Three menstruating females, mean age 44, were diagnosed as having severe atrophic gastritis after gastroscopic study within the last 5 years (Medical records of Internal Medicine, Ramón y Cajal Hospital, Madrid). Physical examination revealed alopecia universalis, multinodular goitre and vitiligo, respectively, as positive findings. Patients denied menometrorrhagia. Laboratory findings showed hypochromic microcytic anaemia, low serum iron levels, low serum B₁₂ in one patient with an abnormal Schilling test, and positive serum antibodies to parietal cells. Gastric function tests yielded high serum gastrin levels and absolute achlorhydria. Oral iron replacement, plus hydroxocobalamin in the patient with vitamin B₁₂ malabsorption, re-established normal haematological data.

We report three patients with autoimmune chronic gastritis, associated in one patient with intrinsic factor hyposecretion, iron deficiency anaemia being the initial manifestation. Hypochromic microcytic anaemia can be secondary to chronic malabsorption of iron due to achlorhydria, but triggered by physiological features such as menstruation, although pathology such as undetectable blood loss might also contribute. Atrophic gastritis might explain some cases of iron deficiency, specially in young women, for two reasons. First, they have higher incidence of autoimmune diseases, including antrofundal chronic gastritis, and second, the presence of physiological mechanisms causing iron loss (menstruation, pregnancy).

We conclude that measurement of serum gastrin levels in all patients with iron deficiency anaemia of an unknown origin is advisable. This is particularly so if they present with autoimmune features such as alopecia universalis, vitiligo and diabetes mellitus. This will lead to an earlier diagnosis of vitamin B₁₂ malabsorption, and periodic gastroscopic examinations to detect the development of gastric carcinoma.

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Retinal infarcts and haemorrhages due to scurvy

Sir,

The main clinical features of scurvy are a vascular purpura with ecchymoses, gingival hyperplasia with gum bleeding. Ocular manifestations are very rare; conjunctival haemorrhage has been reported but we have been able to find no reports of retinal disease. We have recently encountered a patient with scurvy who had evidence of retinopathy. He was a 48 year old white male who presented to casualty with extensive bruising of his legs. Examination revealed confluent ecchymoses and the

classical signs of scurvy with cork-screw hairs and perifollicular hyperkeratoses over the lower abdomen with some perifollicular haemorrhages. There was marked gingival hypertrophy with spontaneous haemorrhage from the gums around very rotten teeth.

He lived alone, cooked for himself and ate mainly cereals and bread and no vegetables. He had eaten one single orange for his Christmas lunch the year before. He drank 80 units of alcohol per week. He had first noticed bleeding from his gums when he had bitten into a piece of cheese some weeks prior to admission. He reported no visual symptoms but ophthalmoscopy revealed several flamed shaped haemorrhages close to the left disc and three areas of 'cotton wool spots'.

His haemoglobin was 7.5 g/dl with a normochromic, normocytic picture. White cell count, platelets and prothrombin times were normal but his serum folate concentration was low (1.4 µg/l). Ascorbic acid absorption test conducted twice after admission revealed a urine ascorbic acid of less than 0.01 mmol/l whilst receiving a ward diet. Following treatment with ward food and a course of ascorbic acid, he rapidly improved and 3 months later his fundi were normal and his haemoglobin concentration was 11.6 g/dl.

Ocular manifestations of scurvy are rare but in children retrobulbar and subarachnoid bleeding has been reported.¹ We know of no other reports of retinal haemorrhages. Whilst the anaemia may have been a contributory factor, his extensive bruising elsewhere strongly suggest that increased capillary fragility was the main cause of his retinopathy.

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Hyperparathyroidism and cerebral haemorrhage

Sir,

Hyperparathyroidism presents with a wide variety of clinical symptoms derived from the well-known manifestations of gastrointestinal, kidney and bone disease,¹ as well as neurological complications, including psychoneurological disorders, myopathy² and rarely, ischaemic stroke.³ We report the dramatic presentation with cerebral haematoma of an asymptomatic young adult with hyperparathyroidism. This 35 year old man had, on routine examination, mild arterial hypertension detected a year before admission. No treatment was given. One day before admission, the patient suffered an abrupt right hemispheric headache, left-sided paralysis, left homonymous hemianopsia and reduction of the level of consciousness. On admission, blood pressure was 150/110 mmHg. Computed tomography disclosed an

haematoma at the level of the right basal ganglia with collapse of the lateral ventricle. Serum calcium measurements ranged between 3.07–3.6 mmol/l. Serum phosphorus was 0.74 mmol/l. Parathyroid hormone concentration, determined by immunoassay, was over 2700 pg/ml. Urinary cAMP excretion was 0.9 nmol/l. Serum creatinine was 141 µmol/l. Urinalysis was normal. Abdominal ultrasound showed increased echogenicity in both kidneys. No phaeochromocytoma was detected. Intravenous urography was normal. Bone X-rays revealed subcortical resorption. Cerebral angiography performed 3 months after the acute episode was considered normal. A 3 × 1.5 × 1 cm parathyroid adenoma was excised from the lower left parathyroid gland. Metabolic anomalies reverted to normal. Propanolol 120 mg/day was instated to maintain blood pressure within normal limits but a severe residual hemiplegia persisted.

Young adults with non-traumatic intracerebral haemorrhage are a heterogeneous group. A cause can be established in most patients,⁴ but in our patient no arteriovenous malformation, ruptured saccular aneurysm or sympathomimetic drug abuse could be demonstrated. Arterial hypertension undoubtedly played a determinant role. Arterial hypertension was notable in 6 of the 343 cases of hyperparathyroidism studied by Cope.² The metabolic anomalies induced in vascular walls by excess parathyroid hormone or vitamin D⁵ can contribute to this grave complication of hyperparathyroidism.

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Partially reversible nerve deafness due to vincristine

Sir,
Sensory-motor peripheral neuropathy is a frequent complication of vincristine therapy.^{1–3} Although involvement of various cranial nerves has also been reported, for some reason the eighth cranial nerve is usually spared.^{4–6} Here

we report a case of bilateral acoustic neuropathy attributable to vincristine.

A 77 year old woman with 3 weeks' history of weight loss, pruritis and generalized lymphadenopathy, was admitted to the hospital with right lower lobe pneumonia. Lymph node biopsy confirmed the diagnosis of low grade malignancy non-Hodgkin's lymphoma (centrocytic centroblastic in type).

After she had recovered from pneumonia, a regime of cytotoxic therapy (vincristine, chlorambucil and prednisolone in the conventional doses) was commenced. A few days later she developed rhinorrhoea, dry cough, and impaired hearing in both ears. She was not able to hear whispered words; however, conversational voice was heard if it was loud. Examination showed congested throat and nasal passages with normal external auditory canals and drums. Hearing returned to normal as the upper respiratory tract infection resolved in a few days.

However, soon after the second course of chemotherapy was given, she became profoundly deaf in both ears. Conversational voice was not heard, no matter how loud it was. Auroscopic examination was normal. A tuning fork test (512 Hz) showed that although both air and bone conduction were considerably impaired, the former was less severely affected, indicating a sensorineural type of deafness. Vincristine was suspected as the cause of her hearing loss and it was withdrawn from the regime. Apart from sluggish ankle jerks she did not have other signs of vincristine toxicity. Hearing returned to near pre-treatment level in the ensuing few weeks.

The anti-tumour properties of vinca alkaloids probably stem from their capacity to bind cellular tubulin.⁷ This results in inhibition of microtubule formation, an essential step in cellular division, causing the arrest of the dividing cells at the metaphase. Neurotubules seem to be vulnerable to some of the actions of vinca alkaloids.^{7,8} Their disruption is thought to be responsible for impairment of the axoplasmic transport mechanism. Hence vinca alkaloids are thought to cause axonal rather than demyelinating type of neuropathy.^{9,10}

Soon after their introduction in 1963 these agents were reported to cause peripheral neuropathy.^{11,12} This has consistently manifested itself as impairment of the tendoachilles reflex.^{1,2} Casey *et al.* reported that all patients who received vincristine developed evidence of peripheral neuropathy clinically or on electrophysiological testing. Nevertheless the neuropathy was largely reversible if the dose of vincristine was reduced or it was discontinued.¹ Cranial neuropathy is far less frequent (around 10%), and involvement of the VIII nerve is extremely rare.⁴ There has been only one previous case report (to our knowledge) of vincristine-induced acoustic neuropathy.¹³ Interestingly, that case was not dissimilar from ours. The patient, like ours, was an elderly lady with non-Hodgkin's lymphoma. Deafness after the first course of chemotherapy was attributed partly to acute otitis media. However, the suspected diagnosis of vincristine neuropathy became clear after the second course was initiated. This supports the view that hyperaemia of the inner ear, due to the accompanying infection, has probably exposed the sensitive neuromechanisms to high doses of vincristine, and might have precipitated the neuropathy.

Awareness of this serious complication of vincristine requires close monitoring of the auditory function in the