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NAME	**: Mrs. SUKHMANI DUGGAL**		
AGE/ GENDER	: 31 YRS/FEMALE	**PATIENT ID**	: 1654689
COLLECTED BY	: SURJESH	**REG. NO./LAB NO.**	**: 012410270019**
REFERRED BY	: CENTRAL PHOENIX CLUB (AMBALA CANTT)	**REGISTRATION DATE**	: 27/Oct/2024 09:36 AM
BARCODE NO.	: 01519631	**COLLECTION DATE**	: 27/Oct/2024 09:51AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	**REPORTING DATE**	: 27/Oct/2024 11:58AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval

CLINICAL CHEMISTRY/BIOCHEMISTRY

GLUCOSE FASTING (F)

GLUCOSE FASTING (F): PLASMA *by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD)*	89.64	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0 DIABETIC: > 0R = 126.0

INTERPRETATION

IN ACCORDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES:

1. A fasting plasma glucose level below 100 mg/dl is considered normal.
2. A fasting plasma glucose level between 100 - 125 mg/dl is considered as glucose intolerant or prediabetic. A fasting and post-prandial blood test (after consumption of 75 gms of glucose) is recommended for all such patients.
3. A fasting plasma glucose level of above 125 mg/dl is highly suggestive of diabetic state. A repeat post-prandial is strongly recommended for all such patients. A fasting plasma glucose level in excess of 125 mg/dl on both occasions is confirmatory for diabetic state.

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Test Name	Value	Unit	Biological Reference interval

URIC ACID

URIC ACID: SERUM	4.59	mg/dL	2.50 - 6.80

by URICASE - OXIDASE PEROXIDASE

INTERPRETATION:-

1.GOUT occurs when high levels of Uric Acid in the blood cause crystals to form & accumulate around a joint.
2.Uric Acid is the end product of purine metabolism . Uric acid is excreted to a large degree by the kidneys and to a smaller degree in the intestinal tract by microbial degradation.

INCREASED:-

(A).DUE TO INCREASED PRODUCTION:-

1.Idiopathic primary gout.
2.Excessive dietary purines (organ meats,legumes,anchovies, etc).
3.Cytolytic treatment of malignancies especially leukemais & lymphomas.
4.Polycythemai vera & myeloid metaplasia.
5.Psoriasis.
6.Sickle cell anaemia etc.

(B).DUE TO DECREASED EXCREATION (BY KIDNEYS)

1.Alcohol ingestion.
2.Thiazide diuretics.
3.Lactic acidosis.
4.Aspirin ingestion (less than 2 grams per day).
5.Diabetic ketoacidosis or starvation.
6.Renal failure due to any cause etc.

DECREASED:-

(A).DUE TO DIETARY DEFICIENCY

1.Dietary deficiency of Zinc, Iron and molybdenum.
2.Fanconi syndrome & Wilsons disease.
3.Multiple sclerosis .
4.Syndrome of inappropriate antidiuretic hormone (SIADH) secretion & low purine diet etc.

(B).DUE TO INCREASED EXCREATION

1.Drugs:-Probenecid , sulphinpyrazone, aspirin doses (more than 4 grams per day), corticosterroids and ACTH, anti-coagulants and estrogens etc.

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Test Name	Value	Unit	Biological Reference interval

ENDOCRINOLOGY

LUTEINISING HORMONE (LH)

LUTEINISING HORMONE (LH): SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	3.51	mIU/mL	MALES: 0.57 - 12.07 FOLLICULAR PHASE: 1.80 - 11.78 MID-CYCLE PEAK: 7.59 - 89.08 LUTEAL PHASE: 0.56 - 14.0 POST MENOPAUSAL WITHOUT HRT: 5.16 - 61.99

INTERPRETATION:

1. Luteinizing hormone (LH) is a glycoprotein hormone consisting of 2 non covalently bound subunits (alpha and beta). Gonadotropin-releasing hormone from the hypothalamus controls the secretion of the gonadotropins, FSH and LH, from the anterior pituitary.
2. In both males and females, LH is essential for reproduction. In females, the menstrual cycle is divided by a mid cycle surge of both LH and FSH into a follicular phase and a luteal phase.
3. This "LH surge" triggers ovulation thereby not only releasing the egg, but also initiating the conversion of the residual follicle into a corpus luteum that, in turn, produces progesterone to prepare the endometrium for a possiblei mplantation.
4. LH supports thecal cells in the ovary that provide androgens and hormonal precursors for estradiol production. LH in males acts on testicular interstitial cells of Leydig to cause increased synthesis of testosterone.

The test is useful in the following situations:

1. An adjunctin the evaluation of menstrual irregularities.
2. Evaluating patients with suspected hypogonadism
3. Predicting ovulation & Evaluating infertility
4. Diagnosing pituitary disorders
5. In both males and females, primary hypogonadism results in an elevation of basal follicle-stimulating hormone and luteinizing hormone levels.

FSH AND LH ELEVTED IN:

1. Primary gonadal failure
2. Complete testicular feminization syndrome
3. Precocious puberty (either idiopathic or secondary to a central nervous system lesion)
4. Menopause
5. Primary ovarian hypo dysfunction in females
6. Polycystic ovary disease in females
7. Primary hypogonadism in males

LH IS DECREASED IN:

1 .Primary ovarian hyper function in females
2. Primary hypergonadism in males

NOTE

1 .FSH and LH are both decreased in failure of the pituitary or hypothalamus.

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Test Name	Value	Unit	Biological Reference interval

FOLLICLE STIMULATING HORMONE (FSH)

FOLLICLE STIMULATING HORMONE (FSH): SERUM *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	3.68	mIU/mL	FEMALE FOLLICULAR PHASE: 2.5 - 11.4 FEMALE MID-CYCLE PEAK: 3.3 - 21.7 FEAMLE LUTEAL PHASE: 1.2 - 7.0 FEMALE POST-MENOPAUSAL: 18.8 - 132 MALE: 1.0 - 12.1

INTERPRETATION:

1. Gonadotropin-releasing hormone from the hypothalamus controls the secretion of the gonadotropins, follicle-stimulating hormone (FSH) and luteinizing hormone (LH) from the anterior pituitary.
2. The menstrual cycle is divided by a midcycle surge of both FSH and LH into a follicular phase and a luteal phase.
3. FSH appears to control gametogenesis in both males and females.

The test is useful in the following settings:

1. An adjunct in the evaluation of menstrual irregularities.
2. Evaluating patients with suspected hypogonadism.
3. Predicting ovulation
4. Evaluating infertility
5. Diagnosing pituitary disorders
6. In both males and females, primary hypogonadism results in an elevation of basal follicle-stimulating hormone (FSH) and luteinizing hormone (LH) levels.

FSH and LH LEVELS ELEVATED IN:

1. Primary gonadal failure
2. Complete testicular feminization syndrome.
3. Precocious puberty (either idiopathic or secondary to a central nervous system lesion)
4. Menopause (postmenopausal FSH levels are generally >40 IU/L)
5. Primary ovarian hypofunction in females
6. Primary hypogonadism in males

NOTE:

1. Normal or decreased FSH is seen in polycystic ovarian disease in females
2. FSH and LH are both decreased in failure of the pituitary or hypothalamus.

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Test Name	Value	Unit	Biological Reference interval

PROLACTIN

PROLACTIN: SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	**44.8^{H}**	ng/mL	3 - 25

INTERPRETATION:

1.Prolactin is secreted by the anterior pituitary gland and controlled by the hypothalamus.
2.The major chemical controlling prolactin secretion is dopamine, which inhibits prolactin secretion from the pituitary.
3.Physiological function of prolactin is the stimulation of milk production. In normal individuals, the prolactin level rises in response to physiologic stimuli such as sleep, exercise, nipple stimulation, sexual intercourse, hypoglycemia, postpartum period, and also is elevated in the newborn infant.

INCREASED (HYPERPROLACTEMIA):

1.Prolactin-secreting pituitary adenoma (prolactinoma, which is 5 times more frequent in females than males).
2.Functional and organic disease of the hypothalamus.
3.Primary hypothyroidism.
4.Section compression of the pituitary stalk.
5.Chest wall lesions and renal failure.
6.Ectopic tumors.
7.DRUGS:- Anti-Dopaminergic drugs like antipsychotic drugs, antinausea/antiemetic drugs, Drugs that affect CNS serotonin metabolism, serotonin receptors, or serotonin reuptake (anti-depressants of all classes, ergot derivatives, some illegal drugs such as cannabis), Antihypertensive drugs ,Opiates, High doses of estrogen or progesterone,anticonvulsants (valporic acid), anti-tuberculous medications (Isoniazid).

SIGNIFICANCE:

1.In loss of libido, galactorrhea, oligomHyperprolactinemia often results enorrhea or amenorrhea, and infertility in premenopausal females.
2.Loss of libido, impotence, infertility, and hypogonadism in males. Postmenopausal and premenopausal women, as well as men, can also suffer from decreased muscle mass and osteoporosis.
3. In males, prolactin levels >13 ng/mL are indicative of hyperprolactinemia.
4. In women, prolactin levels >27 ng/mL in the absence of pregnancy and postpartum lactation are indicative of hyperprolactinemia.
5.Clear symptoms and signs of hyperprolactinemia are often absent in patients with serum prolactin levels <100 ng/mL.
4. Mild to moderately increased levels of serum prolactin are not a reliable guide for determining whether a prolactin-producing pituitary adenoma is present, 5.Whereas levels >250 ng/mL are usually associated with a prolactin-secreting tumor.

CAUTION:

Prolactin values that exceed the reference values may be due to macroprolactin (prolactin bound to immunoglobulin). Macroprolactin should be evaluated if signs and symptoms of hyperprolactinemia are absent, or pituitary imaging studies are not informative.

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Test Name	Value	Unit	Biological Reference interval

ESTRADIOL (E2)

ESTRADIOL (E2): SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	38.998	pg/mL	FEMALE FOLLICULAR PHASE: 19.5 - 144.2 FEMALE MID CYCLE PHASE: 63.9 - 356.7 FEMALE PRE OVULATORY PHASE: 136.0 - 251.0 FEMALE LUTEAL PHASE: 55.8 - 214.2 POST MENOPAUSAL:< 50.0

INTEPRETATION:

OTHER MATERNAL FACTORS AND PREGNANCY	UNITS	RANGE
Hormonal Contraceptives	pg/mL	15.0 – 95.0
1st Trimester (0 – 12 Weeks)	pg/mL	38.0 – 3175.0
2nd Trimester (13 – 28 Weeks)	pg/mL	678.0 – 16633.0
3rd Trimester (29 – 40 Weeks)	pg/mL	43.0 – 33781.0
Post Menopausal	Pg/mL	< 50.0
MALES:	pg/mL	< 40.0

1. Estrogens are involved in development and maintenance of the female phenotype,germ cell maturation,and pregnancy.They also are important for many other,nongender-specific processes, including growth,nervous system maturation,bone metabolism/remodeling,and endothelial responsiveness.
2. E2 is produced primarily in ovaries and testes by aromatization of testosterone.
3. Small amounts are produced in the adrenal glands and some peripheral tissues,most notably fat.E2 levels in premenopausal women fluctuate during the menstrual cycle.
4. They are lowest during the early follicular phase. E2 levels then rise gradually until 2 to 3 days before ovulation, at which stage they start to increase much more rapidly and peak just before the ovulation-inducing luteinizing hormone (LH)/follicle stimulating hormone (FSH) surge at 5 to 10 times the early follicular levels.This is followed by a modest decline during the ovulatory phase.E2 levels then increase again gradually until the midpoint of the luteal phase and thereafter decline to trough,early follicular levels.

INDICATIONS FOR ASSAY: -

1. Evaluation of hypogonadism and oligo-amenorrhea in females.
2. Assessing ovarian status, including follicle development,for assisted reproduction protocols (eg, in vitro fertilization)
3. In conjunction with lutenizing hormone measurements,monitoring of estrogen replacement therapy in hypogonadal premenopausal women
4. Evaluation of feminization,including gynecomastia,in males.
5. Diagnosis of estrogen-producing neoplasms in males,and,to a lesser degree,females
6. As part of the diagnosis and work-up of precocious and delayed puberty in females,and,to a lesser degree, males
7. As part of the diagnosis and work-up of suspected disorders of sex steroid metabolism,eg:aromatase deficiency and 17 alpha-hydroxylase

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Test Name	Value	Unit	Biological Reference interval

deficiency
8. As an adjunct to clinical assessment,imaging studies and bone mineral density measurement in the fracture risk assessment of postmenopausal women,and,to a lesser degree,older men
9. Monitoring low-dose female hormone replacement therapy in post-menopausal women
10. Monitoring antiestrogen therapy (eg, aromatase inhibitor therapy).

CAUSES FOR INCREASED E2 LEVELS:

1. High androgen levels caused by tumors or androgen therapy (medical or sport performance enhancing),with secondary elevations in E1 and E2 due to aromatization
2. Obesity with increased tissue production of E1
3. Decreased E1 and E2 clearance in liver disease
4. Estrogen producing tumors
5. Estrogen Ingestion

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Test Name	Value	Unit	Biological Reference interval

INSULIN FASTING (F)

INSULIN FASTING (F)	6.57	µIU/ml	2.0 - 25.0

by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)

INTERPRETATION:-

1.Insulin is a hormone produced by the beta cells of the pancreas. It regulates the uptake and utilization of glucose and is also involved in protein synthesis and triglyceride storage.
2.Type 1 diabets (insulin-dependent diabetes) is caused by insulin deficiency due to destruction of insulin producing pancreatic islets (beta) cells.
3.Type 2 diabetes (noninsulin dependent diabetes) is characterized by resistance to the action of insulin (insulin resistance).
4.The test is useful for management of diabetes mellitus and for diagnoses of insulinomas, when used in conjunction with proinsulin and C-peptide measurements.

NOTE:

1.No standard referance range has yet been established for INSULIN POST-PRANDIAL (PP) in indian population, therefore same could not be provided along with test. However various studies done on several populations mention that the range of INSULIN PP can vary somewhere from 5-79 mIU/L which can be used for clinical purpose.

2.This assay has 100% cross-reactivity with recombinant human insulin (Novolin R and Novolin N). It does not recognize other commonly used analogues of injectable insulin (ie, insulin lispro, insulin aspart, and insulin glargine).

INTERPRETATIVE GUIDE:

1.During prolonged fasting, when the patient's glucose level is reduced to <40 mg/dL, elevated insulin level plus elevated levels of proinsulin and C-peptide suggest insulinomaS.
2.Insulin levels generally decline in patients with type 1 diabetes mellitus.
3.In the early stage of type 2 diabetes, insulin levels are either normal or elevated. In the late stage of type 2 diabetes, insulin levels decline.
4.In normal individuals, insulin levels parallel blood glucose levels.
5.Patients on insulin therapy may develop anti-insulin antibodies. These antibodies may interfere in the assay system, causing inaccurate results. In such individuals, measurement of free insulin FINS / Insulin, Free, Serum should be performed.

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Test Name	Value	Unit	Biological Reference interval

TESTOSTERONE: TOTAL

TESTOSTERONE - TOTAL: SERUM	0.54	ng/mL	0.0 - 0.80

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

INTERPRETATION:

1.Testosterone is secreted in females by the ovary and formed indirectly from androstenedione in adrenal glands.
2.In males it is secreted by the testes. It circulates in blood bound largely to sex hormone binding globulin (SHBG). Less than 1% of the total testosterone is in the free form.
3.The bioavailable fraction includes the free form and that "weakly bound" to albumin (40% of the total in men and 20% of the total in women) and bound to cortisol binding globulin (CBG). It is the most potent circulating androgenic hormone.
4.The total testosterone bound to SHBG fluctuates since SHBG levels are affected by medication, disease, sex steroids and insulin.

CLINIC USE:

1.Assesment of testicular functions in males
2.Management of hirsutism and virilization in females

INCREASED LEVELS:

1.Precocious puberty (Males)
2.Androgen resistance
3.Testoxicosis
4.Congenital Adrenal Hyperplasia
5.Polycystic ovarian disease
7.Ovarian tumors

DECREASED LEVELS:

1.Delayed puberty (Males)
2.Gonadotropin deficiency
3.Testicular defects
4.Systemic diseases

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Test Name	Value	Unit	Biological Reference interval

DEHYDROEPIANDROSTERONE SULPHATE (DHEA-S)

DIHYDROEPIANDROSTERONE SULPHATE (DHEA-S) *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	349	µg/dL	8.2 - 592.9

INTERPRETATION:-

CLINICAL USE:

1. Marker for Adrenal cortical function and disease
2. Differential diagnosis of virilised patient. In patients with virilising tumours, DHEAS levels usually exceed 7000 g/dL

INCREASED LEVELS:

1. Adrenogenital syndromes due to deficiency of 3 beta-dehydrogenase, 21-hydroxylase and 11 beta-hydroxylase.
2. Congenital Adrenal Hyperplasia
3. Adrenal Carcinoma
4. Virilizing tumor of adrenal gland.
5. Cushing's disease, pituitary dependent.
6. Hirsutism
7. Polycystic ovarian Syndrome (PCOD)

DECREASED LEVELS:

1. Addison's disease
2. Adrenal Hypoplasia
3. Hyperlipidaemia
4. Psychoses
5. Psoriasis
6. Increasing age.

NOTE:

1. DHEA decreases in the elderly to a greater extent than do other steroids.

Rechecked

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VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	61.8	ng/mL	DEFICIENCY: < 20.0 INSUFFICIENCY: 20.0 - 30.0 SUFFICIENCY: 30.0 - 100.0 TOXICITY: > 100.0

INTERPRETATION:

DEFICIENT:	< 20	ng/mL
INSUFFICIENT:	21 - 29	ng/mL
PREFFERED RANGE:	30 - 100	ng/mL
INTOXICATION:	> 100	ng/mL

1.Vitamin D compounds are derived from dietary ergocalciferol (from plants, Vitamin D2), or cholecalciferol (from animals, Vitamin D3), or by conversion of 7- dihydrocholecalciferol to Vitamin D3 in the skin upon Ultraviolet exposure.
2.25-OH--Vitamin D represents the main body resevoir and transport form of Vitamin D and transport form of Vitamin D, being stored in adipose tissue and tightly bound by a transport protein while in circulation.
3.Vitamin D plays a primary role in the maintenance of calcium homeostatis. It promotes calcium absorption, renal calcium absorption and phosphate reabsorption, skeletal calcium deposition, calcium mobilization, mainly regulated by parathyroid harmone (PTH).
4.Severe deficiency may lead to failure to mineralize newly formed osteoid in bone, resulting in rickets in children and osteomalacia in adults.

DECREASED:
1.Lack of sunshine exposure.
2.Inadequate intake, malabsorption (celiac disease)
3.Depressed Hepatic Vitamin D 25- hydroxylase activity
4.Secondary to advanced Liver disease
5.Osteoporosis and Secondary Hyperparathroidism (Mild to Moderate deficiency)
6.Enzyme Inducing drugs: anti-epileptic drugs like phenytoin, phenobarbital and carbamazepine, that increases Vitamin D metabolism.

INCREASED:
1. Hypervitaminosis D is Rare, and is seen only after prolonged exposure to extremely high doses of Vitamin D. When it occurs, it can result in severe hypercalcemia and hyperphophatemia.

CAUTION: Replacement therapy in deficient individuals must be monitored by periodic assessment of Vitamin D levels in order to prevent hypervitaminosis D

NOTE:*-Dark coloured individuals as compare to whites, is at higher risk of developing Vitamin D deficiency due to excess of melanin pigment which interefere with Vitamin D absorption.*

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NAME	**: Mrs. SUKHMANI DUGGAL**		
AGE/ GENDER	: 31 YRS/FEMALE	**PATIENT ID**	: 1654689
COLLECTED BY	: SURJESH	**REG. NO./LAB NO.**	**: 012410270019**
REFERRED BY	: CENTRAL PHOENIX CLUB (AMBALA CANTT)	**REGISTRATION DATE**	: 27/Oct/2024 09:36 AM
BARCODE NO.	: 01519631	**COLLECTION DATE**	: 27/Oct/2024 09:51AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	**REPORTING DATE**	: 27/Oct/2024 12:55PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval

VITAMIN B12/COBALAMIN

VITAMIN B12/COBALAMIN: SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	**1007^{H}**	pg/mL	190.0 - 890.0

INTERPRETATION:-

INCREASED VITAMIN B12	DECREASED VITAMIN B12
1.Ingestion of Vitamin C	1.Pregnancy
2.Ingestion of Estrogen	2.DRUGS:Aspirin, Anti-convulsants, Colchicine
3.Ingestion of Vitamin A	3.Ethanol Igestion
4.Hepatocellular injury	4. Contraceptive Harmones
5.Myeloproliferative disorder	5.Haemodialysis
6.Uremia	6. Multiple Myeloma

1.Vitamin B12 (cobalamin) is necessary for hematopoiesis and normal neuronal function.
2.In humans, it is obtained only from animal proteins and requires intrinsic factor (IF) for absorption.
3.The body uses its vitamin B12 stores very economically, reabsorbing vitamin B12 from the ileum and returning it to the liver; very little is excreted.
4.Vitamin B12 deficiency may be due to lack of IF secretion by gastric mucosa (eg, gastrectomy, gastric atrophy) or intestinal malabsorption (eg, ileal resection, small intestinal diseases).
5.Vitamin B12 deficiency frequently causes macrocytic anemia, glossitis, peripheral neuropathy, weakness, hyperreflexia, ataxia, loss of proprioception, poor coordination, and affective behavioral changes. These manifestations may occur in any combination; many patients have the neurologic defects without macrocytic anemia.
6.Serum methylmalonic acid and homocysteine levels are also elevated in vitamin B12 deficiency states.
7.Follow-up testing for antibodies to intrinsic factor (IF) is recommended to identify this potential cause of vitamin B12 malabsorption.
NOTE:A normal serum concentration of vitamin B12 does not rule out tissue deficiency of vitamin B12. The most sensitive test for vitamin B12 deficiency at the cellular level is the assay for MMA. If clinical symptoms suggest deficiency, measurement of MMA and homocysteine should be considered, even if serum vitamin B12 concentrations are normal.

*** End Of Report ***

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