	85.4 (3.5)	83.1 (4.1)	86.1 (4.6)
SV index (ml/m ²)	37.1 (3.8)	45.9 (2.4)†	42.5 (2.5)*	40.3 (1.8)	36.6 (1.7)

Values are means (SEM). *P < 0.01; †P < 0.001 v controls. IVS, interventricular septum; LV, left ventricle; RV, right ventricle; SV, stroke volume.

INDICES OF CARDIAC OUTPUT

Calculation of the stroke volume index in patients treated with long-term thyroxine showed no significant difference compared with that in controls, in contrast to an increase in patients with thyrotoxicosis at presentation, which was corrected by antithyroid drug treatment (table 2). Linear regression analysis of the stroke volume index results in patients with thyrotoxicosis at presentation showed a significant relation with the left ventricular mass index ($P < 0.01$), but no significant relation was found in the thyroxine treated group.

Measurement of forearm blood flow showed no significant change in the thyroxine treated group, although there was a significant increase in flow in those with untreated thyrotoxicosis; a finding that was corrected by antithyroid drug treatment (mean (SEM) thyrotoxic group at presentation 3.89 (0.23) ml/min/100 g ($P < 0.05$), at 1 month 2.32 (0.35), and at 2 months 2.5 (0.26), and controls 1.87 (0.29)). Calculation of forearm resistance showed a reduction in vascular resistance in those with thyrotoxicosis compared with that in controls but again there was no significant difference in the thyroxine treated patients (mean (SEM) thyroxine treated group 34.8 (4.9) units of resistance, thyrotoxicosis at presentation 25.7 (3.2) ($P < 0.05$), and controls 47.9 (5.6)).

TESTS OF AUTONOMIC FUNCTION

Assessment of increases in systolic and diastolic blood pressure and heart rate in response to tilting, isometric forearm exercise, and a cold pressor stimulus failed to show any difference between patients receiving thyroxine or those with untreated thyrotoxicosis compared with those in normal controls. Responses to the Valsalva manoeuvre were similarly unaffected by thyroxine treatment or thyrotoxicosis (data not shown).

Discussion

In the present study we have shown that long-term thyroxine treatment has no significant effect on systolic or diastolic blood pressure or heart rate when determined by ambulatory monitoring over a 24 h period. Thyroxine treatment was, however, associated with a significant increase in left ventricular mass index of 18.4% compared with that in normal controls. Untreated thyrotoxicosis resulted in more pronounced cardiovascular changes than thyroxine treatment, including an increase in systolic blood pressure, a reduction in diastolic blood pressure, and an increase in heart rate, changes evident throughout a 24 h period of monitoring and during waking and sleep. Patients with thyrotoxicosis at presentation showed a similar degree of left ventricular hypertrophy to that observed in patients receiving long-term thyroxine treatment, as well as an increase in left ventricular systolic function indicated by measures of fractional shortening and ejection fraction, an increase in the stroke volume index, and an increase in forearm blood flow, and decrease in vascular resistance. Treatment of thyrotoxicosis with antithyroid

drugs was effective not only in reducing circulating free thyroid hormone values to the normal range but also in correcting abnormalities of blood pressure, heart rate, left ventricular mass, and left ventricular systolic function by 2 months after starting treatment.

The finding of increased left ventricular mass in patients treated with long-term thyroxine treatment is in accord with a recent study¹¹ which included 10 patients (eight female and two male) who had undergone thyroidectomy for thyroid cancer and who had subsequently received thyroxine in thyrotrophin suppressive doses for a mean of 5 years. We, like Biondi *et al.*,¹¹ showed an increase in interventricular septal thickness but in contrast to that study, we failed to show a significant increase in heart rate or left ventricular fractional shortening in our thyroxine treated group. We found no significant difference in either waking or sleeping measurements of blood pressure and heart rate compared with those in controls, in contrast to a report describing no change in blood pressure but an increase in nocturnal heart rate in normal controls treated short term with incremental doses of thyroxine until thyrotrophin suppression was achieved.⁹

Several previous studies have examined the effect of untreated thyrotoxicosis on cardiovascular function and have described similar changes in blood pressure and heart rate.²³ Our finding of an increase in left ventricular systolic function is in agreement with the results of a study using radionuclide ventriculography, which showed an increase in resting left ventricular ejection fraction in patients with hyperthyroidism and a decrease when euthyroidism was restored.²⁴ Our findings of an increased stroke volume index, an increase in forearm blood flow, and a decrease in vascular resistance are also consistent with a previous study of patients with thyrotoxicosis providing evidence for peripheral vasodilatation.²⁵ Reported studies have compared cardiovascular function in thyrotoxic subjects before treatment and after restoration of euthyroidism but serial assessment of patients at frequent intervals after beginning antithyroid treatment has not been described before. It is notable that antithyroid drug treatment abolished differences in diastolic blood pressure and left ventricular mass index by 1 month after treatment, at a time point before circulating free thyroid hormone concentrations had been restored to normal, and that all changes in the cardiovascular variables examined (except the IVS diastolic dimensions) were eliminated by only 2 months of antithyroid treatment. This would suggest that the cardiovascular risks of promptly treated thyrotoxicosis are minimal.

Our finding of significant left ventricular hypertrophy in patients treated with long-term thyroxine treatment in the absence of significant changes in heart rate, stroke volume, blood pressure, and left ventricular systolic function provides evidence for a direct effect of thyroid hormones on the heart. This direct effect is supported by evidence indicating the expression of functional receptors for thyroid hormone in the myocardium,²⁶ as well as direct

effects of thyroid hormones on myocardial genes, including that encoding the cardiac specific sarcoplasmic calcium adenosine triphosphatase, an enzyme which affects the velocity of diastolic relaxation.²⁷ Other evidence in support of direct actions of thyroid hormones on the myocardium comes from studies of patients with hyperthyroidism. Forfar *et al*²⁴ reported that β adrenergic blockade did not affect an exercise induced change in left ventricular ejection fraction, while a further study⁵ demonstrated that increases in left ventricular ejection fraction in thyrotoxicosis were not associated with changes in ventricular loading conditions, and further, these loading conditions were not altered by restoration of euthyroidism.

The similarity between the clinical manifestations of thyroid hormone excess and those of a hyperadrenergic state has led to extensive investigation of the relation between thyroid status and the autonomic nervous system. It is well recognised that thyrotoxicosis is not associated with an increase in circulating catecholamines²⁸ and the mechanism by which thyroid hormones might alter the responsiveness to catecholamines is unknown. It has been reported that hyperthyroidism is associated with an increase in the sensitivity of heart rate and left ventricular shortening velocity to stimulation by isoprenaline²⁹ and some, but not all, studies have suggested an increase in adrenergic receptor density in various tissues.³⁰⁻³¹ Nonetheless, the absence of change in responses in blood pressure or heart rate to tilting, a cold pressor stimulus, or the Valsalva manoeuvre, which all provide indirect measures of sympathetic activity, provides strong evidence against a change in adrenergic sensitivity in either patients receiving thyroxine long term or those with untreated thyrotoxicosis.

The most notable finding in the present study was significant left ventricular hypertrophy in patients receiving long-term thyroxine treatment and those with untreated thyrotoxicosis. Left ventricular hypertrophy was rapidly reversed by treatment of thyrotoxicosis but despite a considerably less noticeable increase in serum free thyroxine in the thyroxine treated group, an 18% increase in left ventricular mass index was present in these patients, presumably reflecting the duration of thyroxine treatment. The pathophysiological significance of this increase in left ventricular mass is unknown, although left ventricular hypertrophy is a well recognised risk factor for cardiac morbidity and mortality.³²⁻³³ A recent report described an increased incidence of atrial fibrillation with suppression of serum thyrotrophin in patients in the Framingham population (some of whom were treated with thyroxine);³⁴ however, no data were provided for left ventricular mass in this study. Further studies are essential to determine whether cardiac hypertrophy associated with long-term administration of thyroxine in doses that suppress serum thyrotrophin to below normal, as documented in the present study, is itself associated with an increase in cardiac events.

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SHORT CASES IN CARDIOLOGY

Migration of an implantable cardioverter-defibrillator generator into the small bowel

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In February 1992, a previously fit man of 64 experienced an out of hospital cardiac arrest without any prodromal symptoms. The paramedical team found him to be in ventricular fibrillation from which he was successfully defibrillated. There was no evidence of myocardial infarction and no neurological sequelae. He was transferred to our hospital where cardiac catheterisation showed a chronically occluded left anterior descending coronary artery as the only abnormality. An exercise test was normal and there were no inducible ventricular arrhythmias at electrophysiological study. We decided to implant a cardioverter-defibrillator.

Under general anaesthesia, a CPI Endotak 0062 electrode was positioned in the right ventricular apex and a subcutaneous patch (0063) was implanted in the left sub-axillary area. A 10 J, biphasic shock successfully terminated ventricular fibrillation. The leads were tunnelled down into a subcostal pouch. The left rectus muscle was dissected to make a pocket for the generator (CPI Ventak P2) which fitted satisfactorily behind the muscle but still within the rectus sheath. The generator pocket and wound were closed and the patient made a good recovery.

He continued to do well without any shocks and had only one episode of non-sustained ventricular tachycardia detected by the device. He was admitted in December 1994 for a replacement generator. He did not report any symptoms and physical examination showed the generator to be deep to the original incision.

At operation, the original scar was resected

and after careful dissection of the leads the generator was found to have migrated to the peritoneum. With the assistance of a general surgeon a midline incision was made and the defibrillator was found intraluminally within a loop of jejunum and eroding the bowel wall (figure). The posterior walls of the bowel loops adhered to one another as in a surgically created anastomosis. The box was removed, 25 cm of jejunum resected, and the ends of the bowel were anastomosed. Peritoneal lavage was performed and the wound was closed. Because the electrodes were deeply embedded in scar tissue they were not removed. He was treated with intravenous antibiotics and did well postoperatively; it was decided not to reimplant another device. Both leads and the subcutaneous patch had to be removed five months later because he had a discharging abdominal wound sinus. He made a good recovery.

Although migration of a cardioverter-defibrillator into the peritoneum has been described,¹ we believe that this is the first report of migration into the bowel. Although the patient initially denied any gastroenterological symptoms, after the operation he did report some occasional unexplained vomiting episodes in the months before the generator explantation. Had the device not been removed the small bowel would probably have become obstructed.

It is not clear how the device migrated from the subrectus sheath into the jejunum but presumably the device eroded through the posterior wall of the sheath, passed into the peritoneum, and was encompassed by loops of small bowel onto which it became stuck. The device then eroded through the walls of the jejunum. It is fortunate that the outcome was not more serious. It is important to remember that bowel or urinary tract symptoms in patients with an abdominally placed generator could be caused by device migration. Newer generators are smaller and can usually be implanted subpectorally² so such complications may become even rarer.

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- 2 Bardy GH, Johnson G, Poole JE, Dolack GL, Kudenchuk PJ, Kelso D, et al. A simplified, single-lead unipolar transvenous cardioversion-defibrillation system. *Circulation* 1993;88:543-7.

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Photograph of implantable cardioverter-defibrillator generator placed in the surgically resected specimen of jejunum, showing how the bowel conforms to the shape of the generator.