SHORT REPORTS

Randomised trial of bandaging after sclerotherapy for varicose veins

In 1963 Fegan[1] described the principles of successful compression sclerotherapy, which entailed maintaining firm, uninterrupted compression for six weeks, thereby preventing the formation and spread of thrombosis. Since many patients find bandaging for this long uncomfortable some surgeons have modified Fegan's original principles, but there have been no controlled studies to evaluate the importance of the period of compression. We therefore conducted a randomised prospective trial to compare the results of compression bandaging for three and six weeks after sclerotherapy.

Patients, methods, and results

One hundred and forty-eight patients (169 legs) entered the trial, presenting between January 1976 and January 1979 with idiopathic varicose veins suitable for sclerotherapy. At the time of injection they were randomly allocated to be bandaged for three or six weeks; patients allocated to the six-week group had their legs rebandaged after the first three weeks. To help in assessing the results photographs were taken of all legs in two positions before injection with normal and infrared-sensitive film. The two groups were well matched for clinical characteristics, type of varicosities, and number and site of injections. Patients were subsequently assessed blind at three months and thereafter at yearly intervals. A standardised questionnaire was used to score their comments regarding pain, mobility, cosmetic appearance, and general satisfaction with the procedure. The doctor scored his findings regarding phlebitis, pigmentation, induration, and the disappearance of varicosities.

The table shows the mean scores for the two groups at each assessment up to two years. The scores for patients' symptoms and doctors' findings on examination were similar. Although in every case bandaging for three weeks appeared to produce more favourable results than bandaging for six weeks, the differences were not significant. During the study 11 patients in the six-week group (14%) and five (7%) in the three-week group needed further treatment for recurrence after sclerotherapy.

Mean scores for patients' assessments of their symptoms and doctors' assessments of their findings on examination up to two years after bandaging. (Three represents the best possible score, and 11 the worst)

Duration of bandaging	Time after bandaging			
	3 weeks (n = 160)	3 months (n = 153)	1 year (n = 78)	2 years (n = 20)
	Patients' assessment			
3 weeks	5·3	5·9	5·8	6·1
6 weeks	5·6	6·2	5·8	6·9
	Doctors' assessment			
3 weeks	5·4	4·9	5·1	5·5
6 weeks	5·7	5·0	5·9	6·9

All differences between pairs were not significant ($p > 0·1$), using Kolmogorov-Smirnov two-sample test.

Comment

Sclerotherapy has recently become a widely accepted technique, though debate continues about the exact indications.[2] Fegan[1] commented on the potential dangers of sclerotherapy without compression: damage to adjacent normal veins, pulmonary embolism, and recurrence of varicosities may all occur. His suggestion that compression should be maintained for six weeks was based on histological studies of injected and thrombophlebitic veins.[1] Some later reports[2 3] recommended a similar period, while others suggested bandaging for three or four[4] weeks after injection. No studies, however, compared the results of compression bandaging for different periods.

The results of treatment for varicose veins are difficult to measure.[5] Previous studies have used different methods, making comparisons difficult. The technique of injection and bandaging and the evaluation of results may vary widely. In our study the sclerotherapy was performed by only one doctor, and "blind" assessment included both patient satisfaction and objective improvement as evaluated by comparing the results with pretreatment photographs. This study failed to show any significant difference up to two years between the results achieved after three and six weeks' bandaging, and so far there is no evidence to suggest that patients in the three-week group will progress less favourably than those in the six-week group. Reassessment of our patients, however, will be continued for up to five years at least. The extra three weeks in bandages represent unnecessary discomfort and considerable inconvenience for the patient and additional expense for the Health Service.

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Renal function in patients with essential hypertension receiving nadolol

Non-selective beta-blockers such as propranolol and oxprenolol have an adverse effect on renal function especially in patients with impaired renal function.[1 2] This effect is probably mediated by a decrease in renal blood flow.[1] In one study[3] nadolol, a non-selective beta-blocker, caused a dose-related increase in renal blood flow in five hypertensive patients receiving a low-salt diet and might therefore perhaps be expected to improve renal function when used clinically. We therefore assessed renal function in 15 patients with mild essential hypertension before and 10 weeks after they started taking nadolol at an effective antihypertensive dose.

Methods and results

Fifteen men (age range 48-68 (mean 57) years) with mild essential hypertension (WHO I, normal intravenous pyelogram) whose treatment included a beta-blocker stopped taking the beta-blocker but continued any other antihypertensive treatment—namely, a diuretic in 11 cases (cyclopenthiazide in six, tienilic acid in five, prazosin in two, and none in two).

Four to five months after stopping the beta-blocker but while continuing with the other antihypertensive agents and without dietary restrictions, the patients attended as outpatients for the following tests of renal function: serum creatinine and urea concentrations, creatinine clearance, ^{51}Cr-EDTA clearance for estimation of glomerular filtration rate, and ^{125}I-iodohippurate clearance for estimation of effective renal plasma flow as described in a study with atenolol.[4] On test mornings patients fasted but took their tablets at the usual time, which varied from 06.00 to 08.00 between patients but was constant for individual patients. On a separate occasion, when the patients had not fasted but had taken their tablets at the usual time, blood pressure and pulse rate were measured four times with the patients lying and standing by trained technicians using an electronic version of the London School of Hygiene and Tropical Medicine blind manometer and phase IV of the Korotkoff sounds. Nadolol was then started at a dose of 20 mg/day; the patients continued to take the other antihypertensive agent, the dosage remaining unchanged. The dose of nadolol was gradually increased until mean standing blood pressure was below 140/90 mm Hg or standing pulse rate below 55 beats/min. Ten weeks after nadolol was started the tests of renal function and blood-pressure measurements were repeated.

A mean daily dose of 63 ± SD 29 mg (range 40-120 mg) of nadolol caused a significant ($p < 0.001$) fall in mean lying and standing blood pressures and pulse rates (table). Serum creatinine and urea concentrations were normal and did not change when nadolol was given (table). Creatinine clearance, glomerular filtration rate, and effective renal plasma flow were in the low-normal range before nadolol was started but did not change significantly with the drug. Mean 24-hour urinary creatinine, sodium, and potassium excretions likewise were not affected by nadolol.

Mean values of variables indicating renal function, blood pressure, and pulse rate before and after 10 weeks' treatment with nadolol

	No of patients	Before nadolol	After 10 weeks	Difference	SE
Serum creatinine (mmol/l)	15	0·10	0·10	0·0*	0·0
Serum urea (mmol/l)	15	6·0	6·0	+0·03*	0·24
Creatinine clearance (ml/s)	14	1·65	1·72	+0·07*	0·087
Glomerular filtration rate† (ml/s)	15	1·47	1·58	+0·104*	0·104
Effective renal plasma flow† (ml/s)	15	10·59	9·91	−0·684*	0·408
Urinary sodium excretion (mmol/24 h)	9	174	170	−4·0*	12·8
Urinary potassium excretion (mmol/24 h)	9	70	73	+2·6*	7·57
Blood pressure (mm Hg):					
Lying:					
Systolic	15	162	147	−14·7***	3·08
Diastolic	15	93	84	−9·0***	1·90
Standing:					
Systolic	15	152	138	−13·8***	2·92
Diastolic	15	97	88	−9·3***	2·45
Pulse (beats/min):					
Lying	15	80	60	−20·0***	3·02
Standing	15	87	62	−25·1***	2·44

Significance of differences: *Not significant ($p > 0.1$); ***$p < 0.001$ (paired t test).
†Corrected to standard body surface of 1·73 m².
Conversion: SI to traditional units—Serum creatinine: 1 mmol/l ≈ 11·3 mg/100 ml. Serum urea: 1 mmol/l ≈ 6 mg/100 ml. Sodium and potassium excretion: 1 mmol/24 h = 1 mEq/24 h.

Comment

An effective antihypertensive dose of nadolol caused neither a reduction nor an increase in renal function in these patients with mild hypertension who had only slight renal impairment.

Renal perfusion and filtration rate depend on a complex interplay of many factors, and the net effect of giving a beta-blocker is probably determined by its effect on cardiac output and renin release and by a direct effect on renal vasculature, which in the dog is reported to have both β_1- and β_2-receptors.[5] Preservation of renal function during beta-blockade is probably not solely dependent on cardioselectivity and may be important in patients with renal disorders.

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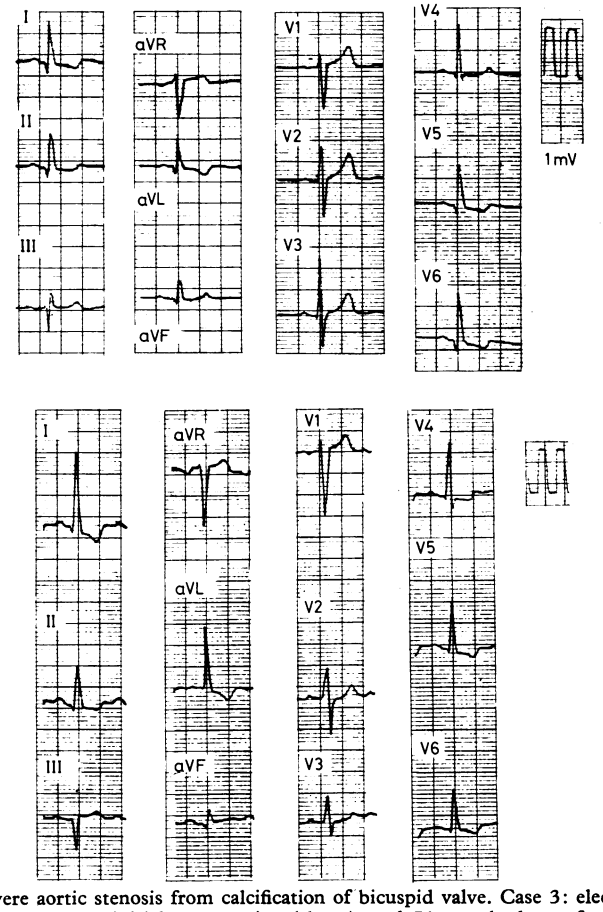
Rapid development of severe aortic stenosis from calcification of congenital bicuspid valve

The development of aortic stenosis from calcification of a bicuspid valve is unpredictable.[1] Three recent cases at the London Chest Hospital illustrate that the process may progress rapidly.

Case reports

(1) A 59-year-old woman presented with breathlessness and signs of aortic stenosis. The electrocardiogram (ECG) showed an R wave of 3·5 mV in V5, and her chest radiograph showed calcification in the aortic valve. She had iron deficiency anaemia. There was a peak systolic gradient of 40 mm Hg across the aortic valve, normal left ventricular contraction and coronary arteries, and mild aortic regurgitation. Her breathlessness responded to correction of her anaemia, but 37 months later she developed exertional angina. The signs were then of severe aortic stenosis. The chest radiograph showed increased valve calcification and the ECG increased QRS voltage with repolarisation changes. The aortic valve gradient was 120 mm Hg and the ventriculogram normal, and there had been no increase in regurgitation. At operation the heavily calcified bicuspid valve was replaced with a Starr-Edwards prosthesis. She was symptom free at follow-up.



Severe aortic stenosis from calcification of bicuspid valve. Case 3: electrocardiograms at initial presentation (above), and 54 months later after the development of severe left ventricular failure (below).

(2) A man aged 55 presented in December 1971 with recurrent palpitation. He had a "slightly slow-rising" pulse, ejection systolic murmur, and soft immediate diastolic murmur. The chest radiograph was normal. The ECG showed left axis deviation and lateral T wave inversion. There was a peak systolic gradient of 15 mm Hg across his lightly calcified aortic valve. Left ventricular contraction was normal. In March 1977 he developed effort angina. The results of physical examination were unchanged, but his chest radiograph showed calcification in the aortic valve and the ECG an increase in R wave voltage (though this was still within normal limits) and more prominent T wave inversion. The aortic valve gradient was 95 mm Hg. There had been no deterioration in left ventricular function or increase in aortic regurgitation. Coronary angiography showed stenoses in all major vessels. At operation a heavily calcified bicuspid valve was replaced and three