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ABSTRACTS OF SHORT PAPERS

(* denotes poster presentation)

Page

TRH—TSH, Thyroid Gland (Abstracts 1—15)

<i>F. W. Erhardt, T. Hashimoto</i> : Biological and immunological activities of high-molecular TSH from human pituitaries. *)	1
<i>K.-D. Döhler, A. von zur Mühlen, U. Döhler, E. Fricke</i> : Development of the pituitary-thyroid axis in male and female rats	2
<i>M. Klett, M. Hüfner, D. Schönberg, H. Rüttgers</i> : Reverse T ₃ immunoassay as a method for detection of neonatal hypothyroidism	3
<i>J. Bommer, B. Krempien, H. Huberti, S. Jedanzik, E. Gengenbach</i> : Influence of thyroid hormone on growth processes	4
<i>M. T. Ziem, G. H. Beastall, J. G. Ratcliffe, J. A. Thomson</i> : A neuropharmacological study of thyroid stimulating hormone (TSH) secretion in the rat	5
<i>W. Klingler, P. Göbel, F. J. Seif, H. Kalbacher, W. Voelter</i> : TSH and prolactin release of TRH and TRH analogues	6
<i>R. Finke, H. Schleusener, P. Kotulla, H. Sörje, C.H. Kim, K. W. Wenzel</i> : The radioligand receptor assay for Graves' disease: immunoglobulins – A prognostic test?	7
<i>J. Habermann, T. Eversmann, G. Ulbrecht, P. C. Scriba</i> : Stress causes increased urinary excretion of thyroid hormones	8
<i>V. Büber</i> : Therapeutic consequence of triiodothyronine deficiency during total fasting	9
<i>J. Hagemann, C. Schneider, J. Engels</i> : Serum thyroglobulin levels in benign and malignant thyroid diseases	10
<i>R. Hehrmann, B. Höffken, H. Creutzig</i> : Biological significance of thyroid hormone autoantibodies under experimental and clinical conditions	11
<i>G. Scherthaner, H. Ludwig, W. R. Mayr</i> : Humoral autoimmunity and immunogenetics in thyrotoxicosis, endocrine ophthalmopathy and in Hashimoto's thyroiditis. *)	12
<i>M. Hüfner, M. Grussendorf</i> : Induction of the T ₄ to T ₃ converting enzyme in rat liver by thyroid hormones and analogues	13
<i>B. Höffken, D. Auf dem Brinke, R. Ködding, R. D. Hesch</i> : T ₄ transformation in liver cell fractions	15
<i>M. Grussendorf, M. Ntokalou, M. Hüfner</i> : Pathways of thyroxine monodeiodination in rat liver homogenate	16

Gonadotropins, STH (Abstracts 16—26)

<i>W. E. Merz, M. Dörner</i> : Studies on the significance of the carboxyterminals of the subunits α and β for the properties of human chorionic gonadotropin (hCG)	17
<i>A. K. Mukhopadhyay, F. A. Leidenberger, G. Bettendorf</i> : Effect of cycloheximide in vitro on LHRH-induced release of LH from the anterior pituitary of male rats. *)	18
<i>H. Kuhl, H.-D. Taubert</i> : Acute effects of low doses of clomiphene upon LH-release and upon L-cystine arylamidase activity in the hypothalamus and the pituitary of the female rat	20
<i>L. Wildt, G. Leyendecker, W. Nocke</i> : Serum concentrations of alpha- and LH-beta-subunits in women under various physiological and pathological conditions	21
<i>J. L. Hancke, W. Beck, H. G. Baumgarten, K. Höhn, W. Wuttke</i> : Modulatory effect of noradrenaline and serotonin on circoral LH release in adult ovariectomized rats	22
<i>F. J. Seif, P. Sadowski, F. Heni</i> : Chorionic gonadotropin (hCG) in pheochromocytomas, carcinoid tumors, and vipomas	23

<i>J. S. E. Dericks-Tan, H. Bradler, H.-D. Taubert</i> : Different concentrations of LH, FSH, PRL, and HCG- β in serum and seminal fluid	24
<i>K. Hackenberg, H. G. Hoff, H. P. Brouwers, D. Reinwein</i> : Gonadotropin stimulation test in female Cushing's disease before and after adrenalectomy	25
<i>W. Winkelmann</i> : Plasma growth hormone response to TRH, LH-RH and bromocryptine in patients with acromegaly	26
<i>K.-H. Gless, E. Zillessen</i> : Selective ACTH and STH deficiency associated with ossification of the auricular cartilage: A case report	27
<i>G. Schwinn, C. McIntosh, C. Blosssey, J. Köbberling</i> : Effect of the dopamine receptor blocking agent pimozide on growth hormone levels in acromegalics	28

LH-RH Analogues (Abstracts 27—36)

<i>L. Tharandt, H. Schulte, G. Benker, K. Hackenberg, D. Reinwein</i> : Serum protein binding of 125-J-LH-RH and 125-J-6-D-Leu-10-Des-Gly-ethylamide-LH-RH (6-D-LH-RH) under treatment with the analogue in patients with isolated gonadotrophin deficiency (IGD)	29
<i>W. v. Rechenberg, J. Sandow, W. König</i> : Biological activity of LH-RH nonapeptides substituted in GLY ⁶ and LEU ⁷	31
<i>J. Sandow, W. König, B. Krauß</i> : Stability of LH-RH under different conditions of storage	32
<i>J. Sandow, W. v. Rechenberg, G. Jerzabek</i> : Endocrine effects of chronic treatment with the LH-RH analogue D-Ser (TBU) ⁶ ethylamide	33
<i>H. Vierhapper, W. Waldhäusl, J. Torres</i> : Release of LH and FSH after i.v.-administration of a new LH-RH-analogue in healthy males	34
<i>W. D. Hetzel, CH. Nierle, E. F. Pfeiffer</i> : Analytical study on the effect of GN-RH (DES-GLY-10-D-LEU-6) ethylamide	35
<i>J. S. E. Dericks-Tan, E. Hammer, H.-D. Taubert</i> : The effect of a new analog of LH-RH, D-SER-(TBU) ⁶ -EA ¹⁰ -LH-RH upon gonadotropin release in normally cyclic women	36
<i>W. Wiegelmann, H. G. Solbach, H. K. Kley, E. Nieschlag, H. L. Krüskemper</i> : A new LH-RH analogue: D-SER (TBU) ⁶ LH-RH 1—9 EA ¹⁰ . Effects on gonadotrophin and gonadal steroid secretion in men after intravenous and intranasal application	37
<i>E. Friedrich, A. Etzrodt, H. Becker, J. P. Hanker, E. Keller, P. Kleißl, A. E. Schindler, H. P. G. Schneider, H. I. Wyss</i> : A dose-response study with a new LH-RH-analogue	39
<i>J. Happ, J. Vogt, Th. Weber, U. Cordes, U. Krause, W. Callensee, J. A. Ermert, F. Kollmann, J. Beyer</i> : Therapeutic use of D-LEU ⁶ -DES-GLY ¹⁰ -GnRH-EA in males with idiopathic hypogonadotropic hypogonadism and in cryptorchidism	39

Prolactin (Abstracts 37—44)

<i>W. Beck, W. Wutke</i> : Desensitization of LH inhibitory dopamine receptors by prolactin or by apomorphine	40
<i>H. G. Bohnet, F. Gomez, H. G. Friesen</i> : Regulation of prolactin and estrogen receptors in the rat mammary gland during lactation	41
<i>H. H. Zippel, K.-D. Schulz, E. del Pozo</i> : Morphological studies in human proliferating mammary tissues under treatment with the prolactin inhibitor bromocryptine (CB 154). *)	42
<i>H. J. Künzig, K. D. Schulz, W. Geiger</i> : The influence of prolactin inhibition on the human menstrual cycle. *)	43
<i>D. Mühlenstedt, W. Wutke, H. P. G. Schneider</i> : Prolactin and short luteal phase infertility. *)	43
<i>W. Saeger, D. Lüdecke</i> : Morphological studies on the etiology of hyperprolactinemia in acromegaly. *)	44

<i>R. Fahlbusch, H. K. Rjosk, K. v. Werder</i> : Perioperative prolactin levels in patients with prolactinomas	46
<i>D. Barwich, H. Junkermann, F. Bahner</i> : Prolactin secretion in the postadrenalectomy syndrome	47

Somatostatin, Pancreas (Abstracts 45—50)

<i>U. Loos, F. Escobar-Jimenez, S. Raptis, J. Birk, G. Rothenbuchner, E. F. Pfeiffer</i> : Inhibition of TSH-induced release of triiodo-thyronine (T ₃) and thyroxine (T ₄) by somatostatin in man	47
<i>O. A. Müller, R. Landgraf, R. Ziegler, P. C. Scriba</i> : Effects of somatostatin on calcitonin and ectopic ACTH release in a patient with medullary thyroid carcinoma	49
<i>U. Schwedes, S. Szabo</i> : Effect of somatostatin on cysteamine-induced duodenal ulcer in rats	50
<i>S. Raptis, F. Escobar-Jimenez, V. Maier, R. Müller, J. Rosenthal</i> : Somatostatin and pentoxifylline modulation of insulin, glucagon and growth hormone following stimulation by arginine	51
<i>U. Gaertner, Chr. Schudt</i> : Adult rat liver parenchyma cells in primary culture: Hormone sensitivity and influence of insulin and glucose on glycogen synthase activity. *)	53
<i>Th. Zilker, H. Wiesinger, R. Ermler, U. Schweigart, P. Bottermann</i> : C-peptide levels following i.v. GTT in different degrees of kidney failure	54

Testes (Abstracts 51—57)

<i>F. Neumann, B. Schenck</i> : Morphogenesis of giant cells following regressive changes of the germinal epithelium in the rat thioglucose model. *)	55
<i>W. Gusek, G. Zendt, O. Tuncer</i> : Leydig Cells in rats and their fine structure after the application of antiandrogen. *)	56
<i>H. Steinbeck, S. H. Hasan, K.-J. Gräf, F. Neumann</i> : Influence of gestonorone caproate on the effects of "pure" antiandrogens in male rats	56
<i>W. Fröhlich, B. Schenck</i> : Effect of oestradiol-17 β on Leydig cell function in rats	58
<i>B. Schenck, F. Neumann</i> : Sertoli cell function and unilateral cryptorchidism in rats	59
<i>E. J. Wickings, E. Nieschlag</i> : Dynamics of the pituitary-gonadal feedback during active immunisation with testosterone in the male rhesus monkey	60
<i>W. Meuser, E. Nieschlag</i> : Sex hormones and vocal register in adult men. *)	61

Binding and Metabolism of Steroids (Abstracts 58—74)

<i>H. G. Bohnet, F. Gomez, H. G. Friesen</i> : Estrogen and progesterone binding to rat uterine cytosol during the estrus cycle and the puerperium	61
<i>G. Daxenbichler, H. J. Grill, J. L. Wittliff, O. Dapunt</i> : Comparison of specific estrogen and progesterone binding proteins in human myometrium and leiomyomas	62
<i>D. Naber, B. Pfeiff, D. Gollmer, K.-O. Mosebach</i> : A disc electrophoretic system for analyzing labelled androgen-binding proteins in target tissues. *)	63
<i>M. Krieg, W. Bartsch, K. D. Voigt</i> : Quantification of androgen binding, androgen tissue levels and sex hormone-binding globulin in human benign prostatic hypertrophy, skeletal muscle and plasma	64
<i>Th. Erdmann, H.-J. Horst</i> : Uptake, binding and metabolism of orally administered testosterone undecanoate in androgen target organs of the rat	65
<i>J. J. Ryan, B. Hoffmann</i> : Occurrence of protein-bound and water soluble metabolites from an anabolic steroid, trenbolone acetate (TBA) in cattle tissue. *)	67
<i>K. Herkner, J. Jörg, W. Waldhäusl, P. Nowotny, H. Haschek</i> : Measurement of steroid biosynthesis in human adrenals	68

<i>J. A. F. Tresguerres, K. Dahm, B. P. Lisboa</i> : In-vitro formation of 11-keto-testosterone in a feminizing adrenocortical adenoma. *)	69
<i>D. Engelhardt, P. Unterburger, F. Bidlingmaier, H. Mickan, H. J. Karl</i> : Virilization due to testosterone-producing stroma cell hyperplasia of the ovaries	70
<i>J. McK. Halket, G. Bettendorf, H.-E. Stegner, B. P. Lisboa</i> : Metabolism of pregnenolone in a virilizing ovarian tumor. *)	71
<i>R. Trolp, M. Breckwoldt, A. Hoff</i> : Metabolism of ^3H $\text{Oe}_1\text{-SO}_4$ in postmenopausal uterine tissue. *)	73
<i>D. Graf, R. Trolp, M. Breckwoldt</i> : Subcellular metabolism of ^3H $\text{Oe}_1\text{-SO}_4$ in human pregnancy myometrium. *)	73
<i>L. Raith, H. J. Bauer, H. J. Karl</i> : The extrahepatic metabolism of androgens in birds and mammals. *)	74
<i>H. Kieczka, H. Kappus, H. M. Bolt, H. Remmer</i> : Inhibition and stimulation of peroxidation of rat liver microsomal lipids by 2-hydroxy-ethinyloestradiol. *)	75
<i>H.-G. Dahnke, K.-O. Mosebach</i> : Influence of castration on the turnover of nuclear and cytoplasmic RNA in the ventral rat prostate	76
<i>P. Doerr, K. M. Pirke</i> : Suppression of the circadian rhythm of plasma testosterone in normal adult males by treatment with dexamethasone or hCG	77
<i>K. M. Pirke, R. Sintermann, P. Doerr</i> : Testosterone and testosterone precursors in the spermatic vein of young and old men	78

ACTH, Adrenal (Abstracts 75—80)

<i>J. Resetić, D. Lüdecke, M. Sekso</i> : Immunoreactive and biologically active ACTH in human plasma and isolated normal pituitary and ACTH adenoma cells	80
<i>H. Gerdes, B. Schollmeier, A. Scheuer, W. Spuck</i> : Experimental studies about the effect of lithium on ACTH-stimulated adrenals	81
<i>T. Herting, Ch. Lucke, H. C. Erbler</i> : Influence of lithium on plasma levels of vasopressin, corticosterone, aldosterone and renin activity in rats	81
<i>W. v. Petrykowski, J. Brämswig, J. Girard, U. Beck, D. Marquetand</i> : Disappointing results with cyproheptadine therapy in Cushing's disease and Nelson's syndrome	83
<i>W. Winkelmann, H. P. Brouwers, U. Fricke, W. Hadam, D. Heesen, H. G. Hoff, R. Mies</i> : Effect of cyproheptadine on vasopressin induced plasma cortisol increase in normal persons and in patients with Cushing's syndrome	84
<i>H. Ludwig, G. Scherthaner</i> : Organ specific autoimmunity in idiopathic Addison's disease. *)	85

Endocrinology in Pregnancy (Abstracts 81—87)

<i>G. Reck, H. Nowostawskij, M. Breckwoldt</i> : Plasma levels of free oestriol and cortisol under ACTH and dexamethasone during late pregnancy	86
<i>J. R. Strecker, R. I. Negulescu, Ch. Lauritzen, S. Pal</i> : The effect of betamethasone on the steroid metabolism of fetus and placenta	87
<i>J. R. Strecker, Ch. Lauritzen, W. Gossler</i> : On the regulation of precursor production for estrogen biosynthesis in the fetal adrenal cortex	88
<i>W. D. Lehmann, J. Strecker</i> : Estrogens in maternal plasma following intraamniotic injection of (^3H)-dehydroepiandrosterone-sulfate in midpregnancy	89
<i>W. Geiger, A. Debus</i> : Radioimmunological measurement of alphafetoprotein in various biological fluids of mother and child during pregnancy	90
<i>F. Ellendorff, M. Forsling, M. Taverne, F. Elsaesser, C. Naaktgeboren, N. Parvizi, D. Smidt</i> : Peripartal plasma oxytocin secretion in the miniature pig	91
<i>A. S. Wolf, Ch. Lauritzen, K. Musch, J. R. Strecker</i> : Studies on the biogenesis of steroids by placenta perfusion	92

Prostaglandins, Aldosterone, Angiotensin (Abstracts 88—94)

<i>H. P. Zahradnik, E. Kraut, U. Flecken, M. Breckwoldt</i> : Local effect of progesterone on prostaglandin $F_{2\alpha}$ -levels in menstrual blood	93
<i>W. Elger, R. Korte, S. H. Hasan</i> : Changes in serum progesterone (SP) and their significance in prostaglandin (PG)-induced abortions in early pregnancy of rhesus monkeys	94
<i>H. Vetter, J. M. Bayer, R. Beckerhoff, G. Brecht, J. Furrer, Ch. Glanzmann, F. Krück, G. Poulidis, W. Siegenthaler, W. Vetter</i> : Validity of lateralization procedures in primary aldosteronism	95
<i>B. A. Schölkens, H. G. Vogel</i> : Significance of (ILE ⁵) and (VAL ⁵) in 1,8-disubstituted antagonists of angiotensin II	95
<i>W. Oelkers, M. Schöneshöfer, G. Schultze, M. Wenzler, B. Bauer, M. L'age</i> : Chronic stimulation of aldosterone secretion by ILE ⁵ -angiotensin II in man	96
<i>R. Beckerhoff, J. Furrer, W. Vetter, W. Siegenthaler</i> : Renal, adrenal and vascular effects of 1-SAR-8-ALA-angiotensin II (Saralasin) in normotensive and hypertensive man	97
<i>C. Lucke, T. Herting, H. Erbler</i> : Studies on the secretion of arginine vasopressin (AVP), aldosterone, corticosterone and plasmarenin activity (PRA) during water deprivation. *)	98

PTH, Calcitonin, Calcium Metabolism (Abstracts 95—101)

<i>G. Dorn, R. Montz</i> : The preparation of a human PTH standard from 75-Se labeled medium of tissue culture. *)	99
<i>H. Jüppner, H. Ebel, J. Thiele</i> : Characterization of the PTH inactivating enzyme	100
<i>A. Schäfer, Th. Exsternbrink, M. Jäckel, H. Minne</i> : The influence of calcium, sodium fluoride, vitamin D and cortisone on the development of disuse atrophy of bone in the rat. *)	102
<i>H. Minne, D. Schäfer, F. Raue, Ch. Herfarth, R. Ziegler</i> : Newborn tetany: An indicator for maternal primary hyperparathyroidism	102
<i>D. Scholz, P. O. Schwille, R. Thun</i> : Intestinal calcium absorption and related blood constituents as influenced by proximal gastric vagotomy	103
<i>J. P. Nordmeyer, M. Clausen, R. Montz</i> : Modifying effect of thyroxine on the renal action of parathyrin in the rat	104
<i>J. Knop, R. Montz, J. P. Nordmeyer, C. Schneider</i> : New aspects of ⁴⁷ calcium kinetics in primary hyperparathyroidism and hyperthyroidism	105

Methods and Varia (Abstracts 102—117)

<i>J. P. Hanker, G. Schellong, H. P. G. Schneider</i> : Influence of high-dose estrogen treatment on pubertal development of tall girls	106
<i>F. Elsaesser, N. Parvizi</i> : Prepubertal active immunization against gonadal steroids: Effect on estrus, ovulation and the oscillatory pattern of plasma LH and progesterone in the female pig	107
<i>F. Hölzel, G. Schwermer, K. P. Hirche</i> : Ovarian cycle and DNA-synthesis in mammary tumor bearing rats	108
<i>K. Demisch, L. Demisch</i> : Activity of human platelet monamine oxydase in endocrine diseases and during endocrine function tests	109
<i>A. von zur Mühlen, T. Hashimoto, K. D. Döhler, E. Emrich</i> : Cytochemical TSH bioassay in plasma of patients with "euthyroid goiter" and negative TRH-test	110
<i>K. Horn, Th. Kubiczek, C. R. Pickardt</i> : Thyroxine-binding globulin (TBG): Preparation, radioimmunoassay and clinical significance. *)	111
<i>C. Bernutz, K. Horn, C. R. Pickardt</i> : Corticosteroid-binding globulin (CBG): Isolation and radioimmunological determination in serum. *)	112

<i>R. Aderjan, Th. Winkler, P. Vecsei</i> : Production of antibodies for a cortolone radioimmunoassay. *)	113
<i>M. Mok, K. H. Gless, P. Vecsei</i> : Tetrahydrodeoxycorticosterone radioimmunoassay in human urine after chromatography. *)	115
<i>H. Meinhold, P. Schürnbrand</i> : Radioimmunoassay for 3,3'-diiodothyronine in serum, amniotic fluid and thyroid tissue. *)	116
<i>R. Hehrmann, J. P. Nordmeyer, R. Wilke, R.-D. Hesch</i> : Radioimmunoassay of human PTH: characterization of two new antisera from sheep and clinical results	117
<i>M. Hüfner</i> : Stereospecific antibodies against L-thyroxine (L-T ₄). *)	118
<i>G. Emons, P. Ball, R. Knuppen</i> : Radioimmunoassay of 2-hydroxyestrone. *)	119
<i>G. Bastert, P. Althoff, K. H. Usadel, H. P. Fortmeyer</i> : Human fetal pituitaries in NU/NU mice	120
<i>K. H. Usadel, U. Schwedes, G. Bastert, H. P. Fortmeyer</i> : Transplantation of human fetal pancreas in NU/NU mice	121
<i>F. Haase, S. Beier, D. Hartmann, W. Elger</i> : Development of a qualitative canine bioassay for gestagens	122

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The association of a calcitonin-producing tumor with ectopic ACTH production is rare.—Although the importance of somatostatin (SRIF) in modulating the secretion of a variety of polypeptide hormones has been amply demonstrated, there is so far no information about the influence of SRIF on calcitonin (CT) and ectopic ACTH release.

Therefore we studied a 38-year-old man who had two weeks prior to our investigations undergone total resection of his goiter because of medullary thyroid carcinoma (MCT). He complained about rapid weight loss, diffuse abdominal pain, dyspnea, profuse diarrhea and muscular cramps. Blood pressure was elevated and a nonregional spread of the MTC in the liver and in a variety of lymph nodes diagnosed. Although immediate combined chemotherapy was started, the patient died two months later due to massive visceral dissemination of the tumor. A postmortem examination was refused.

The main *laboratory findings* postoperatively were the following: The mean serum CT-levels were extremely elevated (about 40 ng/ml). Serum calcium was low normal (4.5 mEq/l); serum phosphorus was normal. Serum K^+ : 2.4 mEq/l and Na^+ : 148 mEq/l; urinary excretion in 24 h for K^+ was 90 mEq and for Na^+ 60 mEq. The arterial blood gases (mm Hg) were: pO_2 66, pCO_2 53; pH 7.57; base excess +22. Basal serum cortisol was 86 μ g/100 ml, after exogenous ACTH 67 μ g/100 ml and after dexamethasone (2 mg) 64 μ g/100 ml. Plasma renin activity was completely suppressed due to a tenfold increase of serum corticosterone (13.4 μ g/100 ml) with increased urinary excretion of tetrahydro-DOC glucuronide (Dr. Vecsei, Heidelberg). Urinary aldosterone excretion, however, was very low. The basal plasma ACTH levels were markedly elevated (initially around 1 ng/ml, increasing within 8 weeks up to 20 ng/ml), both with radioimmunoassay and with bioassay. There was no decrease of plasma ACTH under 8 mg dexamethasone for two days. The common association of MCT with pheochromocytoma could be excluded in our patient by the normal values of metanephrine and vanillylmandelic acid. Low potassium and glucocorticoid excess led to an overt diabetes mellitus with hypoinsulinemia. An additional overproduction of vaso-inhibitory peptide (VIP) could be excluded by normal serum VIP values (Dr. Bloom, London), although profuse diarrhea, low potassium, low gastric acid output after pentagastrin stimulation and elevated basal gastrin values (175 pg/ml) were initially thought to be due to a Verner-Morrison syndrome. Increased serotonin production as cause for the gastrointestinal symptoms could also not be shown.

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Time min	Calcitonin ng/ml	ACTH ng/ml	Insulin μ U/ml	Prolactin μ U/ml	
— 15	41	---	18	163	before SRIF
0	48	20.48	19	129	
15	13	17.25	7	1202	during SRIF
30	17	4.93	9	1297	
60	35	2.12	12	915	
90	80	2.60	9	729	
120	96	1.53	14	557	
150	81	2.54	26	556	after SRIF
180	33	1.23	31	486	

The *influence* of SRIF on calcitonin and ACTH release was tested using 250 µg SRIF (i.v.) as a bolus followed by a two-hour infusion with 500 µg. The effectiveness of the inhibitory action of SRIF could be demonstrated by the insulin release pattern as shown in the Table. CT decreased initially, followed by a marked increase during SRIF infusion. ACTH could be markedly suppressed, followed by a slight rebound after removal of SRIF. There was a so far unexplainable paradoxical rise of prolactin under SRIF. Growth hormone and TSH were already completely suppressed by the glucocorticoid excess and thyroid hormone therapy, respectively.

Conclusion: The inhibitory effect of SRIF is only briefly or not observed on CT secretion, as shown in this patient and further confirmed by the study of a second patient with MCT [1]. In contrast, ectopic ACTH was diminished markedly during the infusion of SRIF. The same phenomenon could be demonstrated in patients with pituitary ACTH excess [2]. Therefore SRIF cannot be regarded as a tool for CT reduction, whereas disorders with ACTH excess might be therapeutically influenced by SRIF.

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