descending order were: 1) BP (Szymon), 2) BP Watch, 3) BP Log My Diary, 4) BP Diary (openit.inc), 5) BP Log (bpresso.com), 6) BP Diary (FRUCT), 7) BloodPressureDB, 8) Cardio Journal, 9) BP (OrangeKit), 10) BP Diary (Health&Fitness Tracker). We found 10/10 BP logs; 2/10 ACC BP guidelines; 3/10 HbA1c/FBS; 1/10 lipid log; 0/10 diet, Na^+ intake, K^+ intake; 2/10 exercise log; 8/10 push notification for medication adherence. **Discussion**: eHealth apps emphasize the use of patient generated health data to manage chronic disease and cost containment. This small study shows that for the most popular, free diabetes and hypertension apps, few follow all the guidelines from the ADA or ACC/AHA.
Conclusion: The Diabetes apps follow the DSMES guidelines more than the Hypertension apps follow the ACC/AHA guidelines. In the future, there is room for improvement for eHealth apps and the management of diabetes and comorbid conditions such as hypertension.

Adrenal

ADRENAL CASE REPORTS I

An Unusual Case Of Intra-adrenal Purely Norepinephrine Secreting Pheochromocytoma

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SAT-188

Background: In 40% of pheochromocytoma/paraganglioma (PPGL) cases a causal germline mutation in a well-defined gene can be identified. The remainder are sporadic. The most common hereditary syndromes are NF1, MEN2 and VHL. Paragangliomas usually produce exclusively norepinephrine (NE) and are more likely to metastasize than adrenal tumors. Exclusively NE producing adrenal tumors are extremely rare and almost always associated with VHL or SDH syndromes (1).
Clinical Case: A 45-year-old South Asian woman with a 5-year history of HTN controlled on losartan presented emergently complaining of chest pressure. Cardiovascular workup was unrevealing. CT chest showed an incidental 3.2 x 2.4 cm lipid-poor left adrenal adenoma. No further follow-up done at that time. Two years later she presented with recurring episodes of chest pressure and uncontrolled HTN on amlodipine, metoprolol and losartan. She denied panic attacks, diaphoresis and other symptoms of anxiety. She denied personal and familial history of clinical features seen in NF1, MEN 2, VHL or SDH. Plasma metanephrines were 26 (<=57 pg/mL) and free normetanephrine 902 (<=148 pg/mL). 24-hour urine metanephrine was 178 (58-203 mcg/24h), normetanephrine 2422 (88-649 mcg/24h) and total metanephrine 2600 (182-739 mcg/24h) confirming the diagnosis of a solely NE secreting PPGL. MRI abdomen showed a well-circumscribed 3.3 cm lipid-poor left adrenal mass. MIBG scan and SPECT CT showed a focal area of intense radiotracer uptake corresponding to a 3.2 x 2.5 cm mass within the left adrenal gland. No extra-adrenal activity was demonstrated. Alpha blockade was started with doxazosin and the patient asked to salt/fluid load. She underwent left adrenalectomy. Pathology revealed a 5 x 2.5 x1.1 cm intra-adrenal pheochromocytoma; Chromogranin (+), synaptophysin (-), MIB1 low reactivity < 5%, S100 positive in sustentacular cells, keratin (-), EMA (-), Inhibin (-). Genetic testing for VHL, SDHD, SDHB, SDHC and MAX has been ordered per guidelines. One month postoperatively the patient had no symptoms of adrenergic overactivity and normal plasma metanephrine levels.
Clinical Lessons: This rare case of norepinephrine-only secreting pheochromocytoma is made even more unusual by absence of features of autosomal dominant hereditary syndromes. This may be sporadic or present a novel VHL, SDHD, SDHB, SDHC or MAX germline mutation (2).

1.
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Cardiovascular Endocrinology

HYPERTRIGLYCERIDEMIA; INFLAMMATION AND MUSCLE METABOLISM IN OBESITY AND WEIGHT LOSS II

The Effect of Energy Deprivation on Metabolic Hormone Responses to Meals

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SUN-542

Background: Intermittent caloric restriction (ICR) has recently gained popularity as a weight-loss strategy; however, fasting metabolic hormones and dynamic meal-related responses, are not well-established in this setting.
Methods: We measured metabolic hormone responses to 5-days of neutral or decreased energy availability (NEA, 45 kcal/kg LBM*d vs DEA, 20 kcal/kg LBM*d) in the early follicular phase (EFP) in 19 regularly-cycling, sedentary, women (age 23.36± 2.08 yr; mean±SD). Hunger was assessed using a visual analogue scale on the 5th day of each condition. Scheduled breakfast and lunch were administered according to assigned caloric intake, while an afternoon snack based on NEA was provided on both occasions. Blood was sampled for leptin, insulin, glucose, and GH at 10-min intervals and cortisol was measured at 30-min intervals over eight hours starting at 0800 h, while Orexin A and adiponectin were measured in fasting

samples. AUC for each hormone for every meal and diet condition were analyzed using linear mixed models. Insulin and insulin/glucose ratio (I/G) were also adjusted for meal calories. Percentage body fat mass was measured every visit using air displacement plethysmography (BodPod®). Results are presented as least square mean ± sem. Results: There were no differences in body mass index or % fat mass after NEA vs DEA although there was a significant increase in hunger with DEA (p=0.002). Fasting levels of glucose and insulin were unchanged while leptin decreased with DEA (1.27±0.07 and 1.04±0.07 ng/mL, NEA and DEA respectively; p<0.0001), and Orexin A increased (0.55±0.04 and 0.60±0.04 ng/mL; p=0.04). The AUC for glucose was lower with DEA across all meals (p<0.0001). Insulin, I/G and I/G normalized for ingested calories (nI/G) decreased in response to DEA (p<0.005, p<0.05 and p<0.0001). The slope of the increase in leptin across the day was not different between NEA and DEA (p=0.20). Adiponectin, GH and cortisol were unaffected by DEA. Conclusion: These studies indicate that although fasting glucose and insulin are unaffected by short-term caloric restriction, the insulin response to glucose is attenuated even when adjusting for meal-related calories. Orexin A increased and leptin decreased with reduced caloric intake, acting, at least in part, to stimulate appetite. Taken together, these hormonal responses, directed at preserving energy homeostasis, have important implications for understanding the potential efficacy of intermittent caloric restriction.

Adrenal

ADRENAL CASE REPORTS I

Metastatic Pheochromocytoma in MEN2A: Clinical Features, Laboratory Data and Radiological Findings of a Rare Association - Case Report

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SAT-223

Background: Multiple endocrine neoplasia type 2A (MEN2A) is an autosomal dominant syndrome caused by inactivating mutations in the RET proto-oncogene. It is characterized by medullary thyroid carcinoma (MTC), pheochromocytoma (PHEO) and hyperparathyroidism (HPTH). MTC is one of the initial manifestations in 90% of patients. PHEO affects approximately 50% of patients, is almost always benign (98% of cases), usually bilateral and confined to the adrenal glands. HPTH occurs in 20-30% of patients. The clinical presentation, evolution and prognosis of metastatic PHEO associated with *RET* mutations are not yet well known.

Clinical Case: A previously healthy man was initially diagnosed with hypertension at 24 years of age. Two years later, after recurrent paroxysms of headache, tremors and tachycardia, the patient was suspected of having bilateral PHEO based on laboratory and radiological findings. Bilateral adrenalectomy was performed and the anatomopathological analysis confirmed the suspected diagnosis.

Soon afterward, although the patient was asymptomatic, with urinary metanephrines in the normal range, two possibly metastatic lesions were identified – one in the liver (9 x 8 mm) and one in the left adrenal bed. Some of the patient's family members were also found to have PHEO and/or MTC, leading to the diagnosis of MEN2A. A *RET* germline mutation in codon 634 (p.Cys634Phe) of exon 11 was then found in the patient's family pedigree. At the time, the patient (index case) had no evidence of MTC or HPTH. Diagnostic ^{131}I-MIBG scintigraphy showed increased uptake in the patient's liver. The subsequent percutaneous liver biopsy confirmed the presence of metastatic PHEO. Interestingly, no significant ^{18}F-FDG uptake was found on the ^{18}F-FDG-PET/CT scan in the metastatic PHEO sites. For more than 10 years of follow-up with no specific treatment, the metastatic lesions demonstrated slow growth rates; metanephrine levels, although increased (total = 1422 mcg/24h, NR <1000; normetanephrine = 676 mcg/24h, NR <320; and metanephrine = 574 mcg/24h, NR <390), were relatively stable; blood pressure and adrenergic symptoms were under control with a few antihypertensive medications.

At 36 years of age, the calcitonin level was slightly increased (8.6 pg/mL, NR <8.4) and a minuscule thyroid nodule (3 x 3 x 2 mm) was identified on the ultrasound scan. Prophylactic thyroidectomy was performed, with a diagnosis of a 2.5-mm MTC. More recently, an increase in the metanephrine levels was found and treatment with iobenguane ^{131}I may be an option.

Conclusion: Patients with metastatic PHEO caused by mutations in the *RET* proto-oncogene (MEN2A) may have a long survival time. In such patients, an ^{18}F-FDG-PET/CT scan may exhibit low sensitivity for the diagnosis of metastasis and the onset of PHEO may precede that of MTC by many years.

Bone and Mineral Metabolism

BONE DISEASE FROM BENCH TO BEDSIDE

Burosumab Improves Bone Density in Patients with X-Linked Hypophosphatemia

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SUN-333

Burosumab Improves Bone Density in Patients with X-Linked Hypophosphatemia

Background: X-linked hypophosphatemia (XLH) causes rickets in children and osteomalacia in adults due to lifelong renal phosphate wasting that is mediated by high circulating levels of FGF-23. Burosumab, is a recently approved fully human monoclonal antibody that blocks