ELECTIVE (SSC5c) REPORT (1200 words)

A report that addresses the above four objectives should be written below. Your Elective supervisor will assess this.

Much like how the pattern of diseases presenting to primary care differ from those that are referred on to secondary care, the patients I saw during my time in a tertiary centre were vastly different to what you might expect to find in a secondary centre. A general practitioner in primary care may very well deal with patients with stable diabetes mellitus requiring adjustments in their medication or insulin doses, or perhaps patients with an under-active thyroid requiring a small bump to their thyroxine dose. In secondary care, patients are often acutely unwell, perhaps having suffered long term complications of their diabetes mellitus such as infection of a previously unnoticed diabetic ulcer, or perhaps the patient is a young adult with type one diabetes mellitus having tipped over into diabetic ketoacidosis. These cases are more dire and certainly not uncommon in the secondary care setting. In fact, the majority of endocrine patients in the general population are technically under endocrine care primarily because of their diabetes mellitus. Here in a tertiary care centre, however, six weeks have gone by and whilst a fair few patients did coincidentally have diabetes mellitus, none of them were admitted as a direct consequence of it. Rather, these were patients with diseases some general practitioners may very well not come across in their entire careers; diseases my peers read with interest in textbooks but never manage to solidify their learning by seeing a patient. Over the past weeks I have come across multiple patients with phaeochromocytoma, acromegaly, and Cushing's disease, to name a few. A moderately-sized general practice may take care of several thousand patients, which sounds like a lot but considering how each of the above named diseases have a prevalence of roughly two to six cases per million people, it is not surprising that some general practitioners never come across them. Even towards the end of my placement I still am enthralled by the fact that these cases are almost one in a million. Aside from the rarity of these diseases, patients presenting to tertiary centres are more often than not highly complex in terms of their co-morbidities and in their presentations themselves; providing care for these patients will always be a challenge.

Endocrinological diseases in other first world countries such as Canada and the United States have a very similar pattern to those presenting here in the United Kingdom. The health care systems, however, differs drastically. This could have been further explored had I planned my elective abroad to experience their health care systems first hand.

Cushing's syndrome is by definition simply the clinical state resulting from an excess of circulating glucocorticoid. There is also loss of diurnal rhythm with regards to glucocorticoid secretion as well as loss of the normal inhibitory mechanism despite high levels of glucocorticoid in the body. The diagnosis of Cushing's syndrome occurs over several steps which should be done in a strict order: clinical assessment, biochemical tests followed by radiographic investigation. Imaging is only warranted if there is a clinical suspicion of Cushing's syndrome followed by biochemical evidence. That is to say, the patient must

present with signs and symptoms sufficient to convince the doctor of Cushing's syndrome. Amongst the more common signs and symptoms of Cushing's syndrome are: weight gain with central adiposity, mood change, proximal weakness, a change in fat distribution (especially to the face and upper back), bruising, thinning of the skin, recurrent infections and poor wound healing. Once these signs and symptoms have been established, the diagnosis should be confirmed by measuring serum cortisol. A raised serum cortisol is most easily demonstrated by measuring the level of cortisol at the time when, in normality, it should be at its lowest: midnight cortisol. Urinary cortisol levels measured over twenty-four hours is an alternative. Cortisol levels above the normal upper limit suggest Cushing's syndrome. Following this, a low-dose dexamethasone suppression test done over forty-eight hours could be done and should show a failure to fully suppress cortisol. Moreover, a cortisol day curve may show a lack of diurnal rhythm, where in normality cortisol is lowest at midnight and highest early in the morning. If the above tests point towards Cushing's syndrome, localisation tests can then be performed to find the lesion. At this stage it is important not to jump in with imaging as non-functioning 'incidentolomas' are found in roughly five percent of adrenal CTs and up to ten percent of pituitary MRIs. The causes of Cushing's syndrome are commonly divided into ACTH-dependent and ACTH-independent; ACTH-dependent causes include Cushing's disease and ectopic ACTH production from, for example, small cell lung cancer and carcinoid tumours, while ACTH-independent causes include iatrogenic, adrenal hyperplasia, adrenal adenoma and adrenal cortical carcinoma. Measuring ACTH levels is therefore a reasonable step towards localisation, since in the former group ACTH would be high, and in the latter group ACTH would be almost, if not entirely, undetectable. With detectable ACTH, to further narrow down the cause, CRH may be given IV and cortisol measured in two hours time; cortisol will tend to rise in pituitary disease but not with an ectopic cause as, although diseased, the pituitary will at least partially respond to manipulation. Should a pituitary cause be suspected at this stage, imaging with a pituitary-dedicated MRI is warranted along with inferior petrosal sinus sampling to confirm a central-peripheral ACTH gradient, and perhaps localise the diseased side, albeit with a low accuracy. If an ectopic cause is suspected, CT CAP with contrast may be performed to hunt for a potentially small ACTH-secreting tumour. Moving back a step, should ACTH be undetectable to begin with, an adrenal cause is most likely and an abdominal CT with contrast would be justified. Adrenal vein sampling is an option in the event that CT fails to reveal a mass.

With regards to treatment, options obviously depend on the cause of disease. In those who are fit, surgery is usually the ideal, potentially curative option. Pituitary causes may be operated on transphenoidally, although recurrence is not uncommon in those who only had half their pituitary removed. Radiotherapy may follow as an adjunct therapy. Ectopic causes should be surgically removed if possible or appropriate. Adrenalectomy may be performed on those with an adrenal cause. Bilateral adrenalectomy followed by medical replacement of hydrocortisone and fludrocortisone remains an option in those whose cause of disease is unidentifiable. Medically, adrenolytic drugs such as metyrapone and ketoconazole may be used following surgery or on their own if the patient is unfit for surgery.

Previous to this elective placement I had in fact been attached to the same team for my student-selected component earlier on this academic year. I had enjoyed my time with the team so much that I decided to return for my elective, and this time my placement was just as educational and fulfilling as my last. The best way to learn about any specialty, I feel, is to

fully engage and be a real part of the team, which I felt able to do under the patient guidance of everyone on Garrod ward. Overall I feel this elective has given me the confirmation I needed with regards to aiming towards endocrinology as a career. Although, as established earlier, the difference between tertiary and secondary care centres is great and I ought to experience secondary care more, which I will be able to do in my second year of foundation training; endocrinology at Colchester is easily the rotation I look forward to the most.