stricture in the sigmoid colon. At laparotomy a large carcinoma was found in the sigmoid colon and a second similar lesion was found in the transverse colon. While resecting the large sigmoid growth a portion of the right ureter was removed. A primary colonic anastomosis was performed and covered by a double-barrelled colostomy fashioned in the transverse colon after resection of the second lesion. Urinary leakage occurred soon after operation and an intravenous pyelogram confirmed a leak from the right ureter. Urinary leakage continued postoperatively, and because of the poor general health of the patient renal embolisation was considered. The left femoral artery was catheterised under local anaesthesia and a right renal arteriogram was obtained. Two, or most probably three, renal arteries were seen supplying the right kidney and embolisation of two of these was achieved with Gelfoam and wire springs. Urinary leakage was considerably diminished subsequently. A further intravenous pyelogram showed minimal delayed excretion from the right kidney, suggesting perfusion from a small accessory artery. Leakage ceased completely after two months.

Comment

Ureteric damage may occur during resection of any extensive colonic or pelvic growth and if it is recognised at the time primary repair or transureteroureterostomy, reimplantation into the bladder and nephrectomy are the procedures of choice.^{1 2} Ureteric ligation is simpler, but there is a risk of subsequent infection and leakage. If this occurs, or if the injury is not recognised at the time, the surgeon is faced with the prospect of further surgery in the postoperative period if the leak does not stop spontaneously. Renal embolisation is an alternative in the elderly, poor-risk patient not fit for further surgery, provided contralateral renal function is adequate. This can be assessed by intravenous urography or divided renal function tests with a renal scan. Leakage of urine by vesicoureteric reflux must also be excluded.

In one patient the leakage ceased dramatically, and was reduced in the second patient, though it did not stop completely for several weeks owing to a small aberrant renal artery. Both patients left hospital after a simple procedure and neither experienced any appreciable loin pain. Transrenal ureteric embolisation with direct closure of the defect has also been described.³

Therapeutic embolisation is now widely practised by radiologists for several indications including renal tumours, hepatic metastases, bleeding oesophageal varices, and complicated arteriovenous fistulae.⁴ In certain poor-risk patients it would appear to be useful for post-operative ureteric leakage.

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(Accepted 8 July 1981)

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Localised amyloid deposit producing paraplegia

Localised deposits of amyloid have been described in the bladder, larynx, lung, and breast¹ without evidence of generalised amyloidism. Although amyloidosis may be localised to the central nervous system, and localised disease of the gasserian ganglion² has been described, we have been unable to find previous reports of localised amyloid affecting the spinal extradural space.

Case report

A 76-year-old woman was admitted with a six-month history of increased weakness and paraesthesia of the legs with nocturia and incontinence of urine. On examination she had a spastic paraparesis with associated loss of position sense in the feet and vibration sensation in the legs. Coned thoracolumbar views with tomography showed a left paraspinal tumour containing calcification adjacent to a considerably narrowed disc space. Myelography

showed a complete block at the level of T11/12. At operation a friable extradural tumour was removed. The result of histological staining with Congo Red was positive, and there were a few clumps of well-differentiated plasma cells and a few multinucleated giant cells.

No further evidence of amyloidosis has been found: results of bone-marrow examination showed no abnormality, and plasma immunoglobulin concentrations were within normal range. Erythrocyte sedimentation rate was normal, and results of analysis of urine for Bence Jones proteinuria was negative. Videocystometry showed uninhibited detrusor contractions with instability at 300 ml filling.

After intensive rehabilitation the patient returned home to live independently and continence returned.

Comment

Amyloid deposits may occur as part of aging³ or may be secondary to conditions such as rheumatoid arthritis, chronic infection, or myelomatosis. In this case, although plasma cells were found in the tumour specimen, there was no other evidence of myeloma either biochemically or in bone-marrow sections.

Amyloidosis is found in about one-quarter of patients with paraplegia at necropsy,⁴ but this is the only case we can find where the paraplegia was caused by the amyloid deposit.

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(Accepted 14 July 1981)

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Skin necrosis after heparin injection

Skin necrosis is an uncommon complication of anticoagulant therapy with coumarin derivatives and is extremely rare after heparin administration.¹⁻³ Only three such patients have been reported to the Committee on Safety of Medicines (personal communication) and we here present the fourth.

Case report

A 79-year-old man was admitted for surgical treatment of his ischaemic left foot. He was given 5000 units of porcine sodium heparin containing chlorocresol preservative (Pularin, Duncan Flockhart) subcutaneously into the abdominal wall before femoral arteriography; he had never been given heparin before. Five days later he was given 10 000 units of heparin intravenously during exploration of the left femoral artery and received three further subcutaneous doses of heparin, each of 5000 units, on the second and third days after operation. By the fourth day areas of skin necrosis were noted at the sites of the three postoperative heparin injections.

These areas were up to 2.5 cm across. Two were excised, and they showed the following histological changes. Blisters were present in the epidermis with some haemorrhage into the blister spaces, and haemorrhagic infarction extended into subcutaneous fat. An acute necrotising angitis was also present, affecting primarily the small dermal blood vessels, but also extending into the subcutaneous fat (see figure). This was associated with fibrinoid changes and infiltration of the walls and adjacent tissue by polymorphonuclear leucocytes. Some of the leucocytes showed fragmentation of their nuclei (leucocytoclastic vasculitis).

Skin biopsy specimen showing necrotising angitis. Haematoxylin and eosin $\times 250$ (original magnification).

Comment

The histological features of the skin necrosis in this patient were consistent with a hypersensitivity reaction to heparin or possibly the preservative. This was corroborated by the demonstration, with an immunoperoxidase technique, of fibrinogen, immunoglobulin (IgG), and complement (C3) in the walls of the inflamed blood vessels.

Presumably the intravenous injection of heparin a few days before this reaction had caused antibody formation. Subsequent local injection would then have resulted in the union of antigen and antibody in the walls of small blood vessels. Complement would have reacted with the antigen-antibody complexes, and the reaction products of complement would have induced inflammation.

We thank Dr I L Brown and technical staff of the University Department of Pathology, Western Infirmary, Glasgow; and Dr I Lauder and technical staff of the University Department of Pathology, General Infirmary, Leeds, for immunohistochemical studies.

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(Accepted 8 July 1981)

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Speech therapy for patients with Parkinson's disease

The main features of the speech disorder of Parkinson's disease are reduced intensity of the voice; reduced variability in pitch, stress, and rhythm (abnormality of prosody¹); and abnormal rates of speaking. These speech disorders greatly affect the intelligibility of speech and

may convey the impression that the patient is demented, depressed, apathetic, or cold and unfeeling.¹ Obvious abnormality often persists on otherwise optimal treatment with drugs.² We have assessed two forms of speech therapy.

Methods and results

Nine patients with Parkinson's disease (eight men and one woman aged from 59 to 72) were treated in two ways in random order.

Intonational exercises—These aim at improving production and recognition of variations in pitch contours, volume, and duration,³ using the Vocalite (a voice-operated light source designed by the Department of Clinical Physics and Bio-engineering of the West of Scotland Health Boards, which enables the patient to monitor some of the prosodic aspects of his own speech) as a reinforcement device.

Proprioceptive neuromuscular facilitation—This aims at generating intensive multisensory input and proprioceptive feedback from manipulations of normal motor patterns.⁴

Each treatment was given for 10 one-hour sessions over two weeks in the patient's home, and patients were assessed after each course of treatment; eight patients were also assessed after six months.

A score of 0 or 1 was given for breathing pattern; initiation of speech and ability to vary volume were given a score from 0 to 5 and prosodic abnormality a score from 0 to 7, the higher number denoting greater abnormality in each case. Samples of speech were recorded on tape before treatment and at each assessment and were scored independently by a second speech therapist. Further details of the scoring system are available from us.

The two observers gave 25 of the 35 tapes identical scores for prosodic abnormality; only three differed by more than one point. Breathing control and initiation of speech showed no change after treatment, but ability to vary volume improved after intonational exercises. Prosodic abnormality improved after both forms of treatment (table). After six months prosodic abnormality had increased (from $2.13 \pm SD 1.81$ to $3.06 \pm SD 2.06$; $p < 0.05$), though it was still significantly less than before treatment ($4.69 \pm SD 1.73$; $p < 0.025$).

Comment

Change in prosodic abnormality score of one point or more was associated with change in the clarity of communication obvious to the patient and to others. Relatives commented on the accompanying improvement in social contacts.

Clinically useful improvement may thus follow speech therapy in patients with Parkinson's disease, and may persist for six months. The speech therapist rather than the treatment method may be responsible. Intonational exercises are much the more practicable of the two methods assessed and should be subjected to a formal controlled trial.

We thank the members of the Parkinson's Disease Society and the department of speech therapy, Southern General Hospital, Glasgow, for their co-operation; Mrs C Button for her help; and the anonymous donor whose generosity made the study possible.

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(Accepted 14 July 1981)

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Speech abnormality scores for nine patients with Parkinson's disease before and after speech therapy. Results are means $\pm$ SD

	Intonational exercises			Proprioceptive neuromuscular facilitation		
	Before	After	p*	Before	After	p*
Breathing control	0.67 $\pm$ 0.50	0.44 $\pm$ 0.53	NS	0.67 $\pm$ 0.50	0.56 $\pm$ 0.53	NS
Initiation of speech	1.67 $\pm$ 1.00	1.33 $\pm$ 0.50	NS	1.44 $\pm$ 0.73	1.33 $\pm$ 0.50	NS
Ability to vary volume	1.44 $\pm$ 1.24	0.78 $\pm$ 0.67	0.05	1.33 $\pm$ 1.12	1.22 $\pm$ 1.09	NS
Prosodic abnormality	4.00 $\pm$ 2.19	2.56 $\pm$ 1.67	0.01	3.89 $\pm$ 1.19	2.83 $\pm$ 1.54	0.025

*Paired t test.