Emotionalism after stroke

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Abstract

Objective—To estimate the prevalence of emotionalism after stroke, to assess its relation with other mood disorders, and to identify clinical variables with which it is associated.

Design—Descriptive study of a cohort of patients consecutively entered on a community stroke register.

Setting—Community based research project.

Patients—A total of 128 patients who had suffered first ever stroke.

Interventions and end points—Patients were interviewed by a psychiatrist at 1, 6, and 12 months after stroke. Mood state was assessed by standardised semistructured interview (present state examination) and self report (Beck depression inventory). Intellectual impairment was assessed by mini mental state examination and Frenchay aphasia screening test. In addition, stroke lesions were localised by computed tomography.

Main results—Emotionalism was reported by 13 of 89 patients (15%) at one month, 25 of 119 (21%) at six months, and 12 of 112 (11%) at 12 months after stroke. Patients with emotionalism had higher scores on both measures of mood disorder (at 6 months: mean Beck score 10.5 v 6.4; present state examination score 7.2 v 5.1) and more diagnosable psychiatric disorder (at 6 months: 40% v 14%; odds ratio 4.2, 95% confidence interval 1.5 to 11.9). Almost all episodes were provoked by clearly identified and appropriate emotional experiences. Patients with emotionalism also had more intellectual impairment and larger lesions on computed tomography. Lesions in the left frontal and temporal regions were particularly associated with emotionalism: at 6 months 8 of 14 patients (57%) with such lesions had emotionalism compared with 10 of 52 (19%) of those with lesions elsewhere (odds ratio 5.6, 95% confidence interval 1.4 to 22).

Conclusions—Emotionalism is common after stroke. It is neither emotionally meaningless and inappropriate, nor is it found mostly in patients with bilateral brain damage. Emotionalism is associated with symptoms of a more general mood disturbance and is found especially in patients with left frontal and temporal lesions.

Introduction

In recent years a number of research studies and review articles have emphasised the importance of psychiatric problems in survivors of stroke.¹⁻³ Depression has received particular attention,^{4,5} whereas other emotional problems which follow stroke have been relatively neglected. One which is well known to clinicians but which has not been studied systematically is pathological emotionalism, sometimes known as emotional lability. It has been described in a variety of disorders of the central nervous system,^{6,7} but there are no detailed accounts of its frequency or clinical course after stroke. Studies of its aetiology are few and have tended to emphasise the location of lesions as the important variable, although the brain areas implicated

have varied widely.⁶⁻⁹ Otherwise the cause of emotionalism in brain disease is unknown, although recent case reports have suggested that it may be associated with depressive disorder.^{9,10}

In this paper we present data on emotionalism obtained as part of a wider investigation of the psychiatric and social aspects of stroke undertaken by the Oxfordshire community stroke project.

Patients and methods

The Oxfordshire community stroke project has been described in detail elsewhere.¹¹ Patients with stroke who were interviewed for this study were registered in the stroke project between 1 March and 30 November 1986; 116 consecutive patients were seen by one of the neurologists and diagnosed as having suffered a first ever stroke.¹² Ninety five of these patients survived to one month and were able to participate in the study. At six months 89 of 91 survivors were available for follow up, and at 12 months 84 of 86 survivors were seen. A further 33 patients with stroke who had been registered in the project between November 1985 and March 1986 were included. These patients, who were all those who had survived to the time of follow up, were therefore seen for the first time six months after notification; 31 of the 33 survived to the interview at 12 months and all were seen. Thus a total of 128 patients were seen at least once in the year after their stroke.

PSYCHIATRIC ASSESSMENTS

All the subjects were interviewed (by AH) with the shorter version of the present state examination, a standardised psychiatric assessment designed for use in community surveys,^{13,14} which has been used in research in the elderly and the physically ill.^{15,16} In addition to its standard questions and ratings the following questions were asked to identify emotionalism:

(1) Have you been more tearful since the stroke than you were beforehand? Have you actually cried more in the past month (not just felt like it)?

(2) Does the weepiness come suddenly, at times when you aren't expecting it? (Suddenly means with only a few moments or no warning, not after several minutes trying to control yourself.)

(3) If you feel the tears coming on, or if they have started, can you control yourself to stop them? Have you been unable to stop yourself crying in front of other people? Is that a new experience for you?

In other words, the defining characteristics used were an increase in tearfulness with episodes of crying that were sudden or unheralded and not all under normal social control. Similar criteria were used to define pathological laughing. Information was obtained on the frequency of the episodes and the context in which they occurred, although "inappropriateness" was not part of the definition of emotionalism.

The patients were also asked to complete the Beck depression inventory.¹⁷ Those who were unable to complete the inventory unassisted because of linguistic, praxic, or other cognitive problems did not

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have it administered to them. Intellectual function was assessed with the mini mental state examination,¹⁸ and language function was assessed with the Frenchay aphasia screening test.¹⁹

NEUROLOGICAL AND RADIOLOGICAL ASSESSMENTS

Patients who were enrolled in the study were all assessed by a neurologist (MD or CW). As part of the initial assessment 117 patients (91%) had computerised tomography of the brain; scans were performed with a Siemens Somatom DR1 scanner with 8 mm cuts and without contrast injection. Brain scans were examined by a consultant neuroradiologist (AM), who reported the anatomical site and pathological type of any visible stroke lesion without knowing the neurological or psychiatric state of the subject. Lesions were also localised according to the topographical criteria suggested by workers at the Johns Hopkins Medical School.⁵ According to this method a lesion (which might be cortical or subcortical) is anterior if no part extends >60% caudally and some part extends <40% rostrally, posterior if some part extends >60% caudally and no part extends <40% rostrally, or intermediate if it lies between both lines or crosses them both. Volume of the lesion was estimated according to the method of Pulicino *et al.*,²⁰ so that volume of the lesion was categorised as small (<4 ml), medium (4-10 ml), or large (>10 ml); normal scans were placed in the small lesion group.

DATA ANALYSIS

Data from the present state examination were analysed with the CATEGO/ID program,²¹ which provides a total score of symptoms and derives syndromes from combinations of the symptoms rated. On the basis of these two measures the program gives a measure of severity of any psychopathology present: the index of definition. By convention, patients who score at levels 1-4 of the index of definition are not considered to have psychiatric disorder; those who score at level 5 and above are regarded as having psychiatric disorder and may then be given a diagnosis according to a standard psychiatric classification.

Other data were analysed with the statistical package for the social sciences (SPSS-X).²² Non-parametric tests of significance with two tailed tests of probability were used.

Results

At one month 89 of 95 patients (94%) were able to participate in the present state examination; exclusions were because of dysphasia or impairment of conscious-

ness. At six months 119 of 122 (98%) could participate and at 12 months the number was 112 of 115 (97%). All these patients were able to answer questions on emotionalism, but the Beck depression inventory could be completed by only 76 (80%) at one month, 107 (88%) at six months, and 88 (77%) at 12 months.

FREQUENCY AND CHARACTERISTICS OF EMOTIONALISM

Emotionalism that met our criteria was reported by 13 of 89 patients (15%; confidence interval 8 to 22%) at one month, by 25 of 119 (21%; 95% confidence interval 14 to 28%) at six months and by 12 of 112 (11%; 95% confidence interval 5 to 17%) at 12 months. All these patients had crying as their primary complaint; two of them also described episodes of pathological laughter. Although the prevalence of patients with the symptom did not fall greatly during the year, almost all reported an appreciable reduction in frequency and severity of the episodes they had experienced.

In patients who were seen at 1, 6, and 12 months all those who went on to develop emotionalism within the year had done so by the first interview. Patients seen only at six and 12 months were asked to date the onset of their symptoms retrospectively and all placed the symptom as starting within the first four to six weeks after the stroke.

Although rapidity of onset was part of our definition, most patients were able to describe typical situations that provoked emotionalism. For the two patients with pathological laughter the context was initially amusing but laughing was disproportionate. The stimuli for crying could be broadly categorised as: (a) sad or depressing—for example, thoughts of illness or dying from the stroke; discussion of illness or death in relatives or friends (even when the event being discussed was in the distant past); pictures of famine or disaster in magazines or on television; (b) sentimental—for example, visits from grandchildren, pictures of the Royal family, conventional expressions of greeting and enquiries about well-being; or (c) discussion of the symptom itself; tearfulness was provoked at interview simply by asking about its presence and causes in about half the patients (6/13 at one month, 11/25 at six months, and 6/12 at 12 months).

Sadness and sentimentality were equally common as provoking factors. Only one patient could not suggest any relevant circumstances surrounding the onset of crying.

OTHER ASPECTS OF MENTAL STATE

Mean scores of total symptom on the two measures of mood were higher in patients with emotionalism than in those without (table I). The differences in scores on the present state examination were significant at one month (Mann-Whitney U test, $z = -2.30$; $p = 0.02$) and six months ($z = -2.35$; $p = 0.02$); the difference in scores on the Beck depression inventory was significant only at six months ($z = -3.01$; $p = 0.003$). This was not simply the result of patients with depression being rated as having emotionalism; the prevalence of psychiatric disorder (index of definition ≥ 5) was not significantly greater in the patients with emotionalism at one month or 12 months, although it was at six months.

Emotionalism was also associated with cognitive problems. Those with emotionalism at one and six months (but not at 12 months) were more likely than those without to score below the usual threshold for intellectual impairment on the mini mental state examination (table I). Dysphasia, defined as a score of <20 on the first two sections of the Frenchay aphasia screening test, was present at one month in 6 of 13 patients (46%) with emotionalism and 13 of 76 (17%) without (odds ratio 4.15; 95% confidence

TABLE 1—Measures of psychiatric symptoms and intellectual impairment in patients with and without emotionalism after stroke. *p* Values from Mann-Whitney U test

	Patients seen at 1 month		Patients seen at 6 months		Patients seen at 12 months	
	With emotionalism (n=13)	Without emotionalism (n=76)	With emotionalism (n=25)	Without emotionalism (n=94)	With emotionalism (n=12)	Without emotionalism (n=100)
Mean score on present state examination	9.1	6.1	7.2	5.1	5.6	4.5
	(p=0.02)		(p=0.02)		(p>0.1)	
No of patients with index of definition*:						
≥5	4	13	10	13	1	11
1-4	9	63	15	81	11	89
Odds ratio	2.15		4.15		0.73	
95% Confidence interval	0.31 to 14.9		1.45 to 11.9		0.04 to 12.64	
Mean score on Beck depression inventory	9.9	7.1	10.5	6.4	7.5	5.3
	(p>0.1)		(p=0.003)		(p=0.07)	
No of patients with score on mini mental state examination:						
≤23	6	13	13	11	2	21
>24	7	63	12	83	10	79
Odds ratio	4.15		8.17		0.75	
95% Confidence interval	1.03 to 16.8		3.0 to 21.9		0.27 to 6.5	

*Index of definition was derived from scores on present state examination.

interval 1.27 to 13.5). No association of emotionalism with dysphasia was found at six and 12 months.

EMOTIONALISM AND LOCATION OF LESION

Of the 117 patients who had computed tomography, 62 (53%) had had cerebral infarct, 9 (8%) primary intracerebral haemorrhage, and 4 (3%) subarachnoid haemorrhage. No lesion was seen in 42 (36%). A scan was not obtained in 11 patients.

The proportion of patients with emotionalism whose brain scan showed a stroke lesion appropriate to the neurological findings was 10/13 (77%) at one month, 20/25 (80%) at six months, and 9/12 (75%) at 12 months. Patients with emotionalism were slightly more likely to have a positive scan than those without emotionalism (table II). There was a trend, which was significant only at one month, for patients with emotionalism to show large lesions on computed tomography (table II).

There was no definite association between location of lesions and emotionalism when the anatomical structures affected were examined. The proportion of patients with emotionalism and a positive scan who had only subcortical damage on the scan was 5/10 (50%) at one month, 6/20 (30%) at six months, and 4/9 (44%) at

12 months. In patients with lesions affecting only cortical structures damage was not exclusively restricted to the frontal lobes. For example, three patients with emotionalism at six months had lesions confined to the occipital lobes. No association was found between emotionalism and the side of the lesion on the scan (table III).

When intrahemispheric distribution of the lesion was examined according to the Johns Hopkins criteria⁵ patients with lesions that had anterior locations seemed more likely to have emotionalism, although the association was significant only at 12 months (table III). When patients with left anterior lesions were compared with those with visible lesions in any other part of the brain there was a significant association with emotionalism at one month (odds ratio 6.2, 95% confidence interval 1.05 to 36.5), six months (odds ratio 5.6, 95% confidence interval 1.44 to 21.7), and 12 months (odds ratio 28, 95% confidence interval 5.7 to 136). Table IV relates emotionalism to location of lesions according to the Johns Hopkins criteria. Of the eight patients seen at six months who had emotionalism and left anterior damage, five had lesions affecting the frontal or temporal lobes, which had been associated with dysphasia. In answering positively the questions about emotionalism they were not, however, reporting merely the frustrated tearfulness that is often thought to accompany speech problems.

TABLE II—Relation between volume of lesion and emotionalism after stroke in patients who had computed tomography

	Patients seen at 1 month		Patients seen at 6 months		Patients seen at 12 months	
	With emotionalism (n=13)	Without emotionalism (n=74)	With emotionalism (n=25)	Without emotionalism (n=92)	With emotionalism (n=12)	Without emotionalism (n=100)
Computed tomogram of brain:						
No of patients with lesion	10	43	20	55	9	61
No of patients without lesion	3	31	5	37	3	39
Odds ratio	2.4		2.69		1.92	
95% Confidence interval	0.41 to 14.0		0.82 to 8.8		0.25 to 14.5	
Volume of lesion:						
<4 ml*	5	43	7	41	3	44
4-10 ml	2	20	4	18	2	20
>10 ml	6	11	14	33	7	36
Odds ratio (lesion >10 ml v ≤10 ml)	4.9		2.27		2.5	
95% Confidence interval	1.2 to 19.7		0.83 to 6.3		0.55 to 11.2	

*Includes normal scans.

TABLE III—Relation between emotionalism after stroke and area of brain affected by lesion (brainstem strokes excluded)

	Patients seen at 1 month		Patients seen at 6 months		Patients seen at 12 months	
	With emotionalism (n=10)	Without emotionalism (n=40)	With emotionalism (n=20)	Without emotionalism (n=52)	With emotionalism (n=9)	Without emotionalism (n=59)
No of patients with lesions on:						
Right side	4	18	7	25	1	26
Left side	6	22	13	27	8	33
Odds ratio	1.23		1.7		6.3	
95% Confidence interval	0.01 to 34.4		0.39 to 7.4		0.92 to 1.1	
No of patients with lesion location:						
Anterior	5	12	9	14	7	16
Intermediate	2	13	5	18	2	21
Posterior	3	11	4	16	0	18
Odds ratio (anterior lesion v others)	2.0		2.43		8.5	
95% Confidence interval	0.2 to 19.6		0.6 to 9.3		1.53 to 47.5	

*Localisation according to Johns Hopkins Medical School criteria.⁵ Lesions on 4 scans in each group could be assigned to side but were too poorly defined to be localised.

TABLE IV—Percentages of patients who had lesions on computed tomography and reported emotionalism. Values in parentheses are numbers with emotionalism/total number with lesion in area of brain

Location of lesion (by Johns Hopkins criteria)	Patients seen at 1 month		Patients seen at 6 months		Patients seen at 12 months	
	Lesion on left side	Lesion on right side	Lesion on left side	Lesion on right side	Lesion on left side	Lesion on right side
Anterior	50 (5/10)	0 (0/7)	57 (8/14)	11 (1/9)	50 (7/14)	0 (0/9)
Intermediate	0 (0/7)	25 (2/8)	20 (2/10)	23 (3/13)	10 (1/10)	8 (1/13)
Posterior	14 (1/7)	29 (2/7)	9 (1/11)	33 (3/9)	0 (0/10)	0 (0/8)

Discussion

There is some confusion in published reports about the terminology and definition of the phenomenon described here. Some authors treat terms such as pathological crying, emotional incontinence, and emotional lability as interchangeable.⁸ Others use these terms to describe what they regard as different conditions, a distinction between different disorders of emotionality being made on the basis of the context in which the episodes occur.⁷ On the other hand, it has been suggested that the context in which crying occurs indicates severity rather than implying that there are two or more qualitatively different syndromes of emotionalism.²³ We did not include inappropriateness as a defining criterion in our study as we wished to study the full spectrum of disorders of emotionality after stroke. To avoid prejudging the issue we have used the general term emotionalism, which is defined in the *Shorter Oxford English Dictionary* as "The habit of . . . weakly yielding to emotion."

An interesting finding was that patients with emotionalism had more symptoms of other sorts on the two measures of psychopathology we used. This, coupled with our observation on context, would support the suggestion that the problem is commonly found in people with mood disorders after stroke and reflects felt as well as expressed emotion.⁹ Such an interpretation would account for the similarities between emotionalism associated with stroke and that seen after other acute life threatening illness such as myocardial infarction.²⁴ Counter to this argument is the finding both of pathological laughing and crying in the same patient and of some emotionalism not associated with other psychopathology. Rare neurological disorders such as pathological laughter as a phenomenon in epilepsy or as the prodrome of cerebral haemorrhage have been described^{6,7,25,26}; this suggests that sometimes crying and laughing can occur unprovoked by emotionally meaningful stimuli. Our results suggest that after stroke such a phenomenon is rare.

The other finding that suggests that emotionalism represents more than a psychologically understandable adjustment reaction was the association with location of the lesion. No site was implicated exclusively, but an

association was found with anterior lesions in the left hemisphere; in patients with these lesions the emotionalism seemed particularly persistent. Disorders of emotional expression that are not typically depressive have long been a recognised feature of damage in the left hemisphere.^{27, 28} This association of emotionalism with a focal hemispheric lesion is interesting because in neurological practice the phenomenon is often associated specifically with bilateral disease of the brain stem or cerebral hemispheres,⁷ although it has been reported after unilateral stroke.⁶

Our findings suggest that emotionalism is rarely inappropriate to the feelings and immediate emotional context of patients with stroke, and at the same time emotionalism is associated with certain characteristics of the cerebral lesion. These two observations are compatible if the emotionalism is considered as a form of disinhibition analogous to, say, urinary incontinence in neurological disorders. By this analogy, it is not that the behaviour is unprovoked by any stimulus or that it is provoked by an irrelevant one; what is lost is the ability to suppress a response at low levels of stimulation. Such a view suggests possibilities for management by modifying responsiveness to emotional stimuli or controlling levels of emotional stimulation.

Patients commonly describe emotionalism as both distressing and socially disabling, and as such it is a symptom which merits treatment in its own right. Several reports have indicated that emotionalism may respond to tricyclic antidepressants or levodopa.^{9, 23, 29-32} In our study no patient had received drug treatment for emotionalism, and there seemed to be little awareness among professionals that drug treatment might have a part to play. Psychological treatments might be considered where drugs are inappropriate or ineffective,³³ but they had not been tried in our patients, and no patient had been referred for specialist help with this problem. Larger scale studies are needed to determine whether drug treatment or psychological therapies are effective in treating emotionalism in the absence of other depressive symptoms.

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- 1 Binder LM. Emotional problems after stroke. *Stroke* 1984;15:174-7.
- 2 Rusking AP. Understanding stroke and its rehabilitation. *Stroke* 1983;14:438-42.
- 3 House A. Depression after stroke. *Br Med J* 1987;294:76-8.
- 4 Robinson RG, Bolduc P, Price TR. Two year longitudinal study of post-stroke mood disorders: diagnosis and outcome at 1 and 2 years. *Stroke* 1987;18:837-42.
- 5 Robinson RG, Kubos K, Starr L, Rao K, Price T. Mood disorders in stroke patients: importance of location of lesion. *Brain* 1984;107:81-93.
- 6 Kinnier Wilson SA. Some problems of neurology. II. Pathological laughing and crying. *Journal of Neurology and Psychopathology* 1924;4:299-332.
- 7 Poeck K. Pathophysiology of emotional disorders associated with brain damage. In: Vinken PJ, Bruyn GW, eds. *Handbook of clinical neurology*. New York: Elsevier, 1969:343-67.
- 8 Ironside R. Disorders of laughter due to brain lesions. *Brain* 1956;79:589-609.
- 9 Ross ED, Stewart RS. Pathological display of affect in patients with depression and right frontal brain damage. *J Nerv Ment Dis* 1987;175:165-72.
- 10 Ross ED, Rush AJ. Neuroanatomical correlates of depression in brain damaged patients. *Arch Gen Psychiatry* 1981;38:1344-54.
- 11 Bamford J, Sandercock P, Dennis M, et al. A prospective study of cerebrovascular disease in the community: the OCSF 1981-1986. 1. Methodology, demography and incident cases of first stroke. *J Neurol Neurosurg Psychiatry* 1988;51:1373-80.
- 12 Aho K, Harmsen P, Hatano S, Marquardsen J, Smirnov V, Strasser T. Cerebrovascular disease in the community: results of a WHO collaborative study. *Bull WHO* 1980;58:113-30.
- 13 Wing J, Cooper J, Sartorius N. *Measurement and classification of psychiatric symptoms*. London: Cambridge University Press, 1974.
- 14 Cooper J, Nixon SA, Mann A, Leff J. Reliability of the PSE (9th edition) used in a population study. *Psychol Med* 1977;7:505-16.
- 15 Murphy E. Social origins of depression in old age. *Br J Psychiatry* 1982;141:135-42.
- 16 Feldman E, Mayou R, Hawton K, Ardern M, Smith E. Psychiatric disorder in medical in-patients. *Q J Med* 1987;63:405-12.
- 17 Beck AT, Ward CH, Mendelson M, Mock J, Erbaugh J. An inventory for measuring depression. *Arch Gen Psychiatry* 1961;4:561-71.
- 18 Folstein MF, Folstein SE, McHugh PR. Mini mental state—a practical method for grading the cognitive state of patients for the clinician. *J Psychiatr Res* 1975;12:189-98.
- 19 Enderby P, Wood VA, Wade DT, Hewer RL. The Frenchay aphasia screening test: a short simple test for aphasia appropriate for non-specialists. *International Rehabilitation Medicine* 1987;8:166-70.
- 20 Pulicino P, Nelson RF, Kendall BE, Marshall J. Small deep infarcts diagnosed on computed tomography. *Neurology* 1980;30:1090-6.
- 21 Wing J, Mann SA, Leff J, Nixon JM. The concept of a case in psychiatric population surveys. *Psychol Med* 1978;8:203-17.
- 22 Nie NH. *Statistical package for social scientists (SPSS-X)*. Chicago: McGraw-Hill, 1983.
- 23 Lawson IR, MacLeod RD. The use of imipramine and other psychotropic drugs in organic emotionalism. *Br J Psychiatry* 1969;115:281-5.
- 24 Leegard OF. Diffuse cerebral symptoms in convalescents from cerebral infarction and myocardial infarction. *Acta Neurol Scand* 1983;67:348-55.
- 25 Martin JP. Fit of laughter (sham mirth) in organic cerebral disease. *Brain* 1950;73:453-64.
- 26 Berkovic SF, Andermann F, Melanson D, Ethier RE, Feindel W, Gloor P. Hypothalamic hamartomas and ictal laughter: evolution of a characteristic epileptic syndrome and diagnostic value of magnetic resonance imaging. *Ann Neurol* 1988;23:429-39.
- 27 Goldstein K. *Language and language disturbances*. New York: Grune and Stratton, 1948.
- 28 Gainotti G. Emotional behaviour and hemispheric side of lesion. *Cortex* 1972;8:41-55.
- 29 Schiffer RB, Cash J, Herndon RM. Treatment of emotional lability with low dosage tricyclic antidepressants. *Psychosomatics* 1983;24:1094-6.
- 30 Schiffer R, Herndon R, Rudick R. Treatment of pathologic laughing and weeping with amitriptyline. *N Engl J Med* 1985;312:1480-2.
- 31 Udaoka F, Yamao S, Nagata H, Nakamura S, Kameyama M. Pathologic laughing and crying treated with levodopa. *Arch Neurol* 1984;41:1095-6.
- 32 Wolf JK, Santana HB, Thorpy M. Treatment of "emotional incontinence" with levodopa. *Neurology* 1979;29:1435-6.
- 33 Brookshire RH. Control of "involuntary" crying behaviour emitted by a multiple sclerosis patient. *Journal of Communication Disorders* 1970;3:171-6.

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ONE HUNDRED YEARS AGO

Sir Lyon Playfair, in a letter to Junius Henri Browne, author of a paper in the *New York Forum*, for October, under the above title, says: "Having represented a large medical constituency (the University of Edinburgh) for seventeen years as a member of Parliament, I naturally came in contact with the most eminent medical men in England. I have put the question to most of them, 'Did you, in your extensive practice, ever know a patient who was afraid to die?' With two exceptions, they answered 'No.' One of these exceptions was Sir Benjamin Brodie, who said he had seen one case. The other was Sir Robert Christison, who had seen one case, that of a girl of bad character who had a sudden accident. I have known three friends who were partially devoured by wild beasts under apparently hopeless circumstances of escape. The first was Livingstone, the great African traveller, who was knocked on his back by a lion, which began to munch his arm. He assured me that he felt no fear or pain, and that his only feeling

was one of intense curiosity as to which part of his body the lion would take next. The next was Rustem Pasha, now Turkish ambassador in London. A bear attacked him, and tore off part of his hand and part of his arm and shoulder. He also assured me that he had neither pain nor fear, but that he felt excessively angry because the bear grunted with so much satisfaction in munching him. The third case is that of Sir Edward Bradford, an Indian officer, now occupying a high position in the India Office. He was seized in a solitary place by a tiger, which held him firmly behind the shoulders with one paw and then deliberately devoured the whole of his arm, beginning at the end and ending at the shoulder. He was positive that he had no sensation of fear, and thinks that he felt a little pain when the fangs went through his hand, but is certain that he felt none during the munching of his arm."

(*British Medical Journal* 1889;i:489)