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Anorexia nervosa
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idiopathic short stature
insulin

Plasma Growth Hormone, Insulin, and Glucagon Responses to Arginine Infusion in Children and Adolescents with Idiopathic Short Stature, Isolated Growth Hormone Deficiency, Panhypopituitarism, and Anorexia Nervosa

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Extract

The effects of intravenous infusion of arginine (20 g/m^2) after an overnight fast on plasma immunoreactive growth hormone (GH), insulin (IRI), and glucagon (IRG), and blood glucose were examined in five groups of children and adolescents: 10 normal individuals, 18 with idiopathic short stature, 6 with isolated growth hormone deficiency, 8 with panhypopituitarism, and 6 with anorexia nervosa. The mean fasting plasma GH concentration was significantly elevated in the group with anorexia nervosa ($P < 0.05$), and similar to the value for the normal group in all other groups. After arginine infusion, four- to sixfold increases of plasma GH were observed in the normal children, and similar increases were seen in those with idiopathic short stature as well as in those with anorexia nervosa; whereas, in the children with isolated growth hormone deficiency or panhypopituitarism, there was no significant increase in plasma GH. Fasting blood glucose concentrations were significantly lower than normal in subjects with isolated growth hormone deficiency ($P < 0.05$), panhypopituitarism ($P < 0.001$), and anorexia nervosa ($P < 0.001$), whereas fasting plasma IRI and IRG concentrations were similar to the values in the normal group. Plasma IRI increased eightfold at the end of the 30-min arginine infusion in the normal subjects; the increase was slightly but not significantly less in those with idiopathic short stature, and significantly less in those with isolated growth hormone deficiency ($P < 0.05$), panhypopituitarism ($P < 0.001$), and anorexia nervosa ($P < 0.05$). Arginine infusion resulted in two- to threefold increases of plasma IRG in the normal group, and similar increases were observed in all of the other groups tested. These results suggest that whereas pancreatic β cell responsiveness may be deficient in children and adolescents with isolated growth hormone deficiency, panhypopituitarism, or anorexia nervosa, pancreatic α cell responsiveness, to arginine at least, appears to be intact under these conditions.

Speculation

Although plasma glucagon responses to arginine infusion were not less in subjects with hypopituitarism or anorexia nervosa than in normal subjects, relative hypoglucagonemia may have existed, since both basal and postarginine infusion plasma glucagon levels were not higher than normal in the presence of significantly lower blood glucose values. Thus, the present study does not exclude a deficient pancreatic α cell response to hypoglycemia. Alternatively, nonavailability of substrates for gluconeogenesis may have a more important influence than hormonal factors in the genesis of the hypoglycemia observed in these states.

The relationship between hypopituitarism and hypoglycemia has long been recognized (6) and is the subject of a recent review by Hopwood *et al.* (26). Among factors regulating the blood glucose concentration, pancreatic insulin, as well as glucagon (48), is recognized to have a prominent role. The influence of growth hormone on pancreatic β cell function is well documented, and insulin responses to provocative stimuli have usually been reported

Table 1. *Characteristics of subjects studied*

Subjects	Age, yr/mo	Height, cm	Weight, kg
Normal children			
Mean ± SE (10)[1]	13/3 ± 1/10	156 ± 12	43.4 ± 4.9
Idiopathic short stature			
Mean ± SE (18)	10/5 ± 1/10	122 ± 5	24.8 ± 2.3
Isolated growth hormone deficiency			
CP	12	130	37
RP	9/6	120	22
SS	14	126	28
GR	14/6	140	50
LP	14	138	36
DO	15	133	29
Mean ± SE (6)	13/8 ± 0/6	131 ± 7	33.7 ± 4
Panhypopituitarism			
MG	4	82	16
BS	4/6	86	17
GC	5/6	87	19
RC	5/6	116	18.5
DL	9/6	126	21
BP	10	127	32
VD	13	142	28
FI	15	145	40
Mean ± SE (8)	9/3 ± 1/4	114 ± 9	24 ± 3
Anorexia nervosa			
CI	12/6	164	37
CN	14/10	168	38
RE	13	154	36
SI	15	157	32
RT	16	160	34
PG	13/3	170	43
Mean ± SE (6)	14/4 ± 0/6	162 ± 3	36.7 ± 1.5

[1] Mean ± SE values for number of subjects indicated in parentheses.

Table 2. *Effect of arginine infusion on plasma growth hormone*[1]

Subjects	−30 min	0 min	30 min	60 min	90 min	120 min
Normal children						
Mean ± SE (10)	2.8 ± 1.0	2.5 ± 0.6	10.7 ± 3.8[2]	15.6 ± 3.2[3]	11.6 ± 3.6[2]	4.7 ± 1.9
Idiopathic short stature						
Mean ± SE (18)	2.5 ± 0.6	2.5 ± 0.7	10.6 ± 1.4	14.3 ± 2.3	6.1 ± 0.8	4.7 ± 0.9
Isolated growth hormone deficiency						
CP	1.9	0.4	1.5	0.9	0.6	0.4
RP	2.1	3.3	4.4	3.0	4.2	3.0
SS	0.7	1.4	3.5	4.7	0.8	1.0
GR	0.8	0.6	1.4	1.0	0.9	1.4
LP	2.0	1.1	0.4	1.6	1.7	2.1
DO	0.7	0.6	4.0	4.0	2.3	1.8
mean ± SE (6)	1.4 ± 0.3	1.2 ± 0.4	2.5 ± 0.7	2.5 ± 0.7[4]	1.9 ± 0.5[5]	1.6 ± 0.4
Panhypopituitarism						
MG	1.0	0.7	1.0	3.4	0.8	0.7
BS	1.1	1.8	1.7	2.2	2.0	2.0
GC	1.6	1.4	0.8	1.0	1.1	1.2
RC	1.0	1.0	0.4	1.6	1.3	1.4
DL	2.0	2.1	2.1	2.0	2.4	1.5
BP	1.0	1.2	1.0	1.5	2.0	1.5
VD	3.2	2.7	3.7	5.2	4.3	3.4
FI	2.0	2.5	1.7	2.9	2.7	2.3
Mean ± SE (8)	2.1 ± 0.5	2.1 ± 0.6	2.0 ± 0.5[5]	3.0 ± 0.8[4]	2.6 ± 0.6[5]	2.5 ± 0.6
Anorexia nervosa						
CI	0.9	1.4	5.0	10.0	6.3	4.4
CN	13.6	11.7	9.2	18.8	36.2	21.4
RE	11.0	1.2	3.4	28.6	11.6	5.0
SI	5.8	4.4	7.0	3.2	34.2	10.6
RT	35.6	32.2	30.3	36.8	47.7	48.4
PG	3.5	3.4	14.0	18.5	8.1	8.4
Mean ± SE (6)	11.7 ± 5.1[5]	7.4 ± 3.3	11.5 ± 4.1	24.1 ± 4.1[5]	24.0 ± 7.2	16.4 ± 6.9

[1] Arginine (20 g/m²) was infused over 30 min (from 0 to 30 min) after an overnight fast, and mean (±SE) plasma growth hormone concentrations (nanograms per milliliter) for numbers of subjects, indicated in parentheses, are shown at −30 to 120 min.

[2] $P < 0.05$ versus preinfusion (mean of −30 and 0 min) plasma growth hormone concentration.

[3] $P < 0.005$ versus preinfusion (mean of −30 and 0 min) plasma growth hormone concentration.

[4] $P < 0.005$ versus mean plasma growth hormone concentrations at corresponding times in the normal group

[5] $P < 0.05$ versus mean plasma growth hormone concentrations at corresponding times in the normal group.

to be decreased in subjects with growth hormone deficiency (8, 13, 18, 39, 41). Effects of growth hormone on pancreatic α cell function have only recently been reported (5, 20). The aim of the present experiments was to test pancreatic glucagon as well as insulin responsiveness in children and adolescents with deficiency of growth hormone secretion, and in others with idiopathic short stature or anorexia nervosa. Arginine infusion permitted the simultaneous assessment of plasma growth hormone, insulin, and glucagon secretory responses.

MATERIALS AND METHODS

SUBJECTS

The characteristics of the subjects studied are shown in Table 1. The first group consisted of 10 children and adolescents (4 females and 6 males, ages 6 years 9 months to 15 years) admitted to the surgery ward for minimal orthopedic trauma, but otherwise healthy, and these subjects served as "normal" control subjects. The second group consisted of 18 subjects (2 females and 16 males, ages 5 years 6 months to 15 years 5 months) with short stature (less than normal by 2 or more SD), and in whom no abnormality of pituitary, thyroid, or gonadal function was found. The third group consisted of 6 subjects (1 female and 5 males) with isolated GH deficiency. Plasma GH responses to insulin-induced hypoglycemia were severely blunted; plasma thyroxine levels were normal. Adrenocorticotropic hormone (ACTH) secretion was normal, as assessed indirectly after administration of metyrapone. Responses to synthetic gonadotropin-releasing hormone were normal in the 3 younger children with isolated GH deficiency, and gonadotropic secretion in the 3 older ones was considered normal, since pubertal development had begun. The fourth group consisted of 8 subjects (3 females and 5 males) with panhypopituitarism. All had GH, TSH, and ACTH deficiencies, and gonadotropic deficiency was demonstrated in 2 of the 8 cases. Diabetes insipidus was present in the 4 cases which had a tumoral origin, whereas, in the 4 idiopathic cases, diabetes insipidus was absent. All the patients with panhypopituitarism were receiving dessicated thyroid gland powder and cortisone substitution (25 mg/m²/24 hr). None of the patients had received human growth hormone before or during the time of testing. The fifth group consisted of 6 female adolescents with anorexia nervosa; weight loss was between 35% and 38% below ideal body weight, and amenorrhea was present.

METHODS

All of the studies were performed after informed consent. After 3 days of a diet consisting of 55% carbohydrate, 17% fat, and 28% protein, and after an overnight fast, an intravenous infusion of isotonic saline was established in an antecubital vein. After a 30-min equilibration period, arginine monohydrochloride 20 g/m² (20 g/100 ml, buffered to pH 7.4) was infused during 30 min (0–30 min). Blood was collected in chilled heparinized plastic tubes containing EDTA and Trasylol at −30, 0, 30, 60, 90, and 120 min. Plasma was stored at −20° until assayed. Blood glucose was

estimated by the AutoAnalyzer ferricyanide method (7). Plasma growth hormone and insulin concentrations were determined by double-antibody radioimmunoassays (24, 30). Plasma immunoreactive glucagon was assayed (49) using an antipancreatic glucagon antiserum which purportedly measures true pancreatic glucagon (30-K antiserum, kindly provided by Dr. R. H. Unger, University of Texas South-Western Medical School, Dallas, Texas).

RESULTS

As indicated in Table 1, the mean age of the control population of normal children was around early adolescence; the ratio of mean weight to mean height was approximately at the 50th percentile. The mean age of the children with idiopathic short stature was 10.5 years. The height of this group corresponded to that of normal 7-year-old children, and they were slightly overweight (at 90th percentile for height). The mean age, height, and weight of the groups with idiopathic short stature and panhypopituitarism were very similar. The group with isolated growth hormone deficiency had an adolescent mean age, and was also overweight (90th percentile for height). The group with anorexia nervosa was significantly underweight for height, the latter being comparable with the normal control group.

PLASMA IMMUNOREACTIVE GH (Table 2)

In normal children, plasma GH rose significantly from a mean (of −30 and 0 min) preinfusion fasting concentration of 2.7 ± 0.7 ng/ml to a peak value of 15.6 ± 3.2 ng/ml ($P < 0.005$) at 30 min after the end of the arginine infusion (60 min). The GH response to arginine infusion was similar in children with idiopathic short stature, with a peak value of 14.3 ± 2.3 ($P < 0.001$) at 60 min. By contrast, in children with isolated growth hormone deficiency and in those with panhypopituitarism, plasma GH levels after arginine infusion did not rise significantly, and the concentrations attained were significantly lower than the values in the normal subjects at corresponding times. The mean fasting preinfusion plasma GH concentration in the group with anorexia nervosa (9.6 ± 4.2 ng/ml) was significantly higher ($P < 0.05$) than the corresponding mean fasting value for the normal group (2.7 ± 0.7 ng/ml), and at 30 min after arginine infusion, plasma GH rose to significantly higher levels ($P < 0.05$) in the group with anorexia nervosa (24.1 ± 4.1 ng/ml) than in the normal subjects (15.6 ± 3.2 ng/ml).

BLOOD GLUCOSE (Table 3)

The mean fasting preinfusion blood glucose concentrations were significantly lower in subjects with isolated growth hormone deficiency (73 ± 5 mg/100 ml, $P < 0.05$), panhypopituitarism (60 ± 6 mg/100 ml, $P < 0.001$), and anorexia nervosa (63 ± 3 mg/100 ml, $P < 0.001$), than in normal subjects (83 ± 2 mg/100 ml). Fasting blood glucose concentrations were not significantly below normal in subjects with idiopathic short stature (83 ± 2 mg/100 ml). There was a small but nonsignificant increase of blood glucose in the normal subjects at the end of the 30-min arginine infusion (from 82 ± 2 to 95 ± 3 mg/100 ml) followed by a return to preinfusion or slightly lower values 30 min after the end of the infusion. Similar changes were seen in all other groups.

Table 3. *Effect of arginine infusion on blood glucose*[1]

Subjects	−30 min	0 min	30 min	60 min	90 min	120 min
Normal children						
Mean ± SE (10)	83 ± 2	82 ± 2	95 ± 3	71 ± 4	76 ± 2	83 ± 5
Idiopathic short stature						
Mean ± SE (18)	83 ± 3	82 ± 2	101 ± 4	73 ± 3	70 ± 3	79 ± 2
Isolated growth hormone deficiency						
CP	77	69	93	65	69	67
RP	52	48	48	34	32	72
SS	86	82	110	79	78	78
GR	82	79	102	64	77	84
LP	80	74	103	85	87	87
DO	75	72	102	90	62	54
Mean ± SE (6)	75 ± 5	71 ± 5[2]	93 ± 9	69 ± 8	67 ± 8	65 ± 9
Panhypopituitarism						
MG	51	46	68	55	34	39
BS	69	71	93	42	57	52
GC	32	31	30	33	38	37
RC	70	69	86	70	62	52
DL	69	70	124	70	72	59
BP	64	73	89	77	66	75
VD	52	57	40	60	45	38
FI	68	80	92	61	63	63
Mean ± SE (8)	59 ± 5[3]	62 ± 6[4]	78 ± 11	59 ± 5	55 ± 5[3]	52 ± 5[3]
Anorexia nervosa						
CI	68	57	75	25	19	52
CN	56	64	78	68	62	68
RE	60	58	71	67	65	63
SI	79	72	97	81	61	63
RT	56	70	69	63	70	50
PG	58	56	71	28	48	43
Mean ± SE (6)	63 ± 4[3]	63 ± 3[3]	77 ± 4[4]	55 ± 9	54 ± 8[2]	56 ± 4[3]

[1] Data are presented as in Table 2. Blood glucose concentrations at the times indicated are expressed in milligrams per 100 ml.

[2] $P < 0.05$ versus mean blood glucose concentration at corresponding times in the normal group.

[3] $P < 0.001$ versus mean blood glucose concentration at corresponding times in the normal group.

[4] $P < 0.005$ versus mean blood glucose concentration at corresponding times in the normal group.

Table 4. *Effect of arginine infusion on plasma insulin*[1]

Subjects	−30 min	0 min	30 min	60 min	90 min	120 min
Normal children						
Mean ± SE (10)	7.1 ± 1.6	7.1 ± 1.1	57.6 ± 8.7[2]	17.5 ± 3.3[3]	7.3 ± 1.5	5.5 ± 0.8
Idiopathic short stature						
Mean ± SE (18)	3.5 ± 0.6	3.9 ± 0.7	31.1 ± 6.1	8.7 ± 1.5	3.6 ± 0.7	3.1 ± 0.6
Isolated growth hormone deficiency						
CP	17.0	11.0	44.0	20.0	8.0	13.0
RP	1.0	1.0	4.5	1.0	1.0	2.0
SS	4.0	3.0	18.0	5.0	3.0	2.0
GR	15.0	15.0	56.0	24.0	7.0	1.0
LP	0.7	0.6	4.0	4.0	2.3	1.8
DO	1.0	1.5	15.0	15.5	4.0	1.5
Mean ± SE (6)	6.5 ± 3.1	5.4 ± 2.5	23.6 ± 9.0[4]	11.1 ± 4.1	4.2 ± 1.1	3.6 ± 1.9
Panhypopituitarism						
MG	1.0	1.0	1.0	1.0	0.5	2.0
BS	3.0	2.0	37.5	5.0	3.0	3.0
GC	3.5	4.0	5.0	4.0	4.5	5.5
RC	3.5	2.0	10.0	5.0	5.0	3.0
DL	0.4	0.6	0.8	0.4	1.0	0.8
BP	0.2	0.6	0.8	0.2	1.0	0.8
VD	5.0	5.0	9.0	9.0	7.0	5.0
FI	5.5	5.0	20.0	10.0	9.0	3.5
Mean ± SE (8)	3.1 ± 0.6	2.7 ± 0.6	10.5 ± 4.5[5]	6.3 ± 2.4	4.3 ± 1.0	3.2 ± 0.5
Anorexia nervosa						
CI	10.5	9.5	68.5	14.5	9.0	6.0
CN	0.5	0.5	2.8	1.5	0.5	0.5
RE	1.0	4.0	10.5	6.0	6.0	3.0
SI	3.0	4.0	20.0	12.0	3.0	2.0
RT	2.5	2.0	17.5	5.0	4.0	2.0
PG	14.5	3.5	30.0	7.5	5.0	6.0
Mean ± SE (6)	5.3 ± 2.4	3.9 ± 1.2	24.9 ± 9.5[4]	7.8 ± 1.9	4.6 ± 1.2	2.8 ± 0.8

[1] Data are presented as in Table 2. Plasma insulin concentrations are expressed in microunits per milliliter.
[2] $P < 0.001$ versus preinfusion (mean of −30 and 0 min), plasma insulin concentration.
[3] $P < 0.02$ versus preinfusion (mean of −30 and 0 min), plasma insulin concentration.
[4] $P < 0.05$ versus mean plasma insulin concentrations at corresponding times in the normal group.
[5] $P < 0.001$ versus mean plasma insulin concentrations at corresponding times in the normal group.

PLASMA IMMUNOREACTIVE INSULIN (IRI) (Table 4)

Fasting plasma IRI levels were not significantly different in the various groups. In normal children, plasma IRI rose significantly from a mean preinfusion concentration of 7.1 ± 1.2 μU/ml to 57.6 ± 8.7 at the end of a 30-min infusion of arginine ($P < 0.001$) and 17.5 ± 3.3 at 60 min ($P < 0.02$). The IRI responses to the infusion of arginine were lower than normal in children with idiopathic short stature (31.1 ± 6.1 μU/ml at 30 min); however, the difference from normal was not significant. By contrast, the increases of plasma IRI after arginine infusion were significantly smaller in the group of children with isolated growth hormone deficiency (23.6 ± 9.0 μU/ml at 30 min; $P < 0.05$), panhypopituitarism (10.5 ± 4.5 μU/ml at 30 min, $P < 0.001$), and anorexia nervosa (24.9 ± 9.5 μU/ml at 30 min, $P < 0.05$).

PLASMA IMMUNOREACTIVE GLUCAGON (IRG) (TABLE 5)

Fasting plasma IRG levels were not significantly different for the different groups. In normal children, plasma IRG increased significantly from a mean preinfusion concentration of 89 ± 23 pg/ml to 231 ± 60 pg/ml at the end of the 30-min infusion of arginine ($P < 0.005$) and 181 ± 39 pg/ml at 60 min ($P < 0.02$). The plasma IRG response to arginine infusion was not significantly less than normal in the group with idiopathic short stature. In both groups with growth hormone deficiency (isolated GH deficiency and panhypopituitarism) and in the group with anorexia nervosa, plasma IRG responses to arginine were slightly higher than in normal children; however, the differences were not significant.

DISCUSSION

In clinical investigation, the intravenous infusion of arginine has become a useful tool for stimulating growth hormone (15, 42), insulin (12), and glucagon secretion (3, 48), and has been widely used for diagnosis of growth hormone deficiency. Similarly, in the present study, plasma GH responses to arginine in children and adolescents with idiopathic short stature were not significantly different from those observed in normal control subjects, but were severely blunted in individuals with either isolated growth hormone deficiency or panhypopituitarism. Thereby, the arginine infusion test has been validated as a diagnostic tool.

In anorexia nervosa, basal plasma GH levels have been reported to be either high (9, 21, 29, 31, 37) or low (14), and plasma GH levels have been reported to be low in marasmus (4, 16, 17) or in maternal deprivation (45). On the contrary, they were found to be high in kwashiorkor (4, 16, 17, 36, 44). In two of our six cases with anorexia nervosa, plasma GH was clearly higher than normal, both in the fasting state, and at 60 and 90 min after arginine infusion.

Both stimulatory (8, 13, 35, 39, 40, 43, 46, 51) and inhibitory (1) effects of growth hormone on insulin secretion have been reported. In children with growth hormone deficiency, plasma IRI responses to arginine (8, 41, 46, 50, 51) or to glucose (47) have been reported as either normal or decreased. The mean plasma IRI responses to arginine in the groups with isolated growth hormone deficiency and panhypopituitarism were significantly decreased; however, there was a great individual variation, some subjects exhibiting a normal insulin response. The latter was usually associated with a

normal basal (preinfusion) plasma insulin level, in turn possibly related to the more normal weight to height ratio in these individuals (26). An age-dependent response of IRI to different stimuli in hypopituitarism has also been shown (26); however, this does not appear to account for the individual differences observed in the present study. It is well recognized that the blood glucose level can modulate the insulin secretory response to different agents, including arginine (10, 19, 34). Indeed, the poorest plasma IRI responses to arginine were observed in the groups (*i.e.*, anorexia nervosa, panhypopituitarism, and isolated growth hormone deficiency) which has blood glucose concentrations (fasting and postarginine infusion) lower than normal; however, the individual differences do not appear to be explained on this basis. Hypoinsulinemia has also been reported in chronic starvation (11, 17, 23) and prolonged fasting has been reported to blunt the insulin secretory response to arginine (2). Similarly, in the present study, the mean plasma IRI response to arginine in the six adolescent females with anorexia nervosa was significantly less than in the normal group. Also, in agreement with several reports (22, 25, 27), the group of children with idiopathic short stature had decreased plasma IRI levels, both before and after arginine, although the differences from normal were not significant.

A diminished plasma IRG response to arginine in growth hormone deficient adults was first reported by Goldfine *et al.* (20, 33). More recently, Blackard *et al.* (5) found normal plasma IRG responses to arginine in adult subjects with hypopituitarism before and after growth hormone administration. Normal glucagon responses were observed (28) in children with idiopathic short stature. In the present study, children and adolescents with isolated growth hormone deficiency or panhypopituitarism had plasma IRG levels similar to normal in the fasting state, and the plasma IRG responses to arginine were also similar to those observed in the normal group. Thus, prolonged pituitary GH deficiency does not appear to influence glucagon secretory responses, whereas insulin secretion may be clearly depressed. In prolonged fasting (2, 32, 38), plasma IRG has been shown to rise until the third day of fasting, and thereafter, to decrease to a level maintained at or slightly above postabsorptive levels (38). In our cases of anorexia nervosa, still largely underweight, fasting plasma IRG was not increased, and IRG responses to arginine were normal or slightly augmented as reported previously in starvation (2).

The present findings of fasting plasma IRG levels in the normal range, together with a blood glucose level significantly lower than normal in the groups with anorexia nervosa, isolated growth hormone deficiency, and panhypopituitarism suggest that the glucagon response to hypoglycemia may be relatively deficient in these conditions. However, plasma glucagon as well as blood sugar increments after arginine infusion were normal or slightly greater than normal. Therefore, whether the hypoglycemia associated with anorexia nervosa or with hypopituitarism may be attributed to deficient glucagon secretion, cannot be answered definitively from the present data.

SUMMARY

The effects of intravenous infusion of arginine (20 g/m²) on plasma immunoreactive GH, IRI, IRG, and blood glucose were examined in 10 normal children and adolescents, 18 with idi-

Table 5. *Effect of arginine infusion on plasma immunoreactive glucagon (IRG)*[1]

Subjects	−30 min	0 min	+30 min	+60 min	+90 min	+120 min
Normal children						
Mean ± SE (10)	102 ± 40	86 ± 23	231 ± 60[2]	181 ± 39[3]	107 ± 28	74 ± 22
Idiopathic short stature						
Mean ± SE (18)	118 ± 33	89 ± 30	182 ± 39	134 ± 29	101 ± 25	104 ± 25
Isolated growth hormone deficiency						
CP	265	175	240	255	150	180
RP	50	210	600	280	250	310
SS	45	35	70	55	20	40
GR	45	10	170	120	40	40
LP	60	40	280	250	100	40
DO	105	105	390	65	165	100
Mean + SE (6)	96 ± 36	102 ± 31	291 ± 76	171 ± 42	121 ± 35	118 ± 45
Panhypopituitarism						
MG	135	130	325	155	145	140
BS	200	160	760	330	275	130
GC	130	132	380	410	325	330
RC	20	40	240	230	110	40
DL	20	30	220	125	90	35
BP	20	25	255	145	62	65
VD	155	155	185	175	170	175
FI	80	45	195	180	145	92
Mean ± SE (8)	95 ± 25	90 ± 21	320 ± 67	219 ± 36	165 ± 32	125 ± 34
Anorexia nervosa						
CI	195	215	300	270	220	265
CN	45	15	160	130	95	75
RE	30	30	230	970	560	470
SI	60	70	240	285	170	150
RT	75	50	195	110	125	90
PG	30	35	75	85	120	30
Mean ± SE (6)	72 ± 26	69 ± 30	200 ± 32	308 ± 137	215 ± 71	180 ± 70

[1] Data are presented as in Table 2. Plasma glucagon concentrations are expressed in picograms per milliliter.

[2] $P < 0.005$ versus preinfusion (mean of −30 and 0 min) plasma glucagon concentration.

[3] $P < 0.02$ versus preinfusion (mean of −30 and 0 min) plasma glucagon concentration.

opathic short stature, 6 with isolated growth hormone deficiency, 8 with panhypopituitarism, and 6 with anorexia nervosa.

In the group with idiopathic short stature, fasting, as well as postarginine infusion plasma GH, IRI, IRG, and blood glucose concentrations, were similar to the values in the normal group. Plasma GH response to arginine was essentially absent in the groups with isolated GH deficiency and panhypopituitarism, but intact in anorexia nervosa; however, in all three groups fasting blood glucose concentrations were significantly diminished and plasma IRI responses to arginine were significantly decreased. Plasma IRG, both fasting and after arginine infusion, was not significantly different from normal in all of the groups studied.

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