References

- ¹ Crowther D, Powles RL, Bateman CJT, et al. Management of adult acute myelogenous leukaemia. Br Med J 1973;i:131-7.
- ² Powles RL, Russell J, Lister TA, et al. Immunotherapy for acute myelogenous leukaemia. A controlled clinical study 21 years after entry of last patient. Br J Cancer 1977;35:265-75.
- ³ Lister TA, Johnson SAN, Bell R, Henry G, Malpas JS. Progress in acute myelogenous leukaemia. In: Neth R, Gallo RC, Graf T, Mannweiler K, Winkler K, eds. Modern trends in human leukaemia. IV. Berlin: Springer-Verlag, 1981:38.
- ⁴ Lister TA, Whitehouse JMA, Oliver RTD, et al. Chemotherapy and immunotherapy for acute myelogenous leukaemia. Cancer 1980;46:2142-8.
- ⁵ Rees JKH, Sandler RM, Callener J, Hayhoe FGJ. Treatment of acute myeloid leukaemia with a triple cytotoxic regimen : DAT. Br J Cancer 1977;36:770-4.
- ⁶ Gale RP, Cline MJ. High remission induction rate in acute myeloid leukaemia. Lancet 1977;i:497-9.
- ⁷ Schimpff SC, Grene WH, Young VM, Fratner CC, Jepson J, Cusack N. Infection prevention in acute non-lymphocytic leukaemia. Laminar air flow room reverse isolation with oral non-absorbable antibiotic prophylaxis. Ann Intern Med 1975;36:770-4.
- ⁸ Armitage P. Statistical methods in medical research. London: Halstead Press, 1971.
- ⁹ Peto R, Pike MC, Armitage P, et al. Design and analysis of randomised clinical trials requiring prolonged observation of each patient. II Analysis and examples. Br J Cancer 1977;35:1-39. ¹⁰ Preisler HD, Bhornsson S, Henderson ES. Adriamycin cytosine arabino-

- side therapy for acute myelocytic leukaemia. Cancer Treat Rep 1977; 61:89-92
- ¹¹ McCredie KB, Bodey GP, Freireich EJ, Hester JP, Rodriguez V, Keating MJ. Chemoimmunotherapy for adult acute leukaemia. Cancer 1981; 47:1256-61.
- ¹² Smith W, Applebaum F, Fefer A, Glucksberg H, Cheaver N, Thomas ED. Intensive therapy of adult acute nonlymphoblastic leukaemia (ANL). Proceedings of the American Society of Clinical Oncology 1980;21:455.
- ¹³ Weinstein HJ, Mayer RJ, Rosenthal DS, et al. Treatment of acute myelogenous leukaemia in children and adults. N Engl J Med 1980;303:473-8. ¹⁴ Arlin Z, Gee T, Frid J, Roenigsberg E, Wamark N, Clarkson B. Rapid
- induction of remission in acute non-lymphocytic leukaemia (ANLL). Proceedings of the American Association for Cancer Research 1979;20:112.
- ¹⁵ Glucksberg H, Cheever MA, Farewell VT, Fefer A, Sale GE, Thomas ED. High dose combination chemotherapy for acute nonlymphocytic leukaemia in adults. *Cancer* 1981;48:1073-81.
- ¹⁶ Jackson RRP, Birkhead BG, Bell R, Lister TA, Gregory WH. Application of Jackson-Aspden acute myeloid leukaemia model. Journal of the Operational Research Society (in press).
- ¹⁷ Mayer RJ, Coral FS, Rosenthal DS. The role of intensive post induction chemotherapy on the management of patients with acute myelogenous leukaemia. Cancer Treat Rep (in press). ¹⁸ Preisler HD, Brecher M, Browman G, et al. The treatment of acute myelo-
- genous leukaemia in children and young adults. (Submitted for publication.)

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Malalignment of the shoulder after stroke

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Abstract

One hundred and ten consecutive patients (51 men, 59 women) admitted to a stroke unit were studied for radiographic changes at the shoulder on the affected side. Malalignment was found in 51 (46%) patients, of whom 37 (72.5%) had changes on the initial x-ray film and a further 14 (27.5%) developed malalignment over the following 12 months. These findings indicate that malalignment of the shoulder is common in the early stages of a stroke and may be missed unless radiographs are taken with the patient erect and the arm unsupported. Malalignment may lead to delay and limitation in restoration of function. The consequences of malalignment can be prevented by correct handling, positioning, and full passive movement of the shoulder from the onset of the stroke.

Introduction

Several studies¹⁻⁶ have shown that the prevalence of subluxation of the affected shoulder after a stroke is between 17% and 60%. No changes have been reported in non-hemiplegic controls.3 Since all of these studies have been retrospective, we do not know whether the changes of subluxation take place

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Royal Victoria Hospital, Edinburgh EH4 2DN I G CRUIKSHANK, FRCPE, FRCR, consultant radiologist SHELAGH DUNBAR, MCSP, physiotherapist A J AKHTAR, FRCPE, consultant physician in geriatric medicine soon after the stroke or as a later result of paralysis and disuse. To determine the incidence and time of occurrence of subluxation, we decided to examine prospectively patients with acute stroke for malalignment of the shoulder and record the progression of the condition and its association with the degree of paralysis.

Patients and methods

A prospective study of 110 consecutive admissions to the stroke unit at the Royal Victoria Hospital, Edinburgh, was planned with the aim of following up each patient for one year. The criteria for admission to the stroke unit have been described.7 The patients were examined within 24 hours of admission and the following information collected: date of onset of stroke; date of admission to the stroke unit; affected side of body; grade of power in affected upper limb at the elbow (using the Medical Research Council scale 0-5); and the relevant history, especially of a previous stroke.

If the patient's condition permitted, radiographs of both shoulders were taken with the patient both supine and erect with the arm unsupported. Radiographic examination was repeated at one, two, three, six, and 12 months. The date of death was recorded if it occurred during the 12 months. At the end of the study all the radiographs were examined and abnormalities noted. The two independent examiners (RGS and JGC) had no difficulty in interpreting the grades of malalignment.

The 110 patients comprised 51 men and 59 women with a mean age of 72.6 years (range 59-93) for men and 75.3 years (range 61-93) for women (table I). Seventy-nine patients were admitted within 48 hours of the onset of the stroke, 51 on the same day. Two patients were not admitted until more than 14 days after the stroke. In 59 patients the left arm was affected and in 51 the right. Various degrees of paralysis of the affected upper limb were found. Forty-one of the 110 patients died within a year of their stroke, 21 within the first month. Ten patients were lost to follow-up as they left the Edinburgh area, and 27 were discharged from follow-up before 12 months as they had made a satisfactory recovery. Thirty-two patients were followed to the end of the study.

Results

Fourteen patients were too ill initially to have erect radiographs taken. At the first radiographic examination 37 of the remaining 96 patients had changes of malalignment, and a further 14 developed changes at subsequent examinations (table II). The overall incidence was therefore 53%. Forty-six patients presented with complete paralysis of the arm, of whom 28 (60%) showed malalignment.

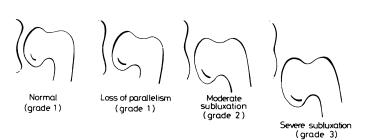
TABLE I—Age and sex of patients

Age (years)	Sex		
	Male	Female	No of patients $\binom{0}{20}$
< 65	3	2	5 (4.5)
65-74	34	27	61 (55.5)
75-84	13	25	38 (34.5)
>85	1	5	6 (5.5)
Total	51	59	110 (100)

TABLE 11—Incidence of malalignment of shoulder at initial and subsequent radiographic examinations

Time of		No of new cases		
examination	No of patients examined	Men	Women	Total
On admission	96	18	19	37
At:	54			
1 Month	76	4	4	8
2 Months	71	1	1	2
3 Months	65	1	1	2
6 Months	53		1	1
12 Months	32		1	1
Total		24	27	51

The degree of malalignment was assessed on a four point scale: grade 0, normal; grade 1, loss of parallelism between glenoid cavity and articular surface of humeral head; grade 2, moderate subluxation; and grade 3, severe subluxation. Fig 1 shows the degrees of malalignment shown on the erect film. This was assessed as grade 1 in 33 of the 37 patients who showed malalignment on admission and as grade 2 in the other four; one of these four had had a stroke on the same side six years previously and the other three had been admitted within 24 hours of their first stroke. Grade 1 changes developed in a further eight patients during the first month and in a further six over the 12 months. Three patients had changes on the contralateral shoulder caused by a previous stroke (two, grade 1, one, grade 2). There was no significant difference between the sexes in those patients with shoulder malalignment (Student's t test).



Degree of malalignment of shoulder joint.

The patients were re-examined at intervals over 12 months. Study of the initial and subsequent radiographs showed improvement in 11 patients and deterioration in 21; only three of the 21 patients had no evidence of malalignment on their initial radiographs. In 19 cases there was no change, but most of these patients were subsequently lost to follow-up or died before the end of the study. Subluxation (grade 2 or 3 changes) occurred in 15 (16%) patients, four initially and 11 subsequently over 12 months. Of the 65 patients examined after three months, 30 (46%) had some degree of malalignment (table III); the corresponding figures at six and 12 months were 22 of 53 (42%) and 10 of 32 (31%) respectively.

TABLE III—Grade of malalignment at three, six, and 12 months after stroke

Grade	3 Months	6 Months	12 Months
0	35	31	22
1	20	15	4
2	-9	7	5
3	1	0	ī
Total No of cases	65	53	32

Discussion

Subluxation of the shoulder after stroke has long been known to occur^{2 5 8 9} and may vary from a few millimetres of malalignment to complete dislocation.² Malalignment is therefore a more appropriate term than subluxation.

The stability of the normal shoulder depends mainly on the support of the surrounding muscle cuff⁴ as the joint capsule has to be sufficiently loose to allow the normal range of movement. After a stroke there is a period of initial flaccid paralysis in 90% of cases,⁶ which can last in some patients for less than 24 hours. During this time of flaccidity the gravitational pull of the affected arm on the musculature of the paralysed shoulder causes stretching of the joint capsule,⁵ as has been shown by arthrography.2 The gravitational pull readily leads to overstretching of the muscles surrounding the joint and predisposes to malalignment of the shoulder. Early malalignment may also be due in part to faulty lifting of the patient by attendants in the early stages of the stroke.⁴ Malalignment of the shoulder may be seen on a routine chest radiograph, but radiographs of the joint itself with the patient erect and the arm unsupported are needed to confirm this.^{2-4 6 10} The present study has confirmed that the conventional supine radiograph of the shoulder is of no value in detecting malalignment and may be misleading. The same is true of the appearance of the shoulder on a standard chest radiograph, although subluxation may be evident in advanced cases. Even with the patient erect malalignment may be missed if the affected arm is held in abduction during radiography.^{3 6} The present study has shown that malalignment of the shoulder after stroke usually occurs during the initial period of flaccidity and is by no means always a consequence of prolonged paralysis or disuse. Of 51 cases of malalignment, 37 (72.5%) were recorded on initial radiography, and only 14 (27.5%) developed subsequently over 12 months.

Previous studies¹⁻⁶ have described subluxation of the shoulder, and the present study detected 15 (16%) patients with moderate or severe subluxation (grades 2-3). This figure of 16% should, therefore, be compared with the wide variation in prevalence rates previously reported in retrospective studies in which the interval between the occurrence of the stroke and the radiographic examination differed widely.¹⁻⁶

Malalignment was correlated with the degree of paralysis but not with age, sex, or side of affected limb. Spasticity was not an important aetiological factor. No statistical difference was observed in mortality between those with and without malalignment. Increasing malalignment occurred in 41% of the 51 patients during the follow-up period. Early diagnosis of malalignment by erect radiographs of the shoulder after a stroke are necessary to avoid subsequent subluxation, which will cause a delay in successful rehabilitation.

Correct positioning in bed and adequate support of the flaccid shoulder are essential when the patient is lifted or moved. The prevalence of subluxation in stroke can be reduced to less than 5% by using these techniques.⁶ Junior nurses and other people handling the patient, including relatives, should be

taught to put the shoulder through its complete range of movement several times a day and to use the Australian technique when lifting the patient.4 5 10 11 A sling was first suggested in 1952¹² to prevent subluxation and frozen shoulder, and there has been considerable disagreement concerning its use. The main disadvantages are that it interferes with body image, the postural support of the arm, good gait training, and reciprocal arm swinging.²¹³ Correct handling of the patient in the early stages of a stroke is crucial in preventing the consequences of malalignment of the shoulder.

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References

¹ Taketomi Y. Observations on subluxation of the shoulder joint in hemiplegia. Phys Ther 1975;55:39-40.

- ² Miglietta O, Lewitan A, Rogoff JB. Subluxation of the shoulder in hemiplegic patients. NY State J Med 1959;59:457-60.
 ³ Najenson T, Pikielny SS. Malalignment of the gleno-humeral joint
- following hemiplegia. Ann Phys Med 1965;8:96-9.
- ⁴ Fitzgerald-Finch OP, Gibson IIJM. Subluxation of the shoulder in hemiplegia. Age Ageing 1975;4:16-8.
- ⁵ Tobis JS. Problems in rehabilitation of the hemiplegic patient. NY State J Med 1957;57:1377-80.
- ⁶ Moskowitz H, Goodman CR, Smith E, Balthazar E, Mellins HZ. Hemiplegic shoulder. NY State J Med 1969;69:548-50.
- ⁷ Garraway WM, Akhtar AJ, Prescott RJ, Hockey L. Management of acute stroke in the elderly: preliminary results of a controlled trial. Br Med J 1980;280:1040-3. 8 Bierman W, Licht S, eds. Physical medicine in general practice. New York:
- Hoeber, 1952:601.
- ⁹ Moskowitz E, Bishop HF, Shibutani K. Posthemiplegic reflex sympathetic dystrophy. JAMA 1958;**167**:836-8. ¹⁰ Anonymous. Shoulder pain from subluxation in the hemiplegic. Br Med J
- 1975;ii:581-2.
- ¹¹ Irvine RE, Strouthidis TM. Stiff shoulder after a stroke. Br Med J 1978; i:1622.
- ¹² Moskowitz E. Upper extremity complications in hemiplegic patients. Manitoba Med Rev 1967;47:448-50.

¹³ Anonymous. Painful shoulders and painful arcs. Br Med J 1977;ii:913-4.

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Objective test for food sensitivity in asthmatic children: increased bronchial reactivity after cola drinks

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Abstract

Ten asthmatic children with a history of cough and wheeze after drinking a cola drink performed histamine inhalation tests before and 30 minutes after a drink of Pepsi-Cola, soda water, and water on three separate study days. There was no significant change in baseline peak expiratory flow after any of the three drinks. Sensitivity to histamine was increased after the cola drink (p <0.005) but was not significantly different after soda water or water.

The detection of change in sensitivity to histamine appears to be a simple and effective method of testing for food sensitivity in asthma.

Introduction

The overall importance of food sensitivity in childhood asthma is unknown. Several substances such as nuts, eggs, milk, chocolate, and fish1 as well as food additives such as sulphur dioxide, benzoate, and tartrazine² cause wheezing in asthmatic subjects. When the reaction is severe and immediate, diagnosis is not a problem. Sometimes, however, the symptoms are mild, intermittent, or delayed, and a proportion of subjects with a history of such wheezing do not respond to an oral challenge test.¹² In a pilot study we found that five asthmatic children

giving a definite history of symptoms of cough and wheeze after a cola drink failed to show any change in their peak expiratory flow measured before and serially for two hours after an oral challenge. We suspected that rather than inducing airways obstruction directly the cola had its clinical effect by enhancing airways reactivity, so that common environmental provoking factors such as exercise could more readily precipitate an attack of asthma.

We designed the present study to compare airway sensitivity to inhaled histamine before and after an oral challenge to see whether a change in the degree of airway hyperreactivity might account for the alleged symptoms of cough and wheeze. Cola was chosen as the test substance as we had already shown no gross change in resting peak expiratory flow after such a drink and it was easy to eliminate from the diet preceding the test.

Methods

Ten asthmatic children giving a history of cough and wheeze after a drink of cola were selected (no particular brand was specified). Their mean age was 13.2 years (range 7-17), and nine were male. They attended the laboratory on three separate days but at the same time of day on each occasion. Beta-agonists were stopped for at least eight hours and sustained-release aminophylline for 24 hours before each study period. None of the subjects were taking cromoglycate, inhaled steroids, or an antihistamine preparation. Nine were from the Indian subcontinent.

A standard protocol was used on each of the three study days. After baseline peak expiratory flow had been recorded with a Wright peak flow meter (the maximum value over three attempts was accepted) histamine challenge was performed after the method described by Cockcroft et al.³ After inhalation of a control solution of 0.9% saline for two minutes increasing concentrations of histamine were inhaled for two minutes each, at five-minute intervals, from a Wright nebuliser. Peak expiratory flow was recorded 30, 90, and 120 seconds after the end of each inhalation. The procedure continued using doubling

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