patients without night shifts was 2.4% within 24 hours (min 0-max 6.6).

Correlation analysis between carbohydrate metabolism and glucose variability revealed a significant strong relationship between the level of postprandial glycemia and mo d (r=0.87, P=0.001) and MAGE (r=0.82, P=0.01), and also established a mean significant correlation between the level of postprandial glucose and Conga (r=0.52, P=0.01) and SD (r=0.61, P=0.05).

Fasting glycemia and congass were moderately correlated (r=0.4, P=0.01).

Weak reliable correlation was found only between HbA1c level and Conga variability index (r=0.27, P=0.04).

Conclusions: The results of the study indicate the lack of adequate glycemic control in persons working night shifts, high variability of glycemia, which is an independent risk factor for cardiovascular disease in patients with DM2. The associative relationship of fasting glycemia and postprandial with the indicator of variability Conga shows that glucose fluctuations during the day in patients are constant.

Neuroendocrinology and Pituitary
CASE REPORTS IN NEUROENDOCRINOLOGY BEYOND THE PITUITARY
Tachycardia and Myocardial Injury Induced Natriuresis - a Rare Clinical Encounter
Bilal Bashir, MRCP (UK), Ambar Basu, FRCP, John-Paul Lomas, FRCA, Steven Little, MRCP (UK), Moulinath Banerjee, FRCP, MD, PhD.
Royal Bolton Hospital, Bolton, United Kingdom.

MON-248
Introduction: Hyponatremia in the context of cerebral salt wasting secondary to intracranial events has well been described due to increased release of natriuretic peptides. We describe a case of natriuresis leading to acute symptomatic hyponatremia associated with tachyarrhythmia and myocardial infarction.

Case: A 72-year-old lady was admitted with atrial fibrillation with rapid ventricular rate between 130-150 beats per minute, troponin positive chest pain, with high sensitivity troponin T of 1307 ng/L (<14 ng/L) and managed as type II myocardial infarction. 12 hours after admission she became acutely confused, agitated, associated with visual hallucinations and myoclonic jerks. On Examination she was dehydrated and there were no focal neurological features. Investigations showed acute hyponatremia with serum sodium (Na⁺) of 117 mmol/L (135 - 145 mmol/L) which 12 hours earlier, on admission, was 137 mmol/L, serum osmolarity 249 mosm/kg (275 - 295 mosm/kg), urine osmolarity 486 mosm/kg, urinary sodium of 160 mmol/L, pro-NTBNP 6575pg/ml (<450 pg/ml), 9am serum cortisol 575 nmol/L (140 - 690 nmol/L), TSH 4.72 mu/L (0.2 - 5.0 mu/L), FT4: 18.6pmol/L (10.0 - 24.0 pmol/L) and normal chest x-ray. She was treated with hypertonic saline (2.7%) 200ml followed by 0.9% saline infusion that corrected chest x-ray. She was treated with hypertonic saline (2.7%) and normal serum sodium levels to 136 mmol/L over next 48-72 hours along with clinical symptoms gradually went back to normal. Repeat pro-NTBNP after recovery was 3153 pg/ml. Echocardiography showed normal left ventricular systolic function with normal atrial size and moderate mitral regurgitation with pulmonary artery pressure of 30mmHg. Based on acute onset hyponatremia and raised urinary sodium, we proposed diagnosis of hyponatremia secondary to salt wasting and we believe that in absence of acute intracranial pathology and raised pro-NTBNP, renal salt wasting was induced by acute rise in natriuretic peptides of cardiac origin either secondary to myocardial infarction or tachyarrhythmia. Paroxysmal SVT is a known cause of transient polyuria after termination but is not known to cause hyponatremia. Although well described in context of intracranial events, this is the first case of myocardial injury or tachyarrhythmia induced natriuresis leading to acute hyponatremia.

Conclusion:
Salt wasting due to natriuretic peptides is not exclusive to intracranial events. Any cardiac event leading to sudden increase in natriuretic peptides can lead to natriuresis which if prolonged enough can lead to acute symptomatic hyponatremia.

Reference:
1. Yee AH, Burns JD, Wijdicks EF. Cerebral Salt Wasting: Pathophysiology, Diagnosis, and Treatment. Neurosurgery Clinics of North America, 21(2), pp.339-352.
2. Tan SY, Nolan J, Craig K, Swainson CP. Supraventricular tachycardia, right atrial pressure, atrial natriuretic peptide and polyuria—a necessary sequence? J Int Med 1993; 233: 415-17.

Thyroid
THYROID DISORDERS CASE REPORTS II
Myxiedema Madness: A Rare Case of Severe Hypothyroidism Presenting as Psychosis
Kevin Kohm, MD, Shivani Veharia, MD, Jack Xu, MD, Carol Nasr, MD, Lauren Hogshire, MD.
Rutgers RWJ Medical School, New Brunswick, NJ, USA.

SAT-480
Myxedema Madness: A Rare Case of Severe Hypothyroidism Presenting As Psychosis
Introduction
Myxedema coma is a rare, life-threatening medical emergency resulting from uncontrolled hypothyroidism. Myxedema coma refers to the neurological sequelae of severe hypothyroidism, which classically manifests as depressed mental status. Rarely, myxedema coma can present with a hyperactive mental state and psychosis. We present an unusual case of a drug overdose secondary to myxedema coma-induced psychosis.

Clinical Case
A 48 year old woman with a history of seizure disorder and hypothyroidism presented to the hospital after lamotrigine overdose. The patient’s spouse witnessed her ingest forty-five tablets of lamotrigine after an argument. The patient had no previous psychiatric diagnoses or suicide attempts. On examination, the patient was hemodynamically stable but was agitated, disoriented, and uncooperative. She had a normal neurologic exam and no peripheral edema. Her lamotrigine level was 25.4 ug/ml (2.5-15.0 ug/ml). The patient’s mental status did not improve with lamotrigine
cessation. Psychiatry determined that the patient’s psychosis was not consistent with lamotrigine overdose. Given these recommendations, alternative causes of psychosis were considered. The patient’s husband stated she had not taken levothyroxine for over one year. Thyroid function tests revealed a thyroid stimulating hormone (TSH) of 299 mcU/ml (0.35-5.50 mcU/ml) with a free thyroxine (T4) level of 0.27 ng/dl (0.89-1.76 ng/gl). The patient was started on levothyroxine intravenously. After five doses of intravenous levothyroxine, her mental status improved to baseline and she was transitioned to oral levothyroxine. She denied that the lamotrigine ingestion was a suicide attempt. Based on the patient’s presentation and clinical course, we concluded that her overdose was due to severe hypothyroidism leading to myxedema madness.

Conclusion
Severe hypothyroidism with myxedema coma often presents with depressed mental status, which can manifest as progressive confusion, lethargy, and eventually coma. However, in the case of our patient, severe hypothyroidism presented as psychosis, a rare manifestation. Remarkably, the patient had no other obvious physical manifestations of severe hypothyroidism. Psychosis, though rare, has been seen in cases typically after thyroidectomy or in patients with previously undiagnosed Hashimoto’s thyroiditis. In this patient’s case, it is likely that her myxedema madness was precipitated by long-term nonadherence with her thyroid replacement therapy, as the patient had no prior psychiatric history. Additionally, her rapid reversal of symptoms after the administration of levothyroxine supports the diagnosis of hypothyroid-induced myxedema madness.

Thyroid
BENIGN THYROID DISEASE AND HEALTH DISPARITIES IN THYROID II

Indiscriminate Thyroid Function Testing on Acute Hospital Admissions Reveals a High Abnormality Rate Requiring Follow Up
Robert P. McEvoy, MB, B Eng, PhD, Anthony O’Riordan, MB, Mark J. Hannon, MB, MSc, MD, FRCPA.
Bantry General Hospital, Cork, Ireland.

SUN-425
The population attending the Medical Assessment Unit at our hospital comprises patients attending electively for investigation and acutely unwell patients presenting for unscheduled care. The standard panel of blood tests taken on arrival includes thyroid function tests (TFTs, i.e. TSH and free-T4), despite a recent review questioning the clinical utility of this practice [1]. We performed a retrospective audit to determine what proportion of our patients had abnormal thyroid function on presentation, and whether these abnormal test results were being followed up. Using the iSoft Clinical Manager software, a list was generated of all patients who attended the hospital between January 2018 and June 2018 inclusive. For each attendance, we recorded the date, medical record number, patient age, gender, and TFT result. Abnormal TFT results were classified as overt or subclinical hyper- or hypothyroid, or non-thyroid illness syndrome (NTIS), based on their admission TSH and free-T4. We then examined the hospital and primary care records of patients with abnormal TFTs to determine if they had ongoing thyroid follow up post discharge.

In total, 2,298 patients attended over the 6-month study period. The mean patient age was 67.2 years, and 49% were female. Thyroid function tests were ordered on the day of attendance for 1,688 patients (73%). Of these, 181 results (11%) were abnormal: 20 overt hyperthyroid (11%), 72 subclinical hyperthyroid (40%), 12 overt hypothyroid (7%), 35 subclinical hypothyroid (19%), and 42 NTIS (23%). Twenty of these patients died within 3 months of the abnormal TFT result (4 overt hyperthyroid, 3 subclinical hyperthyroid, 3 overt hypothyroid, 6 subclinical hypothyroid, and 4 NTIS). Of the remaining 161 patients, 74 (46%) had not been followed up within 3 months (4 overt hyperthyroid, 34 subclinical hyperthyroid, 3 overt hypothyroid, 15 subclinical hypothyroid, and 18 NTIS).

The low percentage of abnormal TFTs (11%) in this audit is in keeping with similar studies where thyroid function testing was performed on unselected hospital populations [1]. Subclinical hyperthyroidism was by far the most common abnormality found. A high percentage of abnormal tests (46%) were not followed up, with poor compliance with thyroid management guidelines [2]. Future work will investigate adoption of an ‘opt-in’ order system [3] and electronic alerts to flag abnormal results for follow-up.

[1] Premawardhana LD. Thyroid testing in acutely ill patients may be an expensive distraction. Biochimia medica. 2017; 27(2): 300-307.
[2] Ross DS et al. 2016 American Thyroid Association Guidelines for diagnosis and management of hyperthyroidism and other causes of thyrotoxicosis. Thyroid. 2016 Oct; 26(10):1343-1421.
[3] Leis B et al. Altering standard admission order sets to promote clinical laboratory stewardship: a cohort quality improvement study. BMJ Qual Saf. 2019; 28(10): 846-52.

Bone and Mineral Metabolism
BONE AND MINERAL CASE REPORTS I

A Tale of Two Mutations: Familial Hypocalciuric Hypercalcemia Caused by a Novel CaSR Start Codon Mutation Found in the Setting of a CaSR Hypercalcemic Variant
Sindhura Bandaru, MD, Elaine Michelle Pelley, MD.
University of Wisconsin Hospital and Clinics, Madison, WI, USA.

SAT-350
Background: The calcium-sensing receptor (CaSR) mediatesPTH production and renal calcium excretion by sensing circulating calcium levels. Activating mutations in the CaSR can cause a spectrum of phenotypes from overt hypoparathyroidism to isolated hypercalcuria. Inactivating mutations of the CaSR lead to the syndrome of familial hypocalciuric hypercalcemia (FHH) where the protein produced is less sensitive to calcium. A mutation in the start codon of the CaSR leading to FHH has not previously been reported. Case: 60-year-old female was seen for evaluation of osteoporosis with lowest T-score of -2.9 at spine. She had no family history of calcium disorders. Biochemical evaluation for secondary etiologies of bone loss