

ever explain why you should suture the peritoneum—or was it an accepted “fact” which everybody took for granted?

Answer (2) also seems valid at face value. An opening in the abdomen must be closed by pulling it together with sutures layer for layer, the first layer being the peritoneum. However, many surgeons disagree and report¹⁻⁴ that suturing the peritoneum does not reduce the incidence of wound dehiscence or hernia; it may even increase it.⁴

Answer (3) needs no comment.

Answer (4) does. Surgeons seem to believe that a denuded peritoneal surface is an open invitation to sprouting adhesions. However, a whole series of publications, from animal experiments and clinical experience of raw areas of bleeding peritoneum healing without adhesions, points in the opposite direction.⁶⁻¹¹ These papers are in accord that adhesion formation is increased by reperitonealisation and by suturing the peritoneum at wound closure. Left alone a raw, deperitonealised surface will be covered by mesothelial cells and indistinguishable from undisturbed areas in 7-8 days.¹³

Why then do we do it? Published work is overwhelmingly against it but the grass-roots surgeon, with few exceptions, continues to suture the peritoneum at wound closure and to cover denuded peritoneal surfaces. R  f¹² showed that 86% of patients with intestinal obstruction caused by adhesions and bands had a history of laparotomy. Could it be that by keeping our hands off the peritoneum we could reduce this figure, saving money and sparing human suffering.

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AEROALLERGEN SKIN SENSITIVITY IN PATIENTS WITH SEVERE ASTHMA DURING A THUNDERSTORM

SIR,—An asthma outbreak in the Birmingham area in July, 1983,¹ was associated with a thunderstorm and a large rise in airborne concentrations of *Didymella exitialis* ascospores. These spores can cause asthma,^{2,3} and it was suggested that their sudden release during heavy rainfall was the cause of the Birmingham outbreak^{3,4}—a view that we tentatively endorsed.¹ High concentrations of *Sporobolomyces* spores were also released and could have contributed to worsening asthma. We present here the results of allergen skin testing on some of the patients affected.

Of the 36 patients seen in hospital with acute asthma during the outbreak 18 responded to a letter and were willing to undergo further investigations. There were 13 males and 5 females (median age 24 years, range 6-59). A doctor administered a questionnaire seeking information about medical history and the relation between symptoms at the time of the outbreak and changes in the weather. Skin-prick tests were done on all patients and on 18 controls (median age 23.5 years; range 6-62) matched for age and sex who were asthmatics attending hospital clinics in Birmingham and who had not required extra treatment at the time of the outbreak. The

extracts used were mixed grass pollen; house dust; house dust mite; *Cladosporium herbarum* and *S roseus* (Bencard); *S roseus* (Dome); *S salmonicolor* (Hollister-Stier); three preparations of *D exitialis* 10 mg/ml (mycelium grown in malt extract and mycelium grown in potato dextrose broth, both extracted with Coca's solution, and whole culture plus medium grown in potato dextrose broth and extracted with buffered saline and phenol) and a control. A positive immediate skin reaction was accepted if there was a weal of 1 mm or more, or, in patients with dermatographism, if there was a weal size at least twice that of the control.⁵

All 18 had a history of hayfever and 17 gave a clear history of asthma. Only 10 had received specific therapy for this, of whom 9 had treatment available at the time of the outbreak. Only 1 patient, an 8-year-old boy, had previously needed hospital treatment for asthma. 13 said that their symptoms were worse in the summer, of whom 9 stated that their asthma was aggravated by newly cut grass and 6 said that they were worse after rain. All patients said that their hayfever had been troublesome in the weeks before the thunderstorm, and that symptoms had become suddenly worse over the evening and night of the storm.

All the patients were atopic. There were no significant differences between the 18 patients and the 18 controls in the numbers positive to each skin test extract. Most patients were positive to grass pollen, 6 in each group were positive to *Didymella* and over half were positive to *Sporobolomyces*:

Extract	Asthmatics	Controls
Grass pollen	17	16
House dust	14	18
House dust mite	14	16
<i>Cladosporium</i>	4	5
<i>Sporobolomyces</i>	11*	14*
<i>Didymella</i>	6*	6*

*Number positive to one or more solutions.

Of those patients positive to *Didymella* and/or *Sporobolomyces*, 5 complained of asthmatic symptoms after rainfall in summer.

The skin test results argue against an important role for *Didymella* or *Sporobolomyces* in the outbreak. The *Didymella* extract we used had given a good agreement between skin and bronchial sensitivity in patients whose asthma worsened whenever *Didymella* spore counts were high,³ and we used three different commercial extracts of *Sporobolomyces*, so variation in extract quality is not likely to be the explanation for the lack of difference between the two groups. The controls were on regular treatment, unlike most of the patients affected during the outbreak, and it is conceivable that some of the controls might have experienced worsening asthma at the time of the outbreak had they not been taking this treatment. 17 of the 18 patients were grass pollen sensitive. Pollen levels were negligible at the time of the storm, but counts had been raised on the four warm days preceding the storm.¹ During the summer, seasonal asthmatics who are sensitive to pollen respond by more severe bronchoconstriction to allergic and non-allergic stimuli,^{6,7} so sudden disturbances in the weather could induce airway narrowing in such patients by, for example, falls in air temperature, ionisation, increased ozone production, and the transport of particles and allergens from high altitude by downdraughts.

We hold by our original view that the asthma outbreak was the result of several interacting factors. Fungal spore allergy is unlikely to have been important.

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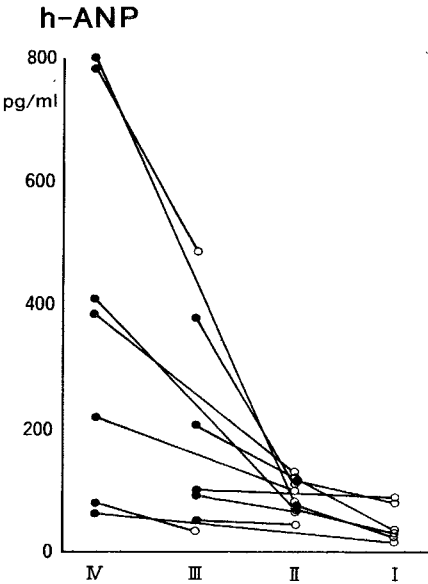
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ATRIAL NATRIURETIC PEPTIDE LEVELS IN TREATED CONGESTIVE HEART FAILURE

SIR,—We describe here a significant fall in plasma atrial natriuretic peptide (ANP) concentrations after treatment for congestive heart failure. Five male and ten female patients aged 38-75 (63±15) years were studied. Seven had congestive heart failure of class IV (New York Heart Association criteria), five were in class III, and three were in class II. Eight patients had coronary heart disease, two valvular heart disease, two hypertensive heart disease, two atrial septal defects, and one dilated cardiomyopathy. Plasma ANP concentrations (by radioimmunoassay) were very high on admission (254.4 [SEM 64.7] pg/ml; normal 31.7 [12.0], n=124). ANP plasma levels fell in all patients as treatment improved their symptoms (see figure), to 92.5 (29.4) pg/ml (p<0.005; paired Wilcoxon test).



Decrease in plasma ANP levels with improvement in congestive heart failure (NYHA criteria).
● = start of treatment; ○ = end of treatment.

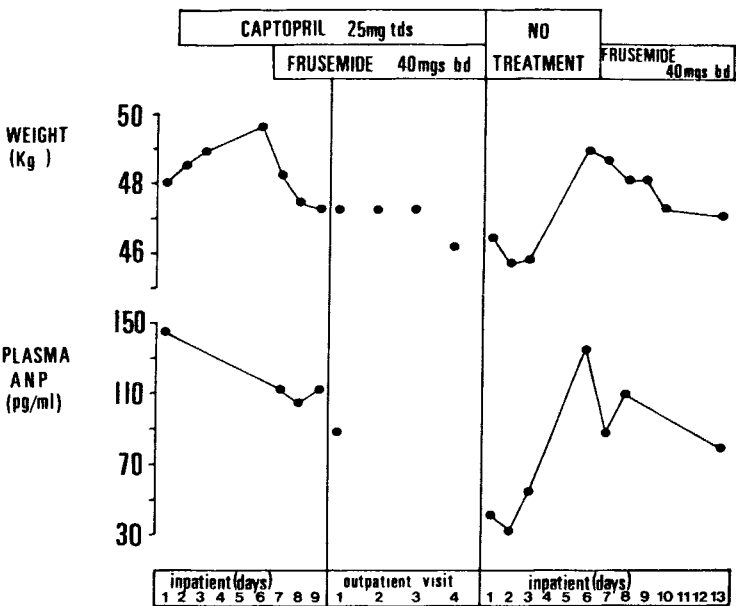
This finding suggests that improvement in heart failure results in a fall in plasma ANP levels, possibly via a reduction in volume overloading in the left ventricle.

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SIR,—Increased plasma levels of atrial natriuretic peptide (ANP) have been reported in congestive cardiac failure, the level appearing to depend on the severity of heart failure. The evidence for this comes from single measurements in different patients.¹⁻⁴ We report here measurements of plasma ANP in one such patient during periods of improvement and of worsening of heart failure.

A 56-year-old man was admitted with severe congestive heart failure following a myocardial infarction 8 months previously. He had mild hypertension and mild renal impairment. 2 months before



Plasma ANP levels in patient with heart failure, showing effect of treatment and withdrawal of treatment.

admission he had shortness of breath with nocturnal dyspnoea and ankle swelling. He had received no treatment for his heart failure during the previous 2 months. His supine blood pressure was 168/94 mm Hg, pulse 100/min in sinus rhythm; there was some ankle oedema. He was started on captopril alone with no improvement, and he continued to gain weight over the next few days. Frusemide was added and led to a loss of sodium and water and a weight loss of 2.1 kg with significant clinical improvement. He was followed up as an outpatient until, 10 weeks later, he had what was thought to be a possible allergic reaction to his drug therapy, with oedema of the lips and tongue. He was readmitted and the captopril and frusemide were stopped. His heart failure was well controlled, and he had no shortness of breath or oedema. Blood pressure was 166/88 mm Hg, pulse 88/min in sinus rhythm, and venous pressure was not raised. After cessation of therapy, he gained weight, his venous pressure rose, and he became short of breath on exertion. Frusemide 40 mg twice daily was reintroduced. He was discharged 9 days later feeling well with no shortness of breath and no oedema.

During the first admission, when his heart failure was severe, plasma ANP levels⁵ decreased as his heart failure improved, and they were almost normal⁶ by his second admission. After withdrawal of treatment plasma levels of ANP increased, and they fell again when frusemide was reintroduced (figure).

This man's plasma ANP varied with the severity of his heart failure and fell with treatment. Plasma levels of ANP may be useful as an index of the degree of heart failure and as a way of monitoring response to drug therapy.

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