investigating patients with inconsistent hypokalaemia or mild hypokalaemia taking thiazide diuretics, and in these circumstances the more reasonable approach is to attempt control with a regimen which includes a potassium retaining diuretic. Where either the raised blood pressure or the low serum potassium concentration cannot be adequately controlled more intensive investigation for primary aldosteronism in a special centre will be needed. With these guidelines the occasional patient with surgically correctable primary aldosteronism will be missed, but the cost of such an error in terms of patient morbidity or mortality will be extremely small.

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Prostaglandins and menstrual disorders

Prostaglandins have potent biological effects, but their instability and rapid metabolism make them short acting. They are produced but not stored by most living cells and act locally. Prostaglandins of the E and F groups cause myometrial contractions as well as having vasoactive properties. Prostacyclin is a potent vasodilator and inhibitor of platelet aggregation and may cause relaxation of the myometrium,[1] while thromboxanes cause vasoconstriction and platelet aggregation. Non-steroidal anti-inflammatory drugs such as aspirin, indomethacin, naproxen, mefenamic acid, and ibuprofen exert their action by partially blocking an enzyme, cyclo-oxygenase, which converts arachidonic acid to the precursors of the various prostaglandins.

Menstruation is a hormonally controlled process of endometrial necrosis, shedding, and bleeding followed by haemostasis. Both the endometrium and myometrium have a considerable capacity to synthesise prostaglandins, particularly during the luteal phase of the cycle,[2] when the presence of the cyclo-oxygenase enzyme in the endometrium may be shown histochemically.[3] Prostaglandin $F_{2\alpha}$, a vasoconstrictor, is produced in large amounts by the uterus and it may trigger the ischaemic necrosis of menstruation. Vasodilatory prostaglandins such as prostaglandin E_2 or prostacyclin are also produced by the uterus, and there is evidence that both are hormonally regulated.[4 5] Increased menstrual bleeding might be due either to a reduction in the synthesis of prostaglandin $F_{2\alpha}$ or to an increase in the production of vasodilatory prostaglandins from endometrium or myometrium. Cultures of endometrium from women having normal menstrual cycles produce more prostaglandin $F_{2\alpha}$ than prostaglandin E_2, whereas from women with menorrhagia endometrial culture produces more prostaglandin E_2 than prostaglandin $F_{2\alpha}$. Furthermore, endometrium from women with menorrhagia is more effective than the endometrium from women with normal menstrual loss in enhancing the production of prostacyclin by myometrium in vitro.[6]

Haemostasis in the uterus (as in the peripheral circulation) is dependent on platelet aggregation and deposition of fibrin. Menstrual blood has fewer platelets than peripheral blood, and their ability to aggregate and to synthesise prostaglandins in vitro is appreciably reduced.[7] Menstrual blood is also lacking in fibrinogen and contains reduced amounts of antiplasmin.[8]

In the absence of a pathological disorder of the uterus menorrhagia is likely to be due to a combination of increased vasodilatation and impaired haemostasis. Treatment with a combined oestrogen-progestogen preparation or with danazol will reduce menstrual loss, but with the disadvantages of metabolic side effects and cost respectively. Alternatively, fibrinolytic activity may be reduced in the uterus by drugs, but these have some effect on the peripheral blood as well. In view of the possible consequences of such treatment attempts have been made to reduce menstrual loss by controlling the synthesis of prostaglandin with non-steroidal anti-inflammatory drugs. These drugs are non-specific in their inhibition of the production of prostaglandin since they block the synthesis of endoperoxides by the cyclo-oxygenase enzyme rather than the synthesis of specific prostaglandins. As long ago as 1976 Anderson *et al*[9] reported a reduction of menstrual loss when a drug inhibiting prostaglandin synthesis was taken during the days of menstrual bleeding. Reduced menstrual loss (assessed subjectively) has been reported with treatment with mefenamic acid.[10] It is important to realise, however, that there seems to be no correlation between measured menstrual loss and the number of pads used. Subsequent double blind randomised placebo controlled crossover trials of mefenamic acid have shown a definite reduction in measured menstrual loss. The treatment has no effect in about one fifth of patients, and it seems impossible to predict who will respond and who will not.[11 12]

When treatment is given with non-steroidal anti-inflammatory drugs they should be taken only immediately before and during the days of heavy menstrual bleeding. Side effects may include allergies, nausea, vomiting, dyspepsia, or, rarely, peptic ulceration. With such limited treatment, however, side effects are uncommon, and spasmodic dysmenorrhoea will also be relieved should this be present. In our present state of knowledge this form of medical treatment is worth trying, particularly in women with proved heavy bleeding who are anxious to avoid hysterectomy. Improved treatment will come only from more knowledge of the biochemical control of menstruation and the subsequent develop-

ment and use of drugs that are more specific inhibitors of the synthesis of the appropriate prostaglandins.

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The migrant sensory neuritis of Wartenberg

Transitory and often painful sensory symptoms commonly occur in a variety of neurological disorders and they may also be precipitated by minimal trauma, as, for example, sleeping with a limb in an awkward position. Similar disturbances in sensation, sometimes in a "non-anatomical" distribution and without objective evidence of neurological dysfunction, may occur in patients with demyelinating disease. Sensory symptoms are often the first indication that the sufferer has demyelinating disease. The diagnosis may be confirmed subsequently by the development of the typical clinical features of the disorder together with the finding of abnormal visual evoked responses and the presence of consistent immunoglobulin (type IgG) abnormalities in the cerebrospinal fluid. It is, of course, important to recognise other causes of transient sensory phenomena apart from multiple sclerosis, and in view of this the recent review[1] of the often forgotten migrant sensory neuritis of Wartenberg is both appropriate and timely.

Wartenberg referred to this condition in several papers which were published between 1940 and 1950, and in his monograph on "neuritis"[2] he reanalysed his nine cases in detail. Unfortunately, the reports of these nine cases were anecdotal with no objective confirmation of abnormal function of the peripheral nerves except in the ninth patient, who developed Guillain-Barré syndrome three weeks after being seen by the author. Wartenberg went on to make the incorrect suggestion that abnormalities of the peripheral nervous system occur in multiple sclerosis and that a migrant sensory neuritis was a mild form of the disease characterised by a predilection for the superficial sensory nerves.

Matthews and Esiri have recently given a brief account of the clinical features in six patients with a disorder which should probably be termed Wartenberg's syndrome.[1] In each case pain and subsequent loss of sensation were induced by movement of a limb which stretched a cutaneous nerve—for example, kneeling while gardening. The results of studies of the conduction velocity of motor nerves in cases 1 and 2 were normal, but the amplitude of the action potential in the sensory nerves of the fingers was reduced. In case 6 no action potential was recordable in the sural nerve. Because of this, a biopsy specimen of the nerve was taken and several minor abnormalities were found, including occlusion of a perineural blood vessel. This gave rise to the suggestion that ischaemia may play a part in the pathogenesis of migrant sensory neuritis. Nevertheless, the pathological changes might have been induced by the minor trauma which occurs in everyday life in an otherwise normal person. The axons of peripheral nerves can stretch during normal movement of a limb,[3] and there is no suggestion that patients who develop migrant sensory neuritis are more likely to develop entrapment of the peripheral nerves or nerve palsies induced by pressure (these are sometimes familial). Thus the pathogenesis of Wartenberg's syndrome is still not understood.

As far as incidence is concerned, it would appear that migrant sensory neuritis is an unusual visitor to the north east of England at least, at any season of the year and may even be an endangered species! In view of this, it is perhaps surprising that the condition appears to "cluster" in the Thames Valley and San Francisco. With reference to this, it is clear that only two of Wartenberg's nine cases conform strictly to his own description of its clinical characteristics, a point noted by Matthews and Esiri. Accordingly Wartenberg's syndrome is likely to prove the explanation for transient sensory disturbances in only a small minority of cases, a point underlined by the fact that it is not mentioned in recently published monographs on peripheral nerve disease[4-7] and the relevant entry in the all embracing *Handbook of Clinical Neurology*[8] is no more than a review of Wartenberg's monograph and the papers which preceded it. Regrettably, therefore, early multiple sclerosis is likely to prove the explanation for such transient sensory experiences in many instances.

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