The clinical management of patients suffering from chronic neurological disease is notoriously difficult and tends to induce an attitude of therapeutic nihilism in medical and nursing staff alike. When such a condition is diagnosed the implicit assumption of inexorable progression of the lesion is accepted, hence the failure to review the patients in this series despite the eminently treatable nature of their disorder.

A particular difficulty with the therapeutic use of folic acid is that it can precipitate subacute combined degeneration of the cord in subjects with occult or undiagnosed vitamin B_{12} deficiency. This is not a reason for withholding folic acid in patients with obscure neurological disease, but rather underlines the need for precise diagnosis. In practice this fear of folate's toxicity is deeply entrenched in the clinical conscience and has proved a powerful disincentive to its prescription. This is all the more unfortunate since available tests permit precise diagnosis of disorders of vitamin B_{12} metabolism. In difficult cases it would be proper to give both to affected patients. Finally, since it takes several months for folate-induced subacute combined degeneration of the cord to develop,[14] a short-term trial (up to four weeks) of folic acid may be conducted in doubtful cases if the techniques, some of which may be esoteric, for distinguishing between the two deficiencies are not readily available to the clinician.

The diagnosis of folate deficiency poses several problems. There are no tests comparable to the Dicopac test in disorders of B_{12}, nor are there parallel markers of folate dysfunction such as antibody studies etc. At present diagnosis depends on showing low folate concentrations in serum or in the red cell. The red cell concentration would be regarded by some workers as a better index of tissue folate deficiency than the serum level. Unfortunately red cell folate values are reduced in some patients with vitamin B_{12} deficiency and cannot be confidently used to discriminate between the two possibilities[15]—a critical consideration in this particular study. It must be accepted that a low serum folate concentration does not necessarily indicate whole body folate deficiency, since the plasma folate compartment is labile and has a rapid turnover. In chronic or long-standing folate deficiency, however, the serum folate concentration represents a true equilibrium state, and low values indicate folate deficiency. The patients in this study were all considered to be suffering from long-standing dietary folate deficiency. The reasons for this have to be carefully elucidated but were not the main purpose of this study.

When the complex interactions between chronic folate deficiency, haemopoiesis, and neurologic function are considered several points emerge. Table II shows that there may be complete dissociation between the haematological and neurological effects and indicates a greater vulnerability of the nervous system to long-standing folate deficiency. The important corollary to this is that a normal haemoglobin level does not exclude the diagnosis of a severe folate-dependent neuropathy. Macrocytosis is a sensitive indicator of occult folate deficiency (table II), but this effect is masked by concurrent iron deficiency,[16] making the diagnosis even more difficult. This problem is illustrated by one of our patients (case 2), who presented with iron deficiency anaemia (nutritional). When this was corrected obvious macrocytosis of the red cells developed. It is interesting, and perhaps clinically significant, that the neurological lesion (spastic paraparesis) progressed rapidly over this same period.

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Deep venous thrombosis of the legs after strokes

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Part I— Incidence and predisposing factors

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Summary

Forty out of 76 patients (53%) who had suffered a cerebrovascular accident developed deep venous thrombosis of the paralysed leg, as detected with the ^{125}I-fibrinogen technique. A further five also had thrombosis in the non-paralysed leg. A study of many predisposing risk factors provided no help either in elucidating the cause of venous thromboembolism or in identifying patients at risk of DVT as a complication of cerebrovascular accidents.

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Introduction

The observation that deep venous thrombosis (DVT) may occur in the paralysed leg of patients after cerebrovascular accidents, or strokes, is not new. In 1810 Ferriar[1] described the clinical signs of DVT in a patient whose leg had been "previously affected by a paralytic stroke" and the necrospy appearance of a thrombus in the deep veins of a paralysed leg was clearly described by Lobstein in 1833.[2] Despite these early observations

Welch[3] made no reference to DVT in paralysed limbs and recent reviews have not drawn attention to the occurrence of DVT in patients with strokes.[4-6] We have therefore studied the incidence of DVT, diagnosed with ^{125}I-fibrinogen, in patients who have suffered strokes and the effect of various predisposing factors on this incidence.

Patients and methods

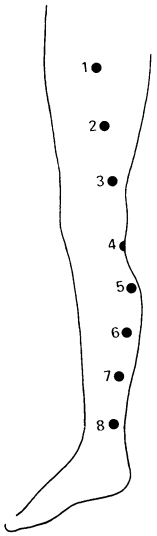
From October 1971 to September 1973 76 patients admitted within 48 hours of the onset of a stroke, causing either an acute hemiplegia or hemiparesis, were studied. Patients were excluded from the study if they had been confined to bed before the stroke, were unresponsive to painful stimuli, were allergic to iodine, thyroid function studies were contemplated, or anticoagulant therapy was thought to be indicated. Patients with a strong possibility of having sustained a cerebral embolus from a cardiac source were therefore not included.

The patients were managed at the discretion of their consultants in a conventional way with physiotherapy and early mobilisation. The occurrence of DVT was reported to their doctors and the patients were treated with compression bandaging and exercises only and not with anticoagulant drugs. Each patient was examined every day for 10 days by one of the authors.

DIAGNOSIS OF DVT

The ^{125}I-fibrinogen technique was used to diagnose DVT.[7-10] Potassium iodide, 120 mg orally, was given on admission and continued in a daily dose of 180 mg for three weeks. Five to 12 hours after admission about 100 μCi of ^{125}I-fibrinogen was given intravenously (Radiochemical Centre, Amersham). The praecordium, neck, and eight points along the course of the long saphenous vein (see fig) were scanned daily for 10 days with a Pitman 235 Isotope Localisation Monitor and DVT diagnosed according to the criteria of Kakkar *et al.*[11] No patient received any isotopic material for the diagnosis of pulmonary embolism until after the diagnosis of DVT with ^{125}I-fibrinogen, and the use of ^{99m}Tc pertechnetate in some patients for brain scanning interfered with the ^{125}I-fibrinogen test for only 48 hours. The problem of the simultaneous use of different tests requiring the use of radioisotopes was, therefore, avoided.[12]

The legs were also examined for clinical signs of DVT, including local tenderness, dilated superficial veins, increased local temperature, and ankle oedema.



Points on leg that were scanned after administration of ^{125}I-fibrinogen.

PREDISPOSING FACTORS

The patients were assessed on admission and daily during the 10-day study for factors that might have affected the incidence of DVT. In all patients the age, sex, month of admission, and side affected by the stroke were recorded. Cardiac failure was recorded as being present or absent on the basis of clinical signs or chest *x*-ray appearances, or both, and atrial fibrillation was diagnosed on a routine admission electrocardiogram. Blood pressure and packed cell volume (PCV) were also recorded on admission.

The degree of obesity of 67 patients was assessed by measuring the skinfold thickness over the mid-point of the left triceps muscle[13] with standard calipers (John Bull, British Indicators Ltd). Any history of venous thromboembolism was obtained from either the patients or their medical records. The presence or absence of varicose veins was recorded in 72 patients and peripheral vascular disease was assessed in 60 patients and recorded as present if no pulses could be felt below the popliteal artery. The presence or absence of voluntary movement of the leg affected by the stroke was recorded on admission, and in 73 patients assessment was made of whether voluntary power was increasing by the end of the 10-day study or before death. In 61 patients muscle tone in the paralysed leg was assessed on admission as being increased, normal, or decreased. The level of consciousness was assessed on admission in 72 patients, who were classified as responsive to painful stimuli only, responsive to verbal commands, drowsy but easily rousable, and fully conscious. The cigarette smoking habits of only 29 patients could be reliably ascertained and they were divided into those who smoked and those who did not.

Finally, the day on which the patients were first mobilised out of bed was recorded. No patient was known to have neoplastic disease and all the women were postmenopausal.

Results

There were 31 men and 45 women patients ranging in age from 52 to 87 years (mean 69·4). Forty-six patients had a right-sided weakness and 30 a left-sided weakness.

Incidence of venous thrombosis—DVT, diagnosed with ^{125}I-fibrinogen, occurred in 40 of the 76 paralysed legs (53%) and in five of the non-paralysed legs (7%). This difference was highly significant (Fisher's exact test; $P<0.001$) and all the patients with DVT in their non-paralysed leg also had DVT in their paralysed leg. Clinical signs of DVT occurred in 24 of the 40 paralysed limbs with DVT and in one of the five non-paralysed limbs with DVT; this difference was not significant (Fisher's exact test; $P>0.1$).

Predisposing factors—Of all the factors studied (tables I-IV) only the presence of varicose veins had a statistically significant association with the development of DVT (Fisher's exact test; $P = 0.03$) but only in

TABLE I—*Predisposing factors and presence of DVT after strokes. Results are numbers (percentages) of patients*

	DVT	No DVT	P*
Men	16 (52)	15	>0·1
Women	24 (53)	21	
Past history of venous thromboembolism	3 (75)	1	>0·1
No past history	37 (51)	35	
Varicose veins in paralysed limbs	12 (63)	7	>0·1
No varicose veins in paralysed limbs	26 (49)	27	
Varicose veins in normal limbs	4 (21)	15	0·03
No varicose veins in normal limbs	1 (2)	52	
PVD in paralysed limbs	9 (50)	9	>0·1
No PVD in paralysed limbs	23 (55)	19	
PVD in normal limbs	2 (11)	16	>0·1
No PVD in normal limbs	2 (5)	40	
No voluntary movement	32 (53)	29	>0·1
Voluntary movement	8 (53)	7	
Improving voluntary power	9 (50)	9	>0·1
No improvement in voluntary power	30 (55)	25	
Increased muscle tone	5 (36)	9	>0·1
Normal or decreased muscle tone	26 (55)	21	
Cardiac failure	3 (43)	4	>0·1
No cardiac failure	37 (54)	32	
Atrial fibrilation	9 (50)	9	>0·1
Sinus rhythm	31 (53)	27	
Smokers	7 (64)	4	>0·1
Non-smokers	12 (67)	6	

*Statistical analyses by Fisher's exact test.
PVD = Peripheral vascular disease.

TABLE II—*Seasonal incidence of DVT in 76 patients with stroke*

Month of stroke	DVT		No without DVT
	No	%	
December-February	7	44	9
March-May	13	48	14
June-August	8	50	8
September-November	12	71	5

$\chi^2 = 3.001$; $P>0.1$.

TABLE III—*Conscious state and presence of DVT after strokes (72 patients)*

Responsiveness	DVT		No without DVT
	No	%	
Reacts to painful stimuli only	2	40	3
Reacts to verbal commands	5	63	3
Drowsy	15	54	13
Fully conscious	17	55	14

TABLE IV—*Effect of blood pressure on admission on incidence of DVT after strokes*

	DVT		No without DVT
	No	%	
Systolic blood pressure (mm Hg):			
<140	7	58	5
140-179	15	44	19
180-219	14	64	8
>219	4	50	4
Diastolic blood pressure (mm Hg):			
<100	18	49	19
100-119	14	58	10
>119	8	53	7

χ^2 = 2·23 (systolic) and 0·574 (diastolic); P>0·1.

the non-paralysed leg. There was no significant difference between the mean (± SD) age of those with DVT (70·4 ± 7·8 years) and those without DVT (68·4 ± 8·5 years) (Student's *t* test; P>0·1; table V). The skin-fold distribution (table VI), although skewed, showed that obesity played no part in the development of DVT in these patients; the mean skinfold thickness in each group was identical—18·5 mm. There was no significant difference between the mean (± SD) PCV of the patients with DVT (44·0 ± 4·5%) and of those without DVT (42·2 ± 4·2) (Student's *t* test; P>0·1). The DVT in these patients was usually unilateral, but the incidence in the paralysed right (50%) and paralysed left (57%) limbs was not significantly different (Fisher's exact test; P>0·1). Finally, the number of days for which the patients were confined to bed clearly had no effect on the incidence of DVT (table VII); the incidence in those mobilised in under four days was almost identical to that in those mobilised after 10 or more days.

TABLE V—*Age range of patients with and without DVT after strokes*

Age (years):	50–54	–59	–64	–69	–74	–79	–84	–89
No with DVT	2	3	3	7	12	8	4	1
No without DVT	2	6	3	9	7	4	5	

TABLE VI—*Obesity, expressed as skinfold thickness, of patients with and without DVT after strokes (67 patients)*

Skinfold thickness (mm):	0–9	–14	–19	–24	–29	–34	>35
No with DVT	7	9	8	4	2	4	4
No without DVT	5	8	4	5	2	4	1

TABLE VII—*Effect of bed rest on incidence of DVT after strokes (75 patients)*

Day out of bed after stroke:	<4	4–10	>10	Died in study period before mobilisation
No (%) with DVT	13 (59)	13 (48)	6 (60)	7 (44)
No without DVT	9	14	4	9

Discussion

DVT of the legs is common after cerebrovascular accidents resulting in either a hemiplegia or hemiparesis. The incidence of DVT, diagnosed with ^{125}I-fibrinogen, was 53% in this series of 76 patients. The fact that the ^{125}I-fibrinogen technique does not detect thrombi in the upper thigh is unlikely to have led to a great underestimate of the incidence of DVT since venous thrombi in the iliofemoral veins, detected at necropsy[14-19] or venographically,[20 21] are usually associated, either in continuity or not, with more peripheral thrombi that can be detected with ^{125}I-fibrinogen.

The incidence of DVT lay between the 60% reported in our preliminary study[22] and the 45% reported by Denham *et al*,[23] who also used the ^{125}I-fibrinogen technique. An incidence of 33% in the hemiplegic limb and 8% in the normal limb, using venography, has been reported in a series of 150 patients with chronic, rather than acute, hemiplegia.[24] The necropsy incidence of DVT in patients with stroke has never been reported although the occurrence of DVT in hemiplegic legs has been commented on by some workers.[2 17 25]

Since venous thromboembolism occurs only in some patients it may be possible to identify predisposing risk factors that could both provide information on the pathogenesis of the condition and contribute towards identifying patients at risk of developing venous thrombosis. Our results were disappointing in both respects since the only risk factor that was significantly associated with DVT was the presence of varicose veins, but this was only with respect to the non-paralysed leg of patients with strokes. Although varicose veins are commonly regarded as a predisposing risk factor the evidence from other studies using ^{125}I-fibrinogen has been conflicting.[26-28] Possibly varicose veins are sometimes associated with DVT because of a history of venous thromboembolism, which has resulted in varicose veins, and which itself is more closely associated with the subsequent development of DVT.[25 29] The lack of significant association between DVT and a past episode of venous thromboembolism in our study may have been due to the difficulty of obtaining a medical history from patients who were often dysphasic and whose case records contained insufficient documentation or were unobtainable.

Increasing age has often been thought to be associated with a higher incidence of venous thromboembolism, although necropsy findings have conflicted,[18 29-31] as have the findings of studies using ^{125}I-fibrinogen.[26 27-32] In our series age had no significant effect on the development of DVT. Some of these discrepancies may have been due to the added effects of neoplasia and cardiac disease, which are commoner in the elderly and also associated with a high incidence of DVT,[33] but it is equally possible that the effect of increasing age is less pronounced in older age groups such as those we studied.

Although obesity is widely quoted as a predisposing factor in venous thromboembolism,[26 30] we found that it had no effect n our patients with strokes. Hills *et al*, also using ^{125}I-fibrinogen, have reported a similar lack of effect in postoperative patients.[34] The patients in our series had almost the same incidence of DVT whether or not they were mobilised early. Although there is good necropsy evidence of an increasing incidence of DVT with increasing periods of confinement to bed this association has never been satisfactorily dissociated from the possible effect of serious illness, which is itself normally associated with prolonged bed rest.[17 18] Bed rest alone clearly cannot be the most important factor determining the high incidence of DVT in hemiplegic limbs since the incidence is much lower in the non-paralysed limbs. Also the incidence of DVT is much higher in patients who have suffered myocardial infarction than in those patients who have undergone similar periods of bed rest but who are subsequently thought not to have developed infarction.[35] Hence probably the early onset of DVT, detected with ^{125}I-fibrinogen, in patients who have been acutely ill is independent of the period of confinement to bed, whereas the subsequent propagation and embolisation of thrombi, detected at necropsy, may not be. Only further studies will clarify this point, but clearly the early mobilisation of patients after strokes will not prevent venous thrombosis.

None of the other predisposing factors studied had any significant effect on the incidence of DVT and conflicting results with respect to many of them have been reported.[30] Perhaps surprisingly there was so little difference in the incidence of DVT between patients with an immobile leg and those who retained some voluntary power. This might suggest that the degree of paresis itself does not determine whether or not a patient is likely to develop DVT, but the fact that one leg is normal and the other paralysed may be sufficient to determine in which leg thrombosis will occur in a susceptible individual. The identification of people who are susceptible is clearly not

dependent on any of the predisposing factors that we have studied.

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Part II—Natural history

Summary

Seven out of 76 patients who had sustained a cerebrovascular accident suffered a pulmonary embolism as diagnosed at necropsy or by unequivocal antemortem criteria. A further five patients had probable embolisation diagnosed only by clinical and chest x-ray criteria. Eleven of these 12 patients had DVT as diagnosed by the ^{125}I-fibrinogen technique. Though ^{125}I-fibrinogen technique has its limitations, thrombosis seemed to be able to develop at several independent sites in the venous system of the leg.

Introduction

In Part I we showed that the incidence of deep venous thrombosis (DVT), as detected by the ^{125}I-fibrinogen technique, in patients who had suffered strokes was 53%. We then examined the incidence of pulmonary embolism in the same patients. Since conventional anticoagulation is probably hazardous after recent cerebral infarction not due to arterial embolis,[1] we were able to study the natural history of venous thromboembolic disease.

Patients and methods

The 76 patients were described in Part I, as were the methods of diagnosing DVT. Follow-up periods ranged from two months to two years.

Pulmonary embolism was diagnosed either by the doctors caring for the patients or at necropsy by the pathologists working in the Aberdeen University department of pathology. Definite pulmonary embolism was diagnosed only if there was either macroscopic embolism at necropsy or very strong antemortem evidence. Probable pulmonary embolism was diagnosed on the basis of clinical symptoms and signs and chest radiography.

Results

Nineteen patients died in the 10-day study period (mortality rate 25%), and by the end of follow-up 43 patients were known to be dead (mortality rate 57%). Nine necropsies were carried out.

INCIDENCE OF PULMONARY EMBOLISM

Macroscopic pulmonary emboli were found in five of the nine patients who came to necropsy, and in one of these patients (case 4)

TABLE I—*Details of embolism and positive sites on ^{125}I-fibrinogen scanning in seven patients with definite and five with probable pulmonary embolism*

Case No	Pulmonary embolism			^{125}I-fibrinogen			
	Day of onset after stroke	Clinical evidence	Necropsy performed	Positive positions in leg		Day of onset of positivity	
				Right	Left	Right	Left
Patients with definite pulmonary embolism							
1	< 7	None	Yes	4, 5, 6 ,7 8	6, 7, 8	3	3
2	28	Pleuritic pain haemoptysis, positive, lung scan	No				
3	7	Acute cor pulmonale	No	1, 2, 4, 5, 6, 7, 8		3	
4	30	Sudden death	Yes	6, 7, 8		3	
5	< 3	None	Yes	? 4		<2	
6	16	None	Yes	1, 2, 3, 4, 5, 6, 7		5	
7	<20	None	Yes				
Patients with probable pulmonary embolism							
8	5	Haemoptysis, pleuritic pain, consolidation on chest *x*-ray film	No	4, 5, 6		5	
9	63	As case 8	No	5, 6 7,		<1	
10	8	Atelectasis on chest *x*-ray film	No		6		<2
11	21	Pleuritic pain, pleural effusion	No	3, 4, 5, 6, 7, 8	6, 7	<2	7
12	42	Pleural effusion	No	7		9	