serious problems, but they are sometimes mentioned in an alarmist and unhelpful way by members of staff.⁶ Patients' main worries were about the side effects of the drugs rather than about addiction, and so optimal management of pain should include adequate reassurance about these fears.

There were significant differences in the degrees of pain perceived by patients and those that they thought the staff perceived. For some patients pain relief was inadequate, and not always given swiftly enough. There seems to be a need for more formal monitoring of pain relief by nursing and medical staff. Also, because pain comes and goes during a crisis, patients should have some control over the amount of analgesia they are given. One can treat different degrees of pain using a simple visual analogue scale and achieve both these objectives.⁷

In conclusion, this study highlights the need for reappraisals of the management of pain in sickle cell disease, and of how pain is monitored and treated. The use of counsellors to help in advising patients on the avoidance of the crises should be encouraged. A

prodromal stage of the painful crisis was common, and this could be used to initiate early treatment.

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Effect of dietary salt on bronchial reactivity to histamine in asthma

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In a recent epidemiological survey increased dietary salt intake was positively associated with bronchial reactivity to inhaled histamine.¹ If this association is important in the pathogenesis of asthma a similar relation would be expected in individual patients. We therefore investigated the effect of increasing salt intake on bronchial activity in a group of asthmatic patients and healthy non-asthmatic volunteers.

Patients, methods, and results

Ten asthmatic patients who attended the hospital outpatient department were studied (six men, four women; age range 18-63 years). Five patients were atopic and five non-atopic. All had a history of intermittent wheezing and a greater than 15% change in forced expiratory volume in one second either spontaneously or after treatment. Three were taking an inhaled β_2 agonist daily, and seven were also receiving an inhaled steroid. All were non-smokers. Five non-asthmatic subjects (one man, four women; age range 23-48 years) volunteered to act as controls.

The dietary salt intake of all participants was assessed by a dietitian. Salt excretion was measured in two 24 hour urinary specimens, and the average was recorded. A histamine challenge test was performed with a modification of that described by Cockcroft *et al.*² Doubling doses of histamine in phosphate buffered saline were inhaled, from 0.09 mmol/l to 26.04 mmol/l. The forced expiratory volume in one second was measured one and two minutes after each inhalation, and the test was stopped when the volume decreased by 20% or more. The histamine concentration that produced a decrease in forced expiratory volume of 20% (PC_{20}) was calculated by linear interpolation of the last two points on the log dose response curve. All challenge tests were performed at midday, bronchodilator treatment having been withdrawn 12 hours beforehand.

Salt intake was then doubled in all participants by increasing dietary salt and also by giving them salt

tablets. At the end of one month the histamine challenge was repeated. For each participant the repeat baseline forced expiratory volume in one second was within 0.25 litres of the value recorded at the initial test. Measurement of 24 hour urinary salt excretion was also performed at that time.

The results were compared by Student's paired *t* test. For the histamine results this was done after logarithmic transformation. There was a significant increase in bronchial reactivity to histamine in nine of the 10 asthmatic patients when their salt intake was increased. The PC_{20} decreased from a mean (SD) of 7.49 (6.51) mmol/l to 5.2 (6.51) mmol/l ($p < 0.05$) (figure). Reactivity in the controls was normal and did not change on either occasion. The 24 hour salt excretion increased from 156 (50) mmol/24 h to 216 (39) mmol/24 h ($p < 0.0005$) in the asthmatics and from 103 (22) mmol/24 h to 183 (48) mmol/24 h ($p < 0.05$) in the controls.

Comment

Another British study has shown that increased salt intake increases bronchial reactivity.³ It suggested, however, that this phenomenon was restricted only to men, whereas we have shown this to occur in women asthmatics as well but not in healthy control subjects.

The way in which increased salt intake may cause increased bronchial hyperreactivity is unclear. Animal studies have shown that sensitised smooth muscle shows hyperreactivity as a consequence of increased activity of the cell membrane sodium pump and sodium influx.⁴ Increased sodium intake may possibly augment this effect.¹ As the degree of histamine reactivity is related to the severity of symptoms in asthma⁵ these results also indicate that a high dietary salt intake may contribute to the severity of asthma.

Further studies are needed to gauge the importance of salt intake in determining this severity and especially the role that salt restriction may have in its treatment.

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Logarithm of histamine concentration that produced 20% decrease (PC_{20}) in forced expiratory volume in one second before and after increased salt intake in asthmatic patients. ●=Men. ○=Women. Bars indicate mean and SD.