Manifestazioni non classiche

Roma, 9 novembre 2017

Massimo Procopio

SCDU Endocrinologia, Diabetologia e Metabolismo

A.O.U. Città della Salute e della Scienza di Torino

NONTRADITIONAL MANIFESTATIONS OF PRIMARY HYPERPARATHYROIDISM

Cardiovascular disease and risk factors

Psychological and cognitive symptoms and quality of life

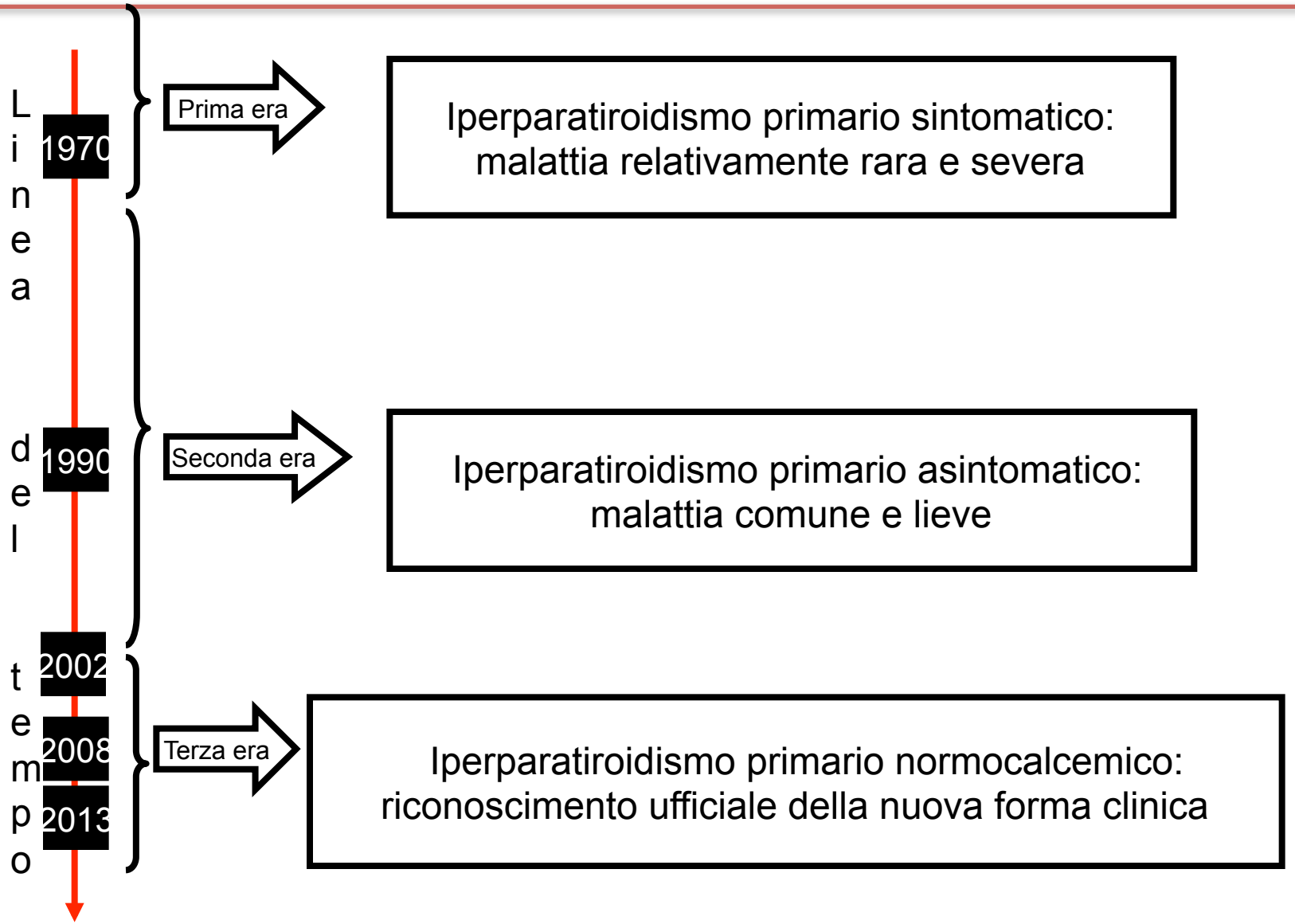
Rheumatic disease

Gastrointestinal disease

Cancer

MD Walker et al. Journal of Clinical Densitometry 16:40-47, 2013

Evoluzione della presentazione clinica del PHPT



Italian Society of Endocrinology Consensus Statement: definition, evaluation and management of patients with mild primary hyperparathyroidism

Definition of mild primary hyperparathyroidism

- Albumin-corrected serum calcium concentration lower than 1 mg/dL (0.25 mmol/L) above the upper limit of normal
- Bone mineral density by DXA: *T* score ≥ −2.5 at lumbar spine, femoral neck, total hip, or distal 1/3 radius
- No evidence of vertebral fractures (X-ray, VFA, MRI or CT)
- Estimated creatinine clearance (or eGFR) >60 mL/min
- 24-h urinary calcium excretion ≤400 mg/day and low renal stone risk by the urinary biochemical stone risk profile
- Absence of nephrolithiasis or nephrocalcinosis (by X-ray or ultrasound)
- Absence of relevant symptoms and complications directly attributable to either hypercalcemia or excess PTH secretion
- Age ≥ 50 years

DXA dual-energy X-ray absorptiometry, *VFA* vertebral fracture assessment (by DXA), *MRI* magnetic resonance imaging, *CT* computed tomography, *eGFR* estimated glomerular filtration rate

C. Marcocci et al. J Endocrinol Invest 38:577-593, 2015

Guidelines for the Management of Asymptomatic Primary Hyperparathyroidism: Summary Statement from the Fourth International Workshop

Table 1. Guidelines for Surgery in Asymptomatic PHPT: A Comparison of Current Recommendations With Previous Ones[a]

	1990	2002	2008	2013
Measurement[b]				
Serum calcium (>upper limit of normal)	1–1.6 mg/dL (0.25–0.4 mmol/L)	1.0 mg/dL (0.25 mmol/L)	1.0 mg/dL (0.25 mmol/L)	1.0 mg/dL (0.25 mmol/L)
Skeletal	BMD by DXA: Z-score <−2.0 (site unspecified)	BMD by DXA: T-score <−2.5 at any site[b]	BMD by DXA: T-score <−2.5 at any site[b]	A. BMD by DXA: T-score < −2.5 at lumbar spine, total hip, femoral neck, or distal 1/3 radius[b]
			Previous fragility fracture[c]	B. Vertebral fracture by x-ray, CT, MRI, or VFA
Renal	A. eGFR reduced by >30% from expected B. 24-h urine for calcium >400 mg/d (>10 mmol/d)	A. eGFR reduced by >30% from expected B. 24-h urine for calcium >400 mg/d (>10 mmol/d)	A. eGFR < 60 cc/min B. 24-h urine for calcium not recommended	A. Creatinine clearance < 60 cc/min B. 24-h urine for calcium >400 mg/d (>10 mmol/d) and increased stone risk by biochemical stone risk analysis[d] C. Presence of nephrolithiasis or nephrocalcinosis by x-ray, ultrasound, or CT
Age, y	<50	<50	<50	<50

JP Bilezikian et al. J Clin Endocrinol Metab 99:3561–3569, 2014.

NONTRADITIONAL MANIFESTATIONS OF PRIMARY HYPERPARATHYROIDISM

Cardiovascular disease and risk factors

Caso clinico B.L. ♀ 52 aa

Nel 11/2012 giunge alla nostra osservazione per riscontro occasionale di **ipercalcemia Ca tot 2.7 mmol/l**

Dati anamnestici salienti:
Familiarità positiva per CAD non precoce, ipercolesterolemia. Eumenorrea. 1 gravidanza a termine. Menopausa a 50 aa. Non fumatrice. Alimentazione parzialmente controllata. Sedentaria

DM2 noto da 5 anni in trattamento con metformina 500 mg x2 die, gliclazide 60 mg die con difficile controllo glicemico negli ultimi tempi: glicemie a digiuno attorno a 200 mg/dl HBA1c 82 mmol/mol (9.7%) (agosto 2012)

Ipertensione arteriosa nota da 3 anni in trt con ramipril 2.5 mg die con instabilità pressoria negli ultimi mesi
Stanchezza, difficoltà di concentrazione, stitichezza
EO: **PA 155/95** FC 96 B/min peso kg 77 h 160 cm **BMI 30.1 kg/m2** Non segni obiettivi tipici di endocrinopatie in atto.

Consigliata attività fisica regolare, dieta ipolipidica ipocalorica, aumentata Metformina 1000 mg x2, gliclazide 60 mg die, atenololo 25 mg/d

Caso clinico B.L. ♀52 aa

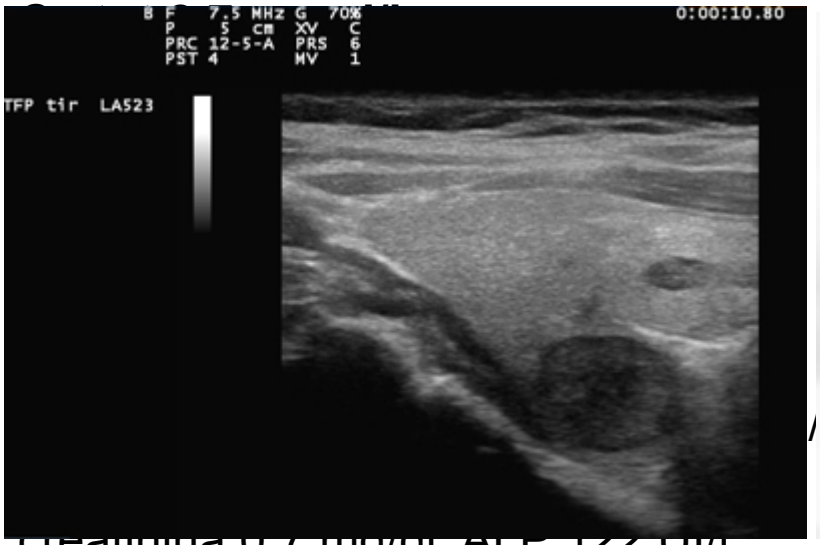
Nel 2/2013:
Ca tot 2.8 mmol/l
albumina 4.1 g/dl,
fosfatemia 0.85 mmol/L
Calciuria 24 ore 4.5 mmol/d
PTH 72 pg/ml
glicemia 210 mg/dl,
HBA 1c 85 mmol/mol (9.9%)
TSH reflex 2.0 mcUI/ml
colesterolo tot 265 mg/dl, HDL 35 mg/dl, trigliceridi 170 mg/dl, LDL colesterolo 196 mg/dl,
ac urico 5.1 mg/dl
creatinina 0.7 mg/dl, ALP 122 UI/l,
EO: **PA 150/90** peso kg 75 h 160 cm **BMI 29.2 kg/m2**

+ Torvast 10 mg/die, + Lantus10 UI BT

Caso clinico B.L. ♀52 aa

6/2013:

Metformina 1000 mg x2, gliclazide 60 mg die, atenololo 25 mg/d + Torvast 10 mg/die, + Lantus 10 UI BT



colesterolo 102

creatinina 0.7 mg/dl, ALP 122 UI/l,

DXA Tsc -1.5 L2-L4, Tsc -1.8 su Fn e Ftot, Tsc -2.2 su 1/3R

Morfometria vert: non deformità

ETG Addome: non litiasi

PA 150/95 kg 72 h 160 cm **BMI 28.1 kg/m2**

PARATIROIDECTOMIA SX 9/2013

Caso clinico B.L. ♀52 aa

4/2014

Attività fisica regolare, dieta ipolipidica ipocalorica, ridotta Metformina 500 mg x3, ramipril 5 mg die , Torvast 10 mg die **STOP Lantus STOP gliclazide STOP Atenololo**

Ca tot 2.3 mmol/l

Riflessione

- Nei pazienti con diabete di tipo 2 di difficile compenso ricordarsi di controllare la calcemia per escludere l'iperparatiroidismo primario.
- La correzione dell'iperparatiroidismo primario può determinare un miglioramento del compenso glicemico e pressorio.

A record linkage study of outcomes in patients with mild primary hyperparathyroidism: The Parathyroid Epidemiology and Audit Research Study (PEARS)

	No adjustment for propensity			Propensity score adjusted		
Outcome	HRs	95% CI	*P*-values	HRs	95% CI	*P*-values
Primary outcomes						
All cause mortality	2·12	1·87–2·39	<0·001	1·64	1·43–1·87	<0·001
Fatal CVD	2·23	1·83–2·72	<0·001	1·64	1·32–2·04	<0·001
Nonfatal CVD	3·57	3·11–4·10	<0·001	2·48	2·13–2·89	<0·001
Secondary outcome						
Cancer deaths	2·29	1·83–2·86	<0·001	1·32	1·03–1·68	0·03
Cerebrovascular	3·17	2·53–3·97	<0·001	2·51	1·95–3·22	<0·001
Hypertension	4·48	3·60–5·57	<0·001	2·60	2·04–3·31	<0·001
Renal failure	19·00	14·55–24·82	<0·001	13·83	10·41–18·37	<0·001
Renal stones	5·07	2·66–9·68	<0·001	5·15	2·69–9·83	<0·001
Psychiatric	6·30	3·76–10·57	<0·001	4·25	2·33–7·77	<0·001
All fractures	2·17	1·73–2·72	<0·001	1·75	1·36–2·26	<0·001
Osteoporotic fractures	2·11	1·63–2·73	<0·001	1·63	1·22–2·19	<0·001
Cancer	2·08	1·71–2·53	<0·001	1·75	1·41–2·18	<0·001
Diabetes	1·43	1·06–1·94	0·02	1·37	1·01–1·86	0·04

N Yu et al. Clinical Endocrinology 75, 169–176, 2011

PREVALENCE OF VARIOUS FORMS OF ALTERED GLUCOSE TOLERANCE IN PRIMARY HYPERPARATHYROIDISM (PHPT) II

Authors	Diagnosed Type2 DM (%)	Undiagnosed Type2 DM (%)	IGT/IFG (%)	Number and charactersitic of PHPT patients	Methodology
M. Procopio et al. (2002)	14.3	15.3	40	59 pts with mostly asymtomatic PHPT	OGTT
S. Ayturk et al. (2006)	13.1	4.9	47.5	61 pts with asymptomatic or normocalcemic PHPT	OGTT
A.A. Khaleeli et al. (2007)	5.5	16.6	19	54 pts with PHPT	OGTT
M.G.Cardenas et al. (2008)	15.9	-	-	609 patients operated for PHPT	Clinical history and ADA criteria

DIABETES/METABOLISM RESEARCH AND REVIEWS
Diabetes Metab Res Rev 2007; 23: 43–48.
Published online 16 May 2006 in Wiley InterScience (www.interscience.wiley.com) DOI: 10.1002/dmrr.637

RESEARCH ARTICLE

Prevalence of glucose intolerance in primary hyperparathyroidism and the benefit of parathyroidectomy

A. A. Khaleeli*
J. N. Johnson
W. H. Taylor

Departments of Medicine and Surgery, Halton General Hospital,

Abstract

Background Increased prevalence of diabetes mellitus (DM) in primary hyperparathyroidism (PHPT) is established, but not glucose intolerance (GI), nor benefit from parathyroidectomy on GI. We determined these during management of a continuous series of patients with PHPT routinely followed

Table 2. Glucose status in primary hyperparathyroidism and the effect of parathyroidectomy

		Normal (NGT)	IGT/IFG	DM	GI
Pre-op *n* (%)		17(49)	6(17)	12(34)	18 (51)
Post-op first OGTT *n* (%)		23(66)	6(17)	6(17)	12 (34)
End follow-up *n* (%)		23(66)	4(11)	8(23)	12 (34)
		Mean FPG		Mean 2-h PG on OGTT	
		mmol/L	*p*	mmol/L	*p*
Normal OGTT	Pre-op $n = 16$	5.0 ± 0.5	ns	5.7 ± 1.2	ns
	Post-op $n = 16$	5.0 ± 0.5		5.1 ± 1.7	
Patients without DM	Pre-op $n = 22$	$5.2 =/- 0.7$	ns	6.2 ± 1.6	<0.05
	Post-op $n = 22$	5.1 ± 0.6		5.4 ± 1.6	
All patients[a]	Pre-op $n = 30$	5.7 ± 1.0	<0.05	7.2 ± 3.0	<0.01
	Post-op $n = 30$	5.4 ± 0.8		6.3 ± 3.1	

[a]Excludes one patient with persistent hypercalcaemia and four with established DM on medication.

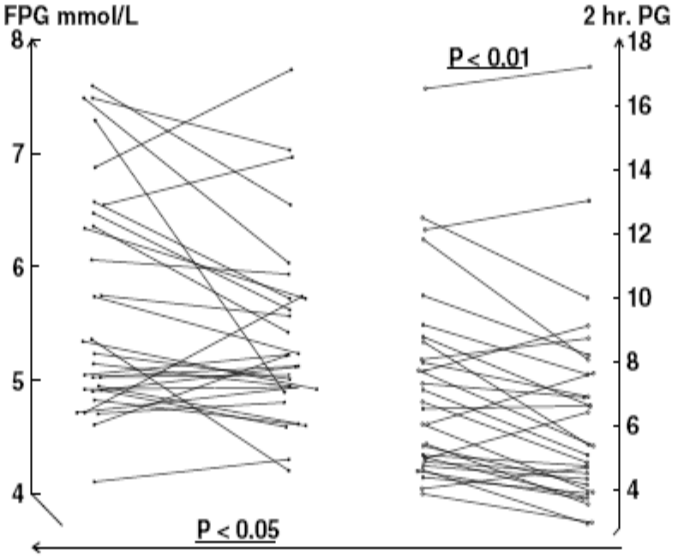


Figure 1. Effects of parathyroidectomy on fasting plasma glucose and 2-h plasma glucose on 75 g oral glucose tolerance test in primary hyperparathyroidism

AA Khaleeli et al., Diabetes Metab Res Rev 2007; 23: 43–48.

DIABETICMedicine

DOI: 10.1111/j.1464-5491.2009.02804.x

Original Article: Metabolism

Insulin resistance is not coupled with defective insulin secretion in primary hyperparathyroidism

F. Tassone, M. Procopio*, L. Gianotti, G. Viscontiţ, A. Pia‡, M. Terzolo‡ and G. Borretta

Division of Endocrinology and Metabolism, Santa Croce e Carle Hospital, Cuneo, *Division of Endocrinology and Metabolism, University of Turin, Turin, ţLaboratory of Clinical Chemistry, Santa Croce e Carle Hospital, Cuneo and ‡Division of Internal Medicine, University of Turin, Azienda Sanitaria Ospedaliera San Luigi, Orbassano, Italy

Table 2 Estimates of insulin sensitivity and insulin secretion (mean ± SD) in pHPT patients and control subjects

	pHPT	Control subjects	*P**
QUICKI	0.15 ± 0.01	0.16 ± 0.01	< 0.03
Log ISI composite	0.63 ± 0.25	0.68 ± 0.23	< 0.05
Log EIR	1.58 ± 0.34	1.48 ± 0.28	< 0.04
Log AUC insulin	3.83 ± 0.25	3.75 ± 0.21	< 0.03

*Adjusted for age, BMI and systolic blood pressure.
AUC, area under the curve; BMI, body mass index; EIR, early insulin response; ISI, insulin sensitivity index; pHTP, primary hyperparathyroidism; QUICKI, quantitative insulin sensitivity check index; SD, standard deviation.

Table 5 Multivariate analysis in pHPT patients

	$R = 0.39$	$R^2 = 0.15$
Dependent variable QUICKI	β	*P*
Independent variables		
Log BMI	−0.27	0.004
Log PTH	0.06	0.59
Log serum calcium	−0.31	0.004
Log serum phosphate	−0.01	0.89
	$R = 0.40$	$R^2 = 0.16$
Dependent variable ISI composite	β	*P*
Independent variables		
Log BMI	−0.33	0.0002
Log PTH	0.09	0.38
Log serum calcium	−0.29	0.005
Log serum phosphate	−0.09	0.32

BMI, body mass index; ISI, insulin sensitivity index; pHTP, primary hyperparathyroidism; PTH, parathyroid hormone; QUICKI, quantitative insulin sensitivity check index.

F Tassone et al. Diabet. Med. 26: 968–973, 2009

NORMOCALCEMIC HYPERPARATHYROIDISM AND INSULIN RESISTANCE

Table 3
Glucose Metabolism Assessments

	NCPHP (n = 25)	Controls (n = 25)	P
Fasting glucose (mg/dL)	97.9 ± 10.7	92.3 ± 9.0	.05
2-hour glucose (mg/dL)	113.2 ± 28.8	106.6 ± 40.6	.51
Insulin resistance			
HOMA-IR	2.4 (1.7-3.1)	1.8 (1.2-3.0)	.17
Insulin sensitivity index			
$ISOGTT$	4.0 (2.5-6.5)	5.0 (4-9.5)	.22
AUC (insulin)	6,285 (4,443-10,863)	5,775 (2,977-7,657)	.33
AUC (glucose)	16,578 ± 3,867	14,542 ± 4,120	.10
HbA1c (%)	5.6 ± 0.6	5.4 ± 0.3	.15
Impaired fasting glucose, n	10	8	.55
Impaired glucose tolerance, n	3	6	.46
Insulin resistance (HOMA-IR >2.5), n	12	7	.14

S Temizkan et al. Endocr Pract. 21: 1-7, 2015

Dyslipidemia In Primary Hyperparathyroidism

Variable	Case	Control	*P*
Age (years)	66·7 ± 5·74	66·9 ± 5·66	NS
Ionized p-calcium (mmol/l)	1·28 ± 0·06	1·17 ± 0·03	***
Total s-calcium (mmol/l)	2·57 ± 0·12	2·36 ± 0·07	***
Calcium/creatinine (mmol/μmol × 10^4)§	4·17 ± 2·40	3·0 ± 1·6	***
Intact s-PTH (ng/l)	53·5 ± 21·5	29·5 ± 9·7	***
s-Creatinine (μmol/l)	83·5 ± 13·4	84·5 ± 13·1	NS
BMI (kg/m^2)†	26·9 ± 5·01	26·1 ± 4·3	NS
s-Total cholesterol (mmol/l)	6·83 ± 1·28	6·81 ± 1·27	NS
s-HDL-cholesterol (mmol/l)	1·40 ± 0·361	1·52 ± 0·37	*
s-LDL-cholesterol (mmol/l)	4·79 ± 1·13	4·85 ± 1·19	NS
s-VLDL-cholesterol (mmol/l)	0·53 ± 0·46	0·40 ± 0·28	*
s-Total triglycerides (mmol/l)	1·72 ± 0·87	1·36 ± 0·54	**
s-HDL-triglycerides (mmol/l)	0·16 ± 0·06	0·16 ± 0·07	NS
s-LDL-triglycerides (mmol/l)	0·52 ± 0·18	0·48 ± 0·15	NS
s-VLDL-triglycerides (mmol/l)	1·09 ± 0·81	0·74 ± 0·47	**
Atherogenic index‡	4·19 ± 1·64	3·73 ± 1·40	*

Results are given as mean ± SD. NS, not significant. †Calculated as body weight/(height)^2. ‡Calculated as (total cholesterol – HDL-cholesterol)/HDL-cholesterol. §24 hour urine collection. *$P < 0·05$, **$P < 0·01$, ***$P < 0·001$.

Variable	Inclusion	Five years	*P*†	*P*‡
Age (years)	66 ± 5·8	71 ± 5·7	ND	ND
Ionized p-calcium (mmol/l)	1·29 ± 0·06	1·15 ± 0·05	***	NS
Intact s-PTH (ng/l)	56·4 ± 22·7	35·2 ± 15·4	***	NS
s-Creatinine (μmol/l)	82·7 ± 14·1	84·7 ± 17·4	NS	NS
BMI (kg/m^2)§	27·1 ± 4·57	27·6 ± 4·35	**	NS
s-Total cholesterol (mmol/l)	6·67 ± 1·21	6·43 ± 1·32	NS	NS
s-HDL-cholesterol (mmol/l)	1·37 ± 0·33	1·52 ± 0·37	**	NS
s-LDL-cholesterol (mmol/l)	4·63 ± 1·07	4·23 ± 0·92	**	NS
s-Total triglycerides (mmol/l)	1·67 ± 0·94	1·84 ± 1·39	NS	NS
Atherogenic index¶	4·20 ± 1·84	3·57 ± 1·40	***	NS

Results are given as mean ± SD. NS, not significant; ND, not done. †Comparison between cases at 5 years and at inclusion. ‡Comparison between cases and controls at 5 years. §Calculated as body weight/(height)^2. ¶Calculated as (total cholesterol – HDL-cholesterol)/HDL-cholesterol. *$P < 0·05$, **$P < 0·01$, ***$P < 0·001$.

Hagström E, Clinical Endocrinology 56, 253–260, 2002

Arterial Hypertension, Metabolic Syndrome and Subclinical Cardiovascular Organ Damage in Patients with Asymptomatic Primary Hyperparathyroidism before and after Parathyroidectomy: Preliminary Results

Meccanismi potenziali in grado di produrre ipertensione in PHPT

- ↑ livelli di calcio sierico
- ↑ livelli di PTH
- ↑ livelli di PRA
- ↑ livelli di calcio intracellulare
- ipomagnesiemia

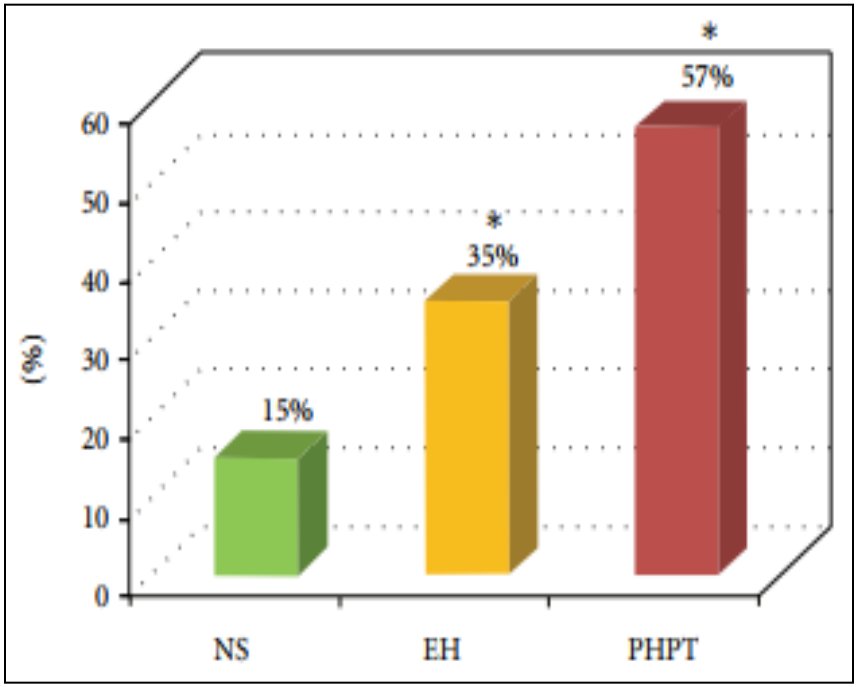


Figure 1: Prevalence of "non-dipping pattern" in all groups studied. NS: normal subjects; EH: patients with essential hypertension; PHPT: patients with primary hyperparathyroidism. ∗P = 0.02 versus NS.

P Luigi et al. International Journal of Endocrinology 2012, doi:10.1155/2012/408295

Cardiovascular risk and metabolic syndrome in primary hyperparathyroidism and their correlation to different clinical forms

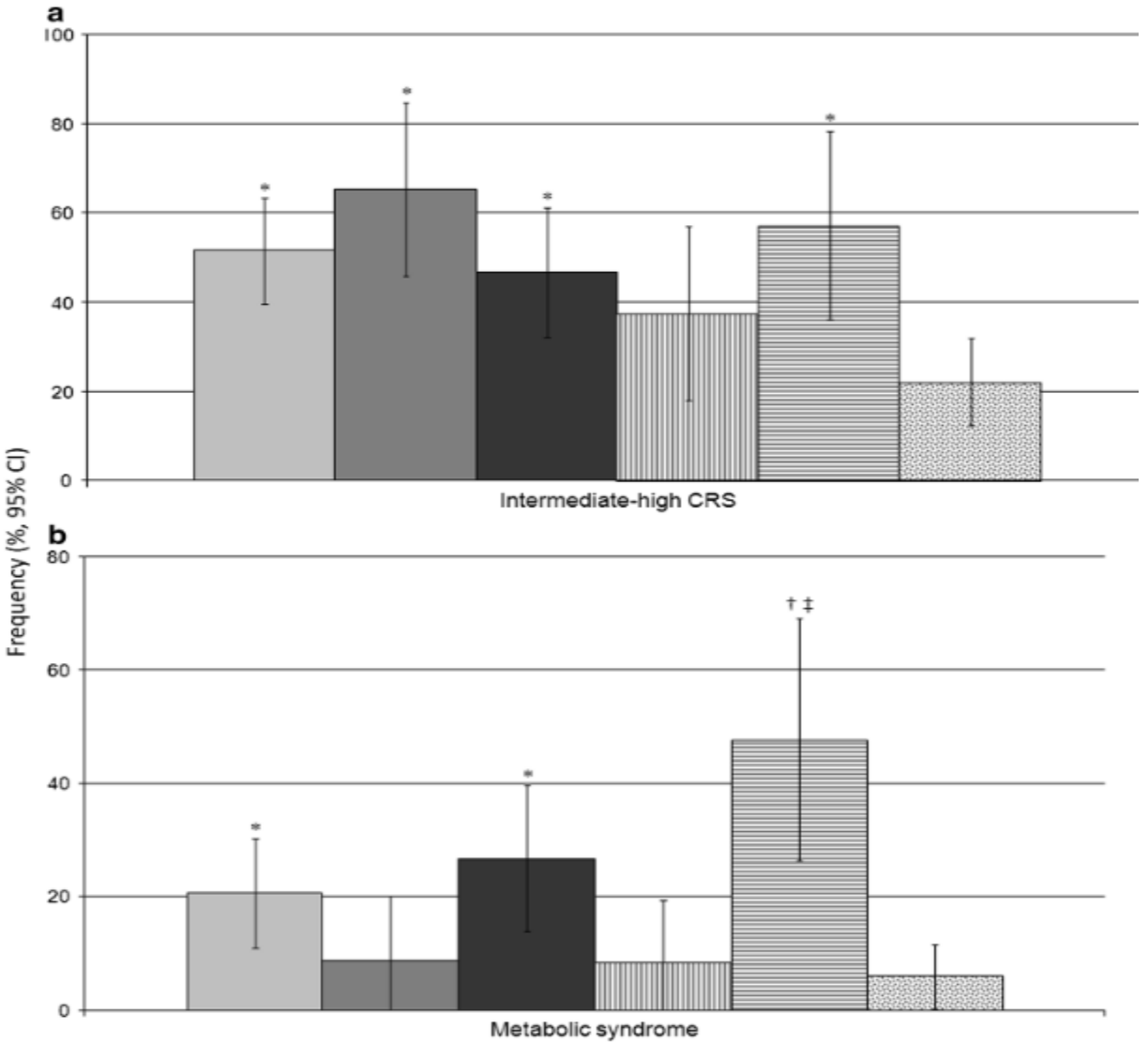


Fig. 3 Frequency of intermediate-high CRS (**a**) and metabolic syndrome (**b**) in PHPT patients, either as whole group (▒, $n = 68$), or according to different clinical forms (▒ symptomatic, $n = 23$; ■ asymptomatic, $n = 45$; ▥ high-risk asymptomatic, $n = 24$; ▤ low-risk asymptomatic PHPT, $n = 21$) and in controls (▨, $n = 68$). $*,^{\dagger}$ denotes the significance level with respect to controls ($p < 0.05$ and $p < 0.000005$, respectively); ‡ denotes the significance level with respect to symptomatic and high-risk asymptomatic PHPT ($p < 0.004$)

M Procopio et al. Endocrine 47:581–589, 2014

Arterial Hypertension, Metabolic Syndrome and Subclinical Cardiovascular Organ Damage in Patients with Asymptomatic Primary Hyperparathyroidism before and after Parathyroidectomy: Preliminary Results

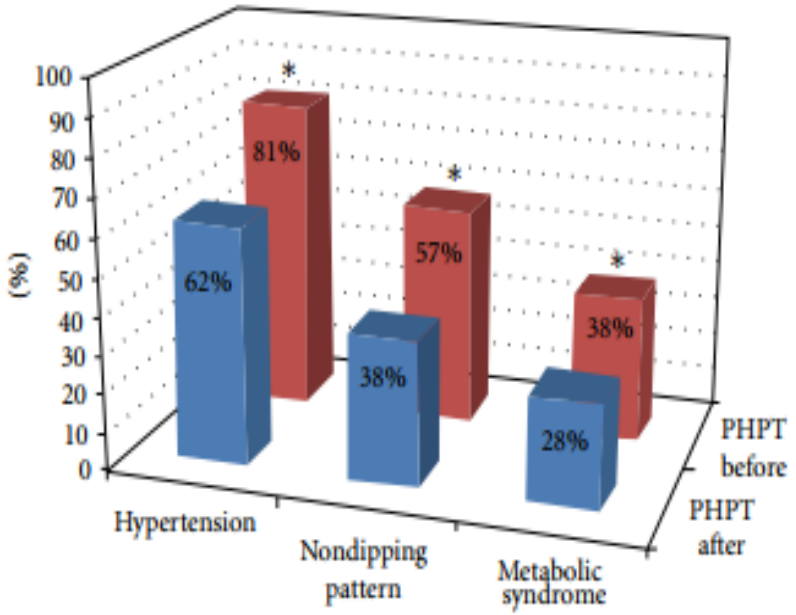
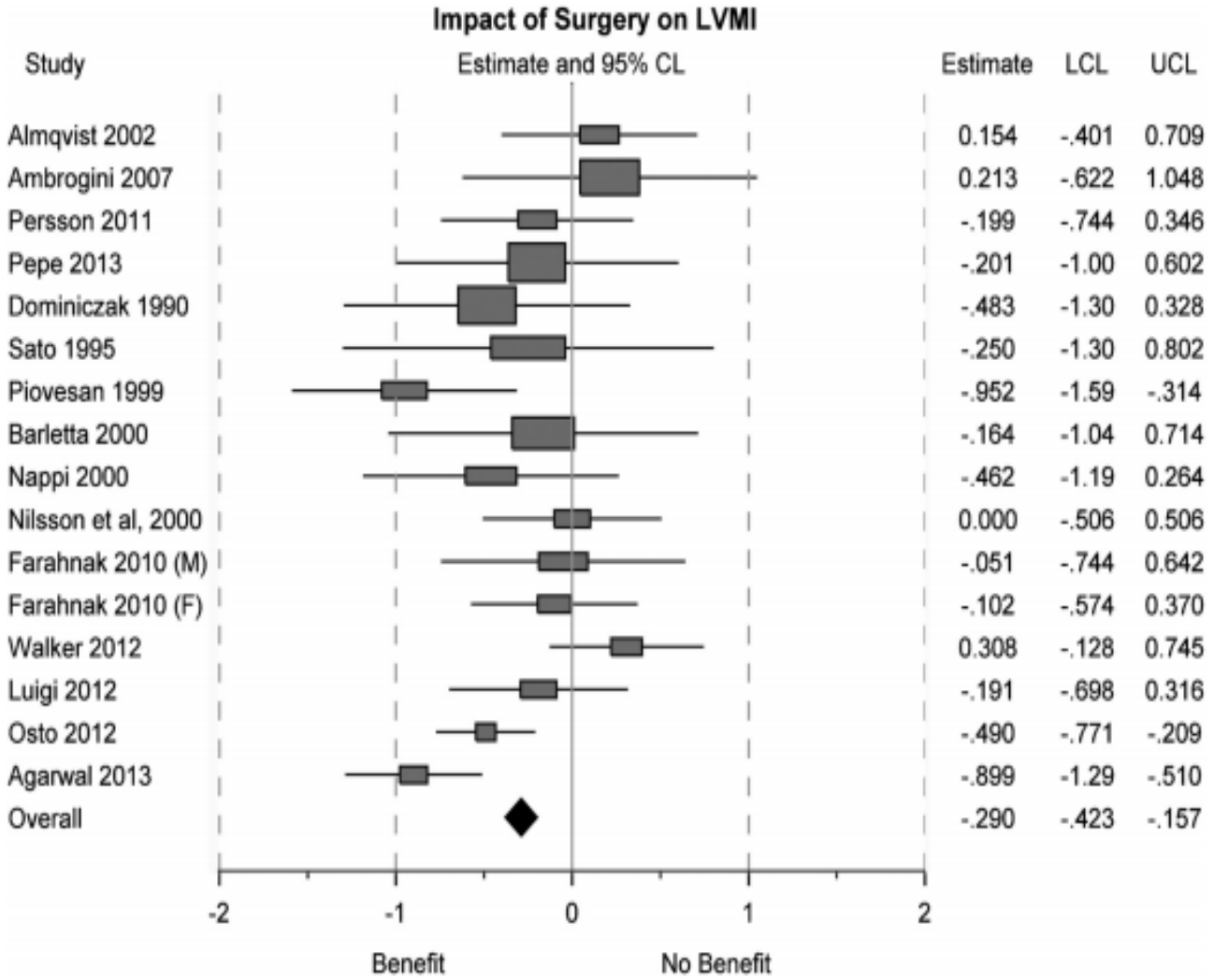


FIGURE 3: Prevalence of arterial hypertension, "non-dipping pattern," and metabolic syndrome in PHPT patients before (PHPT before) and after surgery (PHPT after). $^{*}P < 0.05$.

P Luigi et al. International Journal of Endocrinology 2012, doi:10.1155/2012/408295

Effect of Parathyroidectomy Upon Left Ventricular Mass in Primary Hyperparathyroidism: A Meta-Analysis

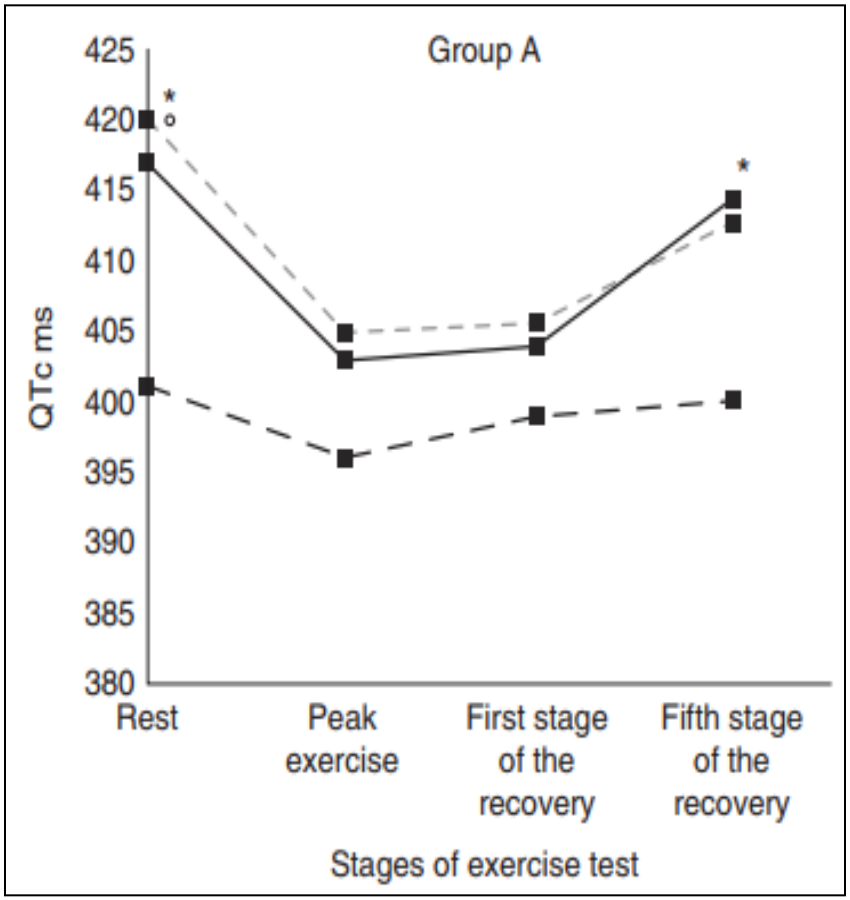


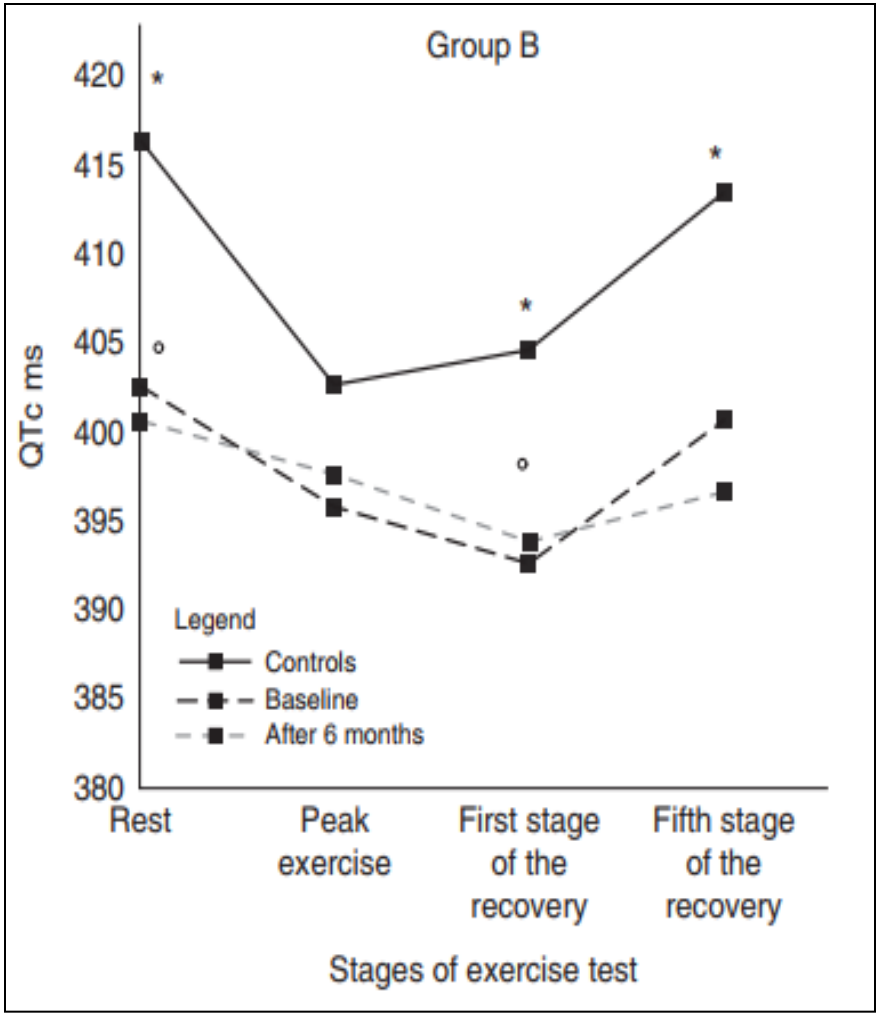
RCT

Figure 2. Forest plot indicating effect of PTX on LVM in patients with primary hyperparathyroidism. M, male; F, female.

DJ McMahon et al. J Clin Endocrinol Metab 100: 4399 – 4407, 2015

Parathyroidectomy eliminates arrhythmic risk in primary hyperparathyroidism, as evaluated by exercise test





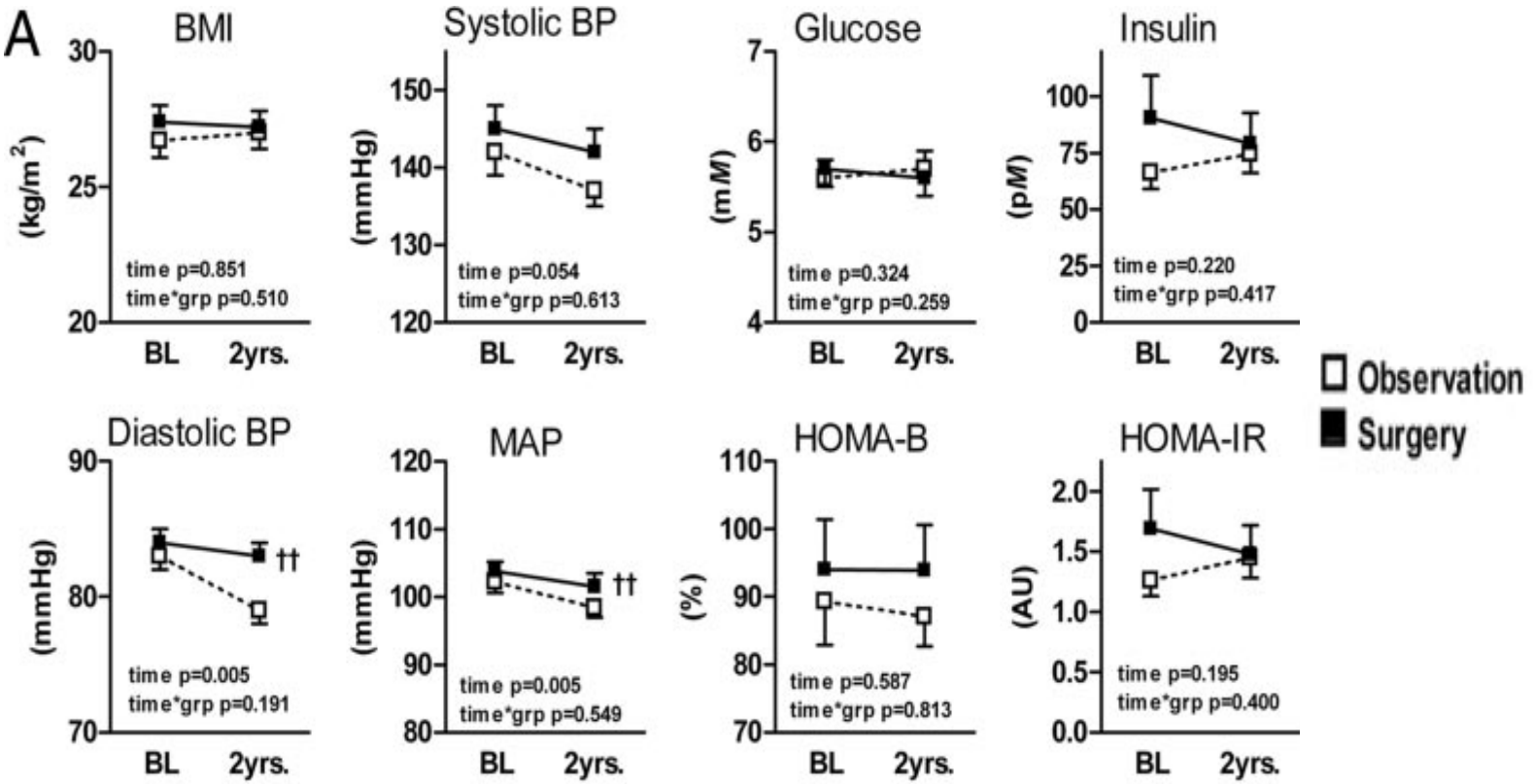
J Pepe et al. European Journal of Endocrinology 169:255–261, 2013

ORIGINAL ARTICLE

Endocrine Care

Effect of Surgery on Cardiovascular Risk Factors in Mild Primary Hyperparathyroidism

Jens Bollerslev, Thord Rosen, Charlotte L. Mollerup, Jörgen Nordenström, Marek Baranowski, Celina Franco, Ylva Pernow, Gunhild A. Isaksen, Kristin Godang, Thor Ueland, and Svante Jansson on behalf of the SIPH Study Group



J Clin Endocrinol Metab 94:2255–2261, 2009

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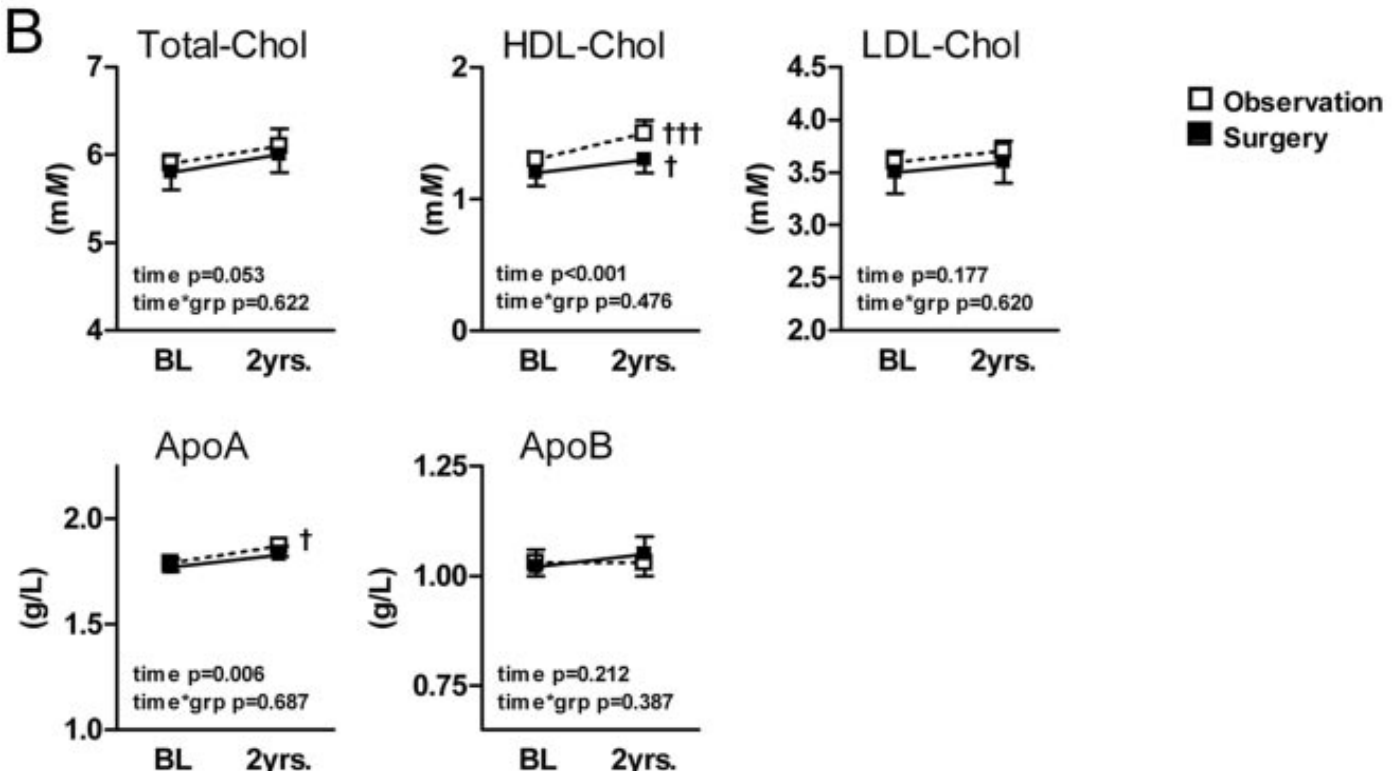


J Clin Endocrinol Metab 94:2255–2261, 2009

NONTRADITIONAL MANIFESTATIONS OF PRIMARY HYPERPARATHYROIDISM

Psychological and cognitive symptoms and quality of life

Neuropsychological Features in Primary Hyperparathyroidism: A Prospective Study

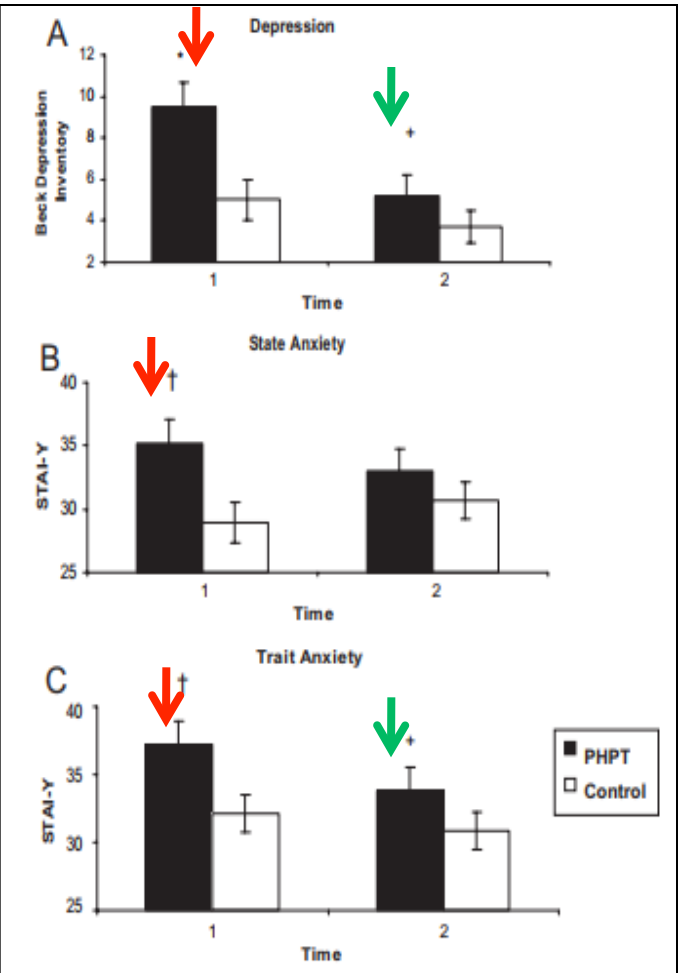


FIG. 1. A, Depression measured by BDI. B and C, State and trait anxiety as measured by STAI-Y. Higher scores indicate more symptoms. Scores are adjusted for age, IQ, and education. *, $P < 0.01$ compared with control group; †, $P < 0.05$ compared with control group; +, $P < 0.01$ compared with baseline. The test used to determine significance is the linear mixed model for repeated measures. *Error bars* represent 1 SEM.

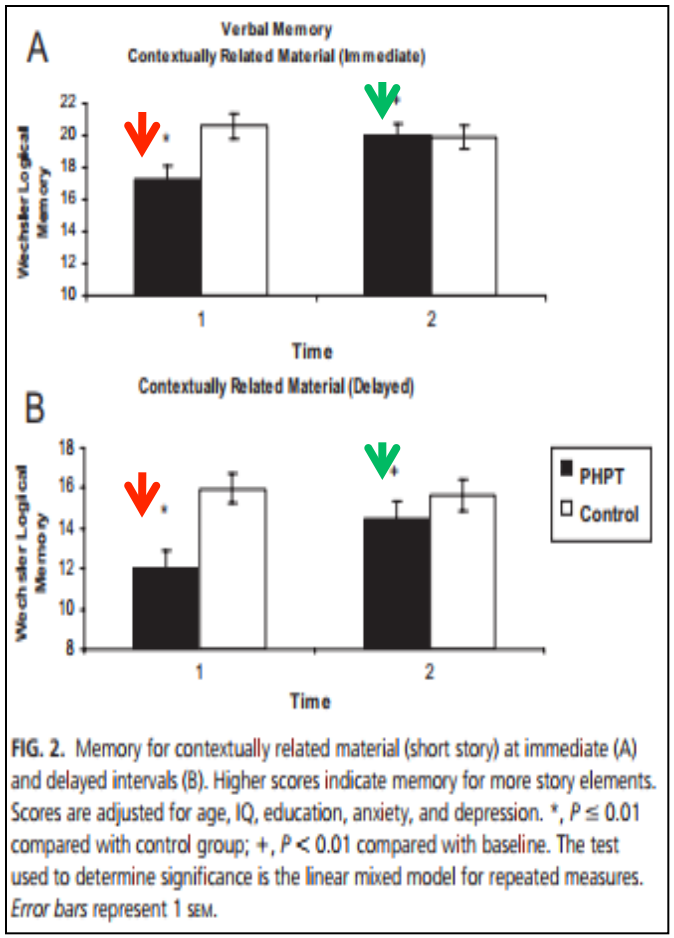


FIG. 2. Memory for contextually related material (short story) at immediate (A) and delayed intervals (B). Higher scores indicate memory for more story elements. Scores are adjusted for age, IQ, education, anxiety, and depression. *, $P \leq 0.01$ compared with control group; +, $P < 0.01$ compared with baseline. The test used to determine significance is the linear mixed model for repeated measures. *Error bars* represent 1 SEM.

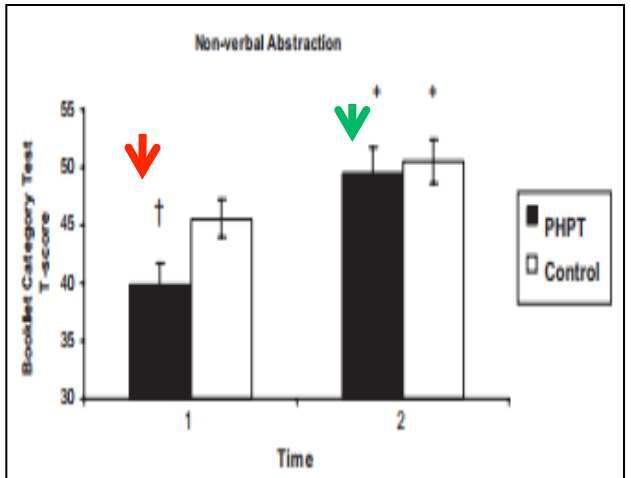


FIG. 4. Nonverbal abstraction assessed by BCT. Higher scores indicate better performance. Scores are adjusted for age, IQ, education, anxiety, and depression. †, $P < 0.05$ compared with control group; +, $P < 0.01$ compared with baseline. The test used to determine significance is the linear mixed model for repeated measures. *Error bars* represent 1 SEM.

PHPT
Control

1 **Baseline**
2 **6mo. after PTX**

MD Walker et al. J Clin Endocrinol Metab 94: 1951–1958, 2009

The effect of parathyroidectomy on neuropsychological symptoms and biochemical parameters in patients with asymptomatic primary hyperparathyroidism

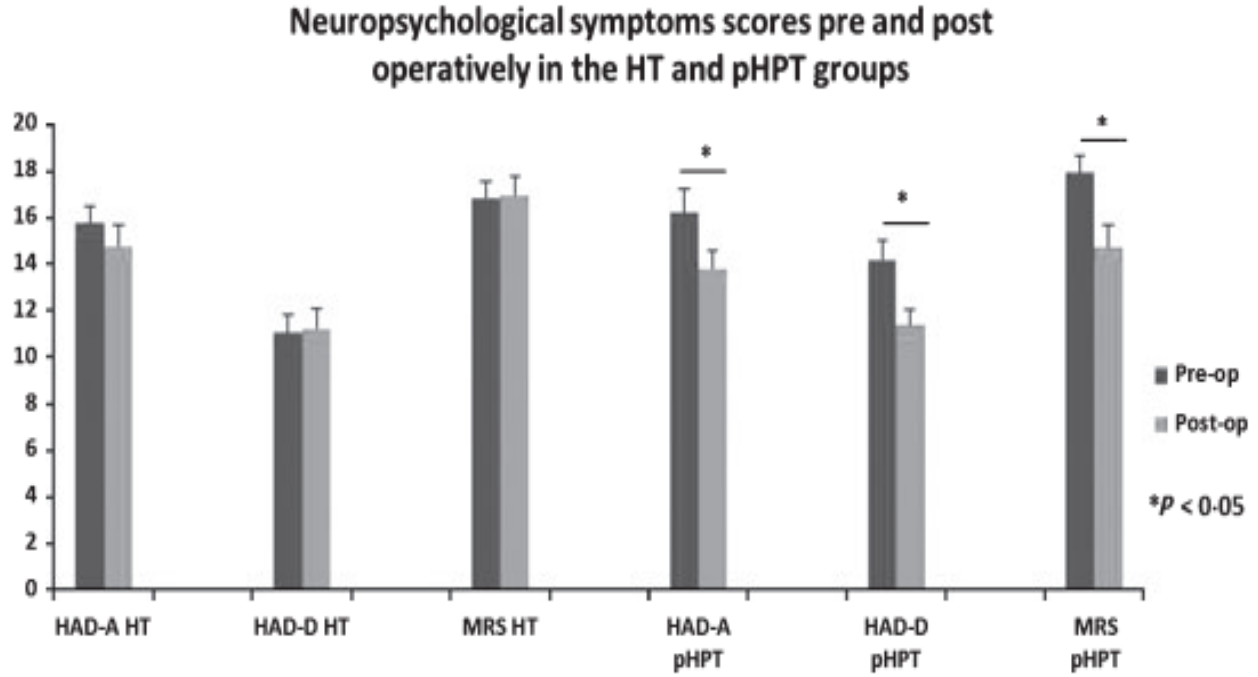
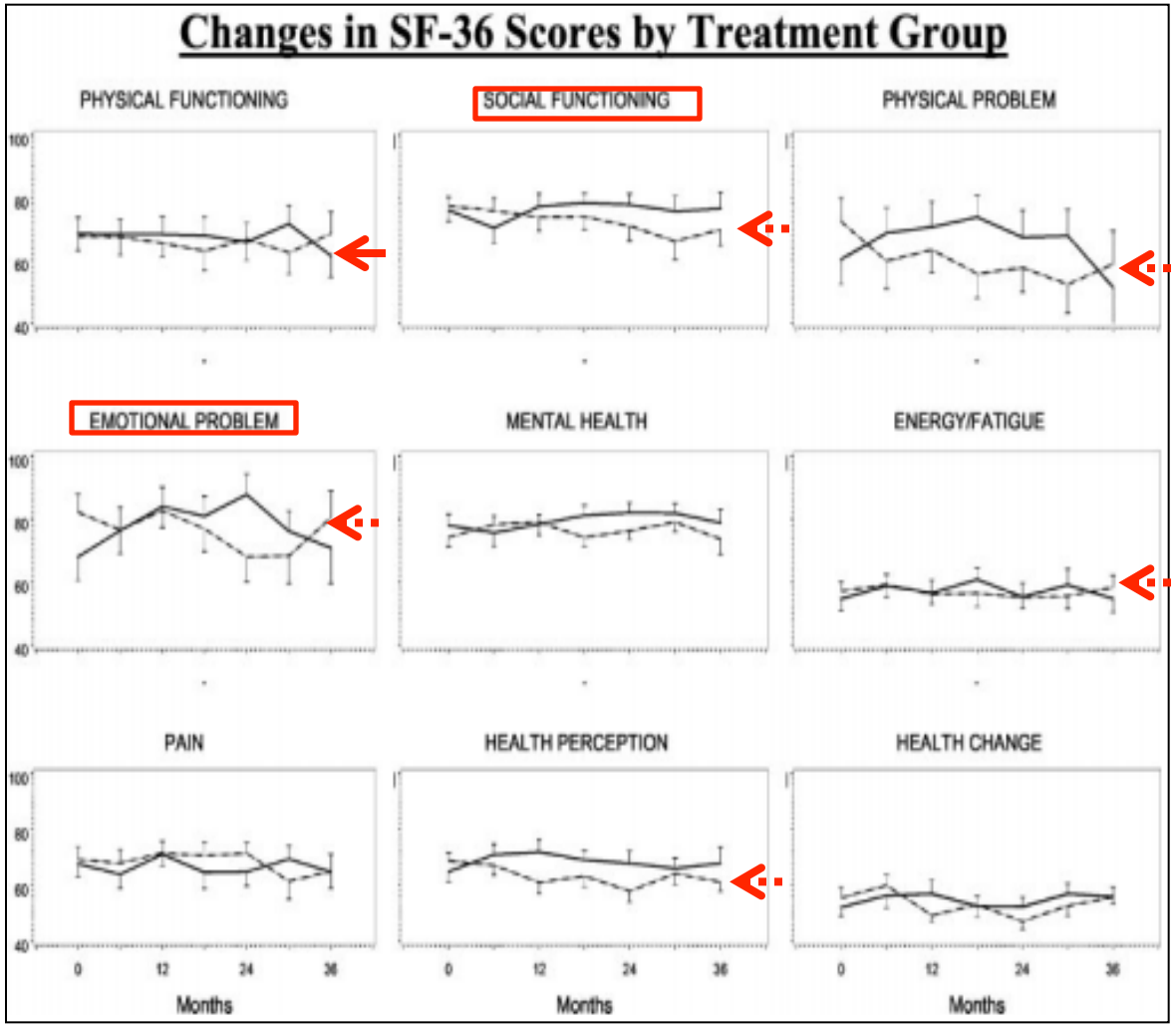
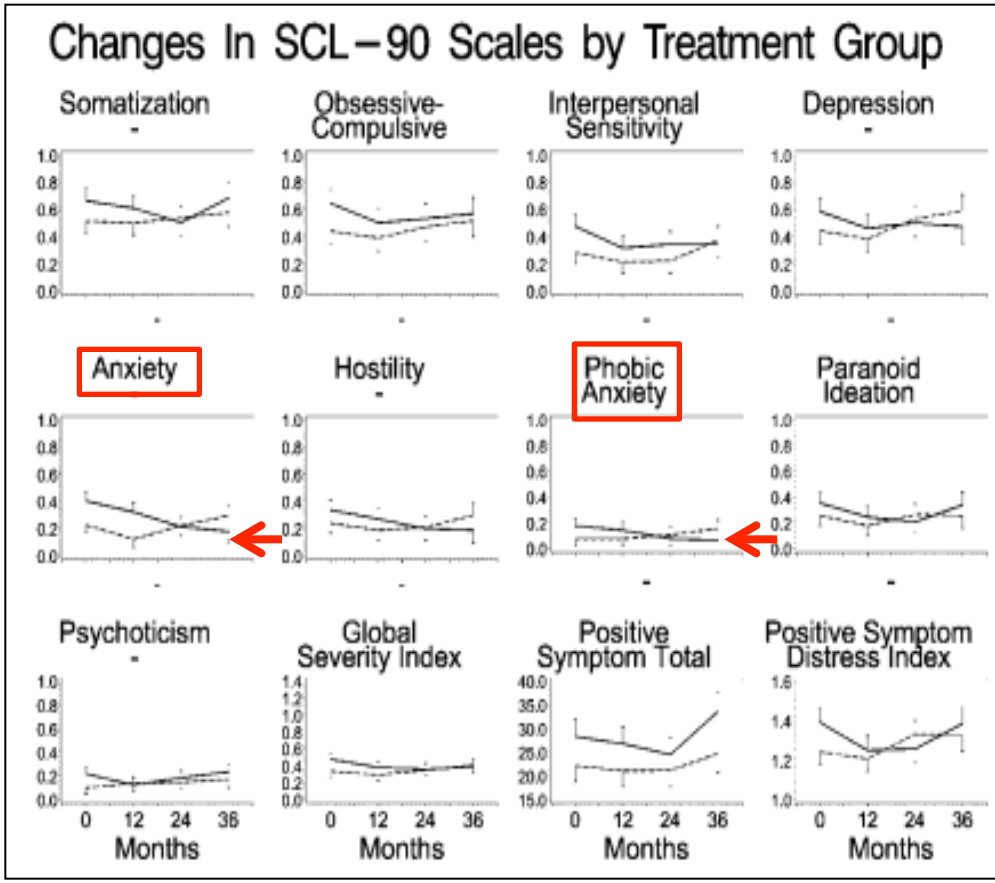


Fig. 1 Neuropsychological symptoms scores pre and post operatively in the hemithyroidectomy and primary hyperparathyroidism groups. Data presented as mean and standard error of the mean.

H Kahal et al. Clinical Endocrinology 76:196–200, 2012

Randomized Controlled Clinical Trial of Surgery Versus No Surgery in Patients with Mild Asymptomatic Primary Hyperparathyroidism



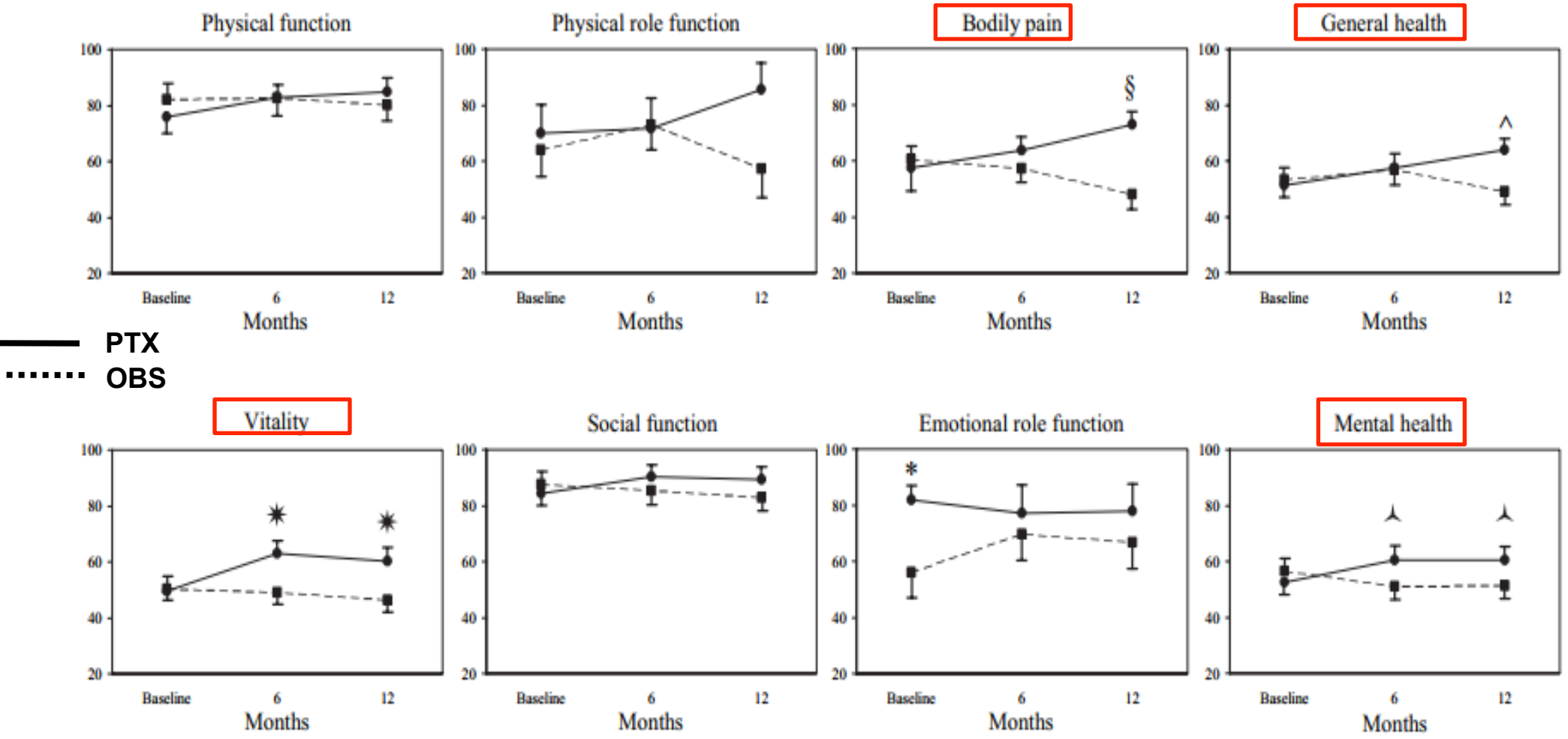


PTX

OBS

DS Rao et al. J Clin Endocrinol Metab 89: 5415–5422, 2004

Surgery or Surveillance for Mild Asymptomatic Primary Hyperparathyroidism: A Prospective, Randomized Clinical Trial



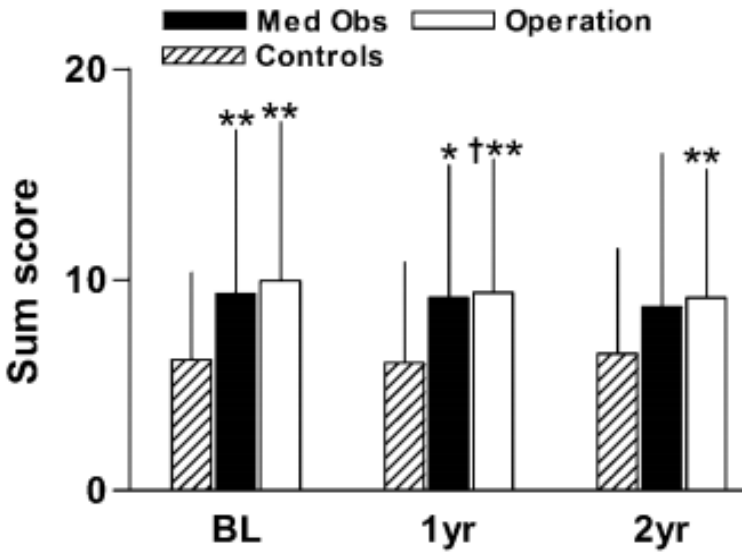
E Ambrogini et al. J Clin Endocrinol Metab 92: 3114 –3121, 2007

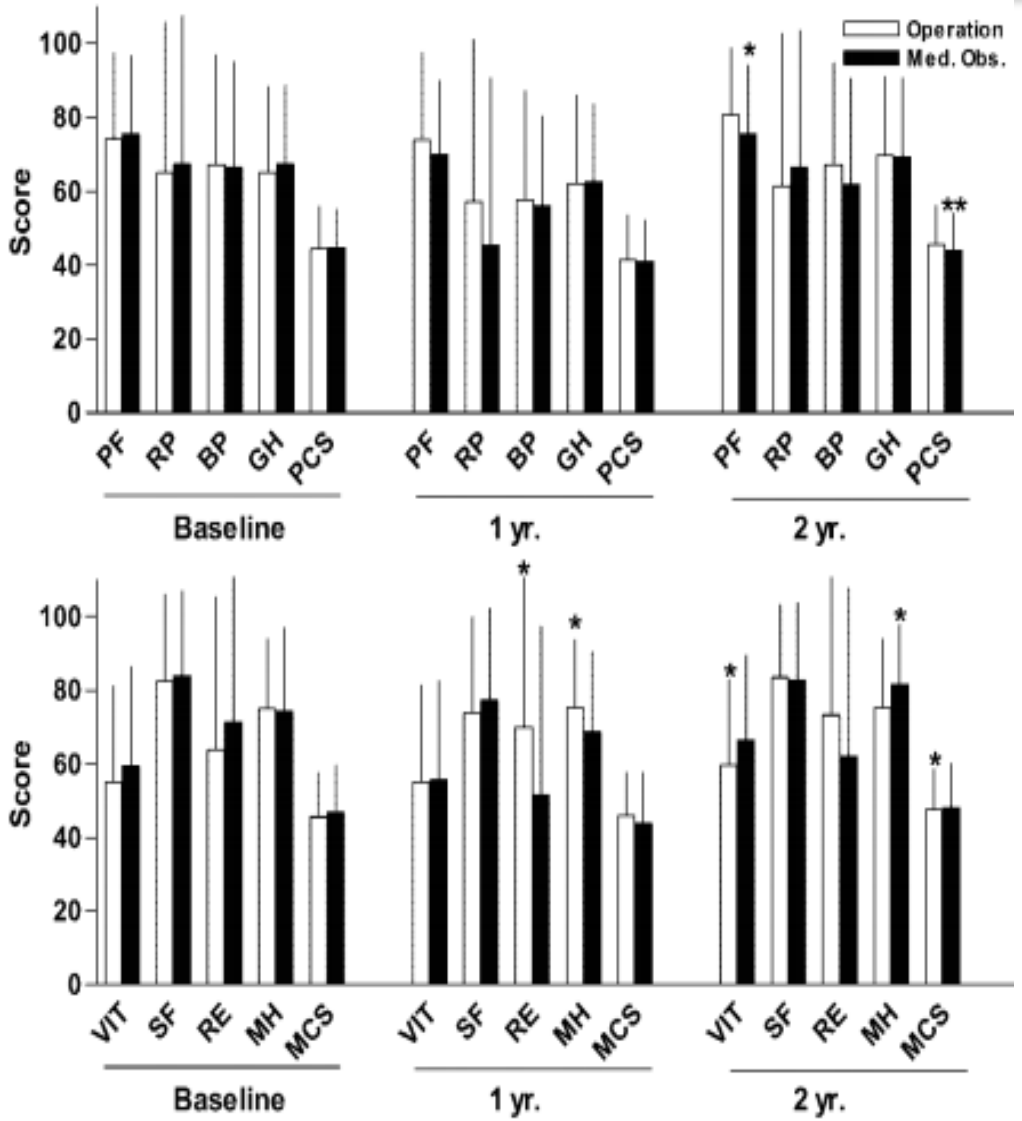
Medical Observation, Compared with Parathyroidectomy, for Asymptomatic Primary Hyperparathyroidism: A Prospective, Randomized Trial

TABLE 3. Baseline QoL score (SF-36) and CPRS both in relation to normative Swedish data

Description	Patients	Normal values
SF-36 standardized scores		
Physiological functioning	74.9 ± 22.1	75.1 ± 26.1
Role physical	66.3 ± 40.5	72.5 ± 38.6
Bodily pain	66.8 ± 29.0	68.0 ± 28.7
General health	66.4 ± 22.2	68.9 ± 23.7
Vitality	57.5 ± 26.4[a]	67.7 ± 25.4
Social functioning	83.2 ± 23.3[b]	86.7 ± 22.0
Role emotional	67.8 ± 41.1[a]	80.9 ± 33.6
Mental health	74.6 ± 21.1[a]	80.9 ± 33.6
Physical component summary	44.6 ± 11.0	45.2 ± 11.6
Mental component summary	46.3 ± 12.4[a]	51.0 ± 10.4
CPRS	9.63 ± 7.63[a]	6.22 ± 4.10

Data are presented as mean and SD.
[a] $P < 0.05$.
[b] $P < 0.01$.





J Bollerslev et al. J Clin Endocrinol Metab 92: 1687–1692, 2007

Conclusioni

Guidelines for the Management of Asymptomatic Primary Hyperparathyroidism: Summary Statement from the Fourth International Workshop

John P. Bilezikian, Maria Luisa Brandi, Richard Eastell, Shonni J. Silverberg, Robert Udelsman, Claudio Marcocci, and John T. Potts Jr

Cardiovascular Manifestations

….. A reasonable conclusion to be drawn at this time, therefore, is that **tests for cardiovascular involvement should not be part of the evaluation in PHPT**, and that parathyroid **surgery should not be performed to improve cardiovascular endpoints**, if abnormalities are present.

Neurocognitive Symptoms

……Until more definitive evidence becomes available, such **nonspecific symptomatology should not be used to recommend parathyroidectomy**.

Nonetheless, some patients with neurocognitive symptoms do appear to benefit from surgical intervention. Formal **neuropsychiatric or neurocognitive testing** in PHPT, although appropriate for a research agenda in this disease, **is not recommended**.

J Clin Endocrinol Metab 99: 3561–3569, 2014

Conclusioni

Italian Society of Endocrinology Consensus Statement: definition, evaluation and management of patients with mild primary hyperparathyroidism

C. Marcocci · M. L. Brandi · A. Scillitani · S. Corbetta · A. Faggiano · L. Gianotti · S. Migliaccio · S. Minisola

• 20. We **recommend against instrumental cardiovascular evaluation** (echocardiogram, carotid intima-media thickness) in all patients with mild PHPT (1 ØØOO).

• 21. We **recommend against psychometric or cognitive evaluation routinely** in all patients with mild PHPT (1 ØØOO).

• 22. We suggest to evaluate clinical cardiovascular measures such as blood pressure and heart rate as well as metabolic parameters such as BMI, blood glucose and lipid levels in all patients with mild PHPT accordingly to the standard clinical practice (2 ØOOO).

• 30. We suggest **considering PTX in patients with mild PHPT and increased cardiovascular risk**, **if the surgical risk is low** (2 ØOOO

• 31. We recommend **against PTX in patients with mild PHPT because of associated neurocognitive disorders** (1 ØØOO).

J Endocrinol Invest. 38:577–593, 2015

Grazie dell'attenzione