changing needs of the patient. This is not care by committee, it is sensible use of expensive personnel, recognising that no single profession has all the answers all the time.

It is said that the nursing process is not researched. Professor Mitchell suggests that medicine provides a model of perfection in research. If this is the case, why has nobody given adequate time to research into the use of electroconvulsive therapy? Why did nobody insist on adequate research in the 1950s and '60s, when we undertook some fairly crude operations on the brain, all in the name of psychosurgery? There are other examples. Much of the thinking behind the nursing process has been tested by highly respected nurse researchers. Many studies published by the Royal College of Nursing show that individual care can affect outcome for the patient. Departments of nursing research are beginning to blossom in Britain. I hope that a centre will undertake a major piece of work related to models for nursing care; evidence is already available to support the nursing process.

Professor Mitchell also misunderstands the extended role of the nurse. In my own hospital nurses undertake nearly all intravenous work, but not one is in possession of a certificate signed by a doctor—the law does not demand this. What our own professions demand is that where a nurse extends her function there must be mutual agreement between the professions as to the shape and scope of the extended role.[4] When agreement is reached, the law demands that extension is ratified not by a consultant but by the employing authorities.

Professor Mitchell raises some interesting issues. I know that he echoes the fears and anxieties of some colleagues and it is essential that we maintain a dialogue. I can assure readers that relationships between nurses and doctors at the Royal Marsden are sound, the best I have experienced; the nursing process does not have to threaten these relationships. Perhaps there would be greater understanding if students of health care trained together for some first and final year studies and if we developed more joint projects at postgraduate levels.

Florence Nightingale's questions about relations between our professions remain relevant, but there are no clear cut answers. In my view genuine teamwork is the best model for the provision of health care, and the nursing process can do much to help the development of the nurse's contribution to the team. I look forward to many more years of help and cooperation from medical colleagues in the education of nurses; equally, I hope that the experienced nurse will continue to help the education of the junior houseman.

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Lesson of the Week

Acute schistosomiasis (Katayama fever)

P C STUIVER

Three clinical syndromes are recognised as manifestations of infection by the three main trematode species in human schistosomiasis (*Schistosoma mansoni*, *S haematobium*, and *S japonicum*)[1]: (1) swimmers' itch or dermatitis due to cercarial penetration of the skin; (2) acute schistosomiasis or Katayama fever, which coincides with larval maturation and migration and early oviposition; (3) chronic schistosomiasis, in which the lesions are the result of the inflammation and subsequent fibrosis caused by the schistosoma eggs.

Infection is acquired by exposure to water containing cercarias released by certain infected snails, the intermediate host. The cercarias swim until they come into contact with a suitable definitive host. Within 24 hours of the skin being penetrated by the cercarias some patients experience intense itching, sometimes followed by a papular rash (cercarial dermatitis). This may last for two or three days and is therefore almost never seen in Western Europe. The syndrome of acute schistosomiasis usually develops three to six weeks after the initial infection and may last for three to four months. Until recently[2 3] it also was an imported disease that was rarely seen. The numbers of people travelling to the tropics by air have greatly increased over the past decade, however, and tourists in particular are exposed to the risk of exotic diseases; hence it might be expected that this syndrome will be imported more often in the future. I describe three patients with acute schistosomiasis.

Until recently acute schistosomiasis was rarely recognised in Western Europe; it occurs almost exclusively in non-immune visitors to endemic areas in the tropics

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Case histories

Three Dutch patients—a 34 year old man (case 1), his 33 year old wife (case 2), and her 36 year old sister (case 3)—spent a holiday in Mali, west Africa, from 19 July to 13 August 1982. The husband became ill on 21 August with fever, rigors, sweating, headache, backache, myalgia, arthralgia, unproductive cough, urticaria, and loss of weight. There was no history of cercarial dermatitis. The intermittent fever lasted for five weeks and the urticaria for one week; the other symptoms persisted for almost 15 weeks, the cough being most exhausting. His wife became ill on 22 August, and her sister on the 21st. Both gave a history of a distressing itch after swimming in Mali. Neither could remember the exact date. Both had the same symptoms as case 1, the fever and cough, however, being less severe.

The sister had a short period of facial oedema. Physical examination elsewhere four weeks after leaving Mali and later at our hospital did not show anything abnormal. Chest *x* ray appearances were normal. Malaria, typhoid fever, and so on were ruled out.

The table lists the most relevant laboratory data. Twelve weeks after leaving Mali eggs of *S mansoni* and *S haematobium* were found in the stools of all three patients. The numbers of eggs in cases 1 and 2 were very small, but in case 3 numerous eggs of both types were easily detectable. Eggs were not found in the urine. Acute schistosomiasis was diagnosed. The illness developed three to four weeks after exposure and the acute disease lasted for a total of three and a half months.

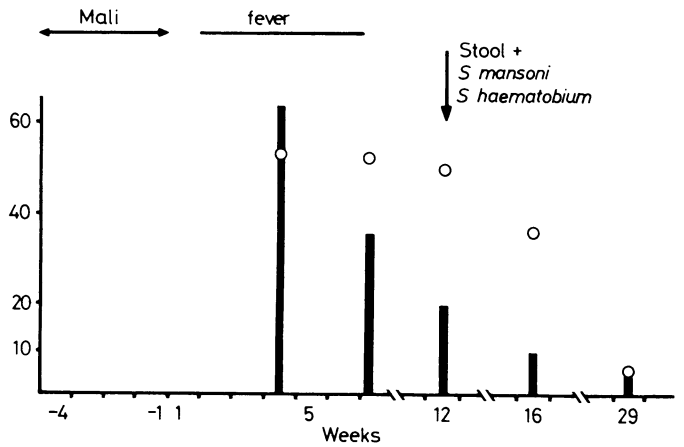
Results of laboratory studies four, 12, and 29 weeks after leaving Mali. (Studies at four weeks done elsewhere)

	Case 1 (man aged 34)			Case 2 (woman aged 33)			Case 3 (woman aged 36)		
Week No	4	12	29	4	12	29	4	12	29
Sedimentation rate (mm in first hour)	63	19	5	39	21	16	56	16	10
Eosinophils (%)	54	51	5	38	30	2	16	13	2
Eggs:									
Stool *S mansoni*	ND	+	−	ND	+	−	ND	+	−
Stool *S haematobium*	ND	+	−	ND	+	−	ND	+	−
Urine	ND	−	−	ND	−	−	ND	−	−

ND = Not done.

Maturation was assumed to have been completed and the adult worms established in their final habitat by 16 weeks. Each patient then began treatment with praziquantel (Biltricide). Except for some abdominal discomfort no side effects or serum sickness like symptoms were noted. When seen at 29 weeks haematological values had returned to normal in all patients and schistosoma eggs were no longer detectable.

The figure shows the course of the disease in the husband (case 1), who was the most severely affected. The erythrocyte sedimentation rate paralleled the clinical severity very closely. The eosinophil count at week 4 was 54% (absolute count $10{\cdot}0 \times 10^9$/l) and had declined to 37% (absolute count $4{\cdot}5 \times 10^9$/l) at week 16 before treatment was instituted.



Course of acute schistosomiasis in case 1. ■ = Erythrocyte sedimentation rate (mm in first hour). ○ = Eosinophils (%).

Comment

Acute schistosomiasis is rarely recognised and occurs almost exclusively in non-immune visitors to endemic areas. The clinical manifestations may vary considerably from general malaise to a very protean and severe illness. The most common findings are spiking fever, profuse sweating, abdominal pain, myalgia, arthralgia, diarrhoea, dry cough, loss of weight, hepatomegaly, splenomegaly, urticaria, and swollen eyelids. The fever often occurs in the late afternoon or at night. Katayama fever is the term used to describe this prolonged fever at the onset of the infection. It was first observed in *S japonicum* infection (Yangtze River fever). The acute phase of schistosomiasis is frequently confused with other feverish diseases. It simulates typhoid fever, malaria, salmonellosis, or bacillary dysentery. Eosinophilia, although not specific, is a conspicuous and constant feature which should be the clue to a correct diagnosis. In the prepatent period, before eggs are being excreted in stool or urine, some immunodiagnostic tests may detect antischistosome antibodies, indicating recent infection.[4 5] Definite diagnosis, however, is based exclusively on finding the typical eggs in the stool or urine.

After penetration through the skin the organisms migrate via the venous or lymphatic system to the lungs and are then carried in the systemic circulation to the liver, where they mature and move to their final habitat in the veins of the intestines or urinary bladder. During this period of migration and maturation there is an intense allergic response to the parasite, which is suppressed as the infection becomes chronic. That symptoms of acute schistosomiasis occur as early as three weeks after infection is strong clinical evidence that the disease is initiated by schistosoma stages before oviposition. Many clinical and immunological manifestations of acute schistosomiasis are similar to those of serum sickness, suggesting that immune complexes are important in the pathogenesis of the acute disease.[1 4]

Generally the severity of illness varies with the intensity of infection as measured by faecal egg counts.[6] In some patients, however, heavy infection may not be necessary for severe acute disease, as shown by case 1.

It is important to recognise this syndrome because after the acute stage general health gradually improves and the patient appears to be cured, particularly in the absence of localising signs as in the early chronic stage of schistosomiasis mansoni. Effective treatment will halt the disease if instituted early.

Antischistosomal drugs probably have no effect on the course of the acute illness.[4] Arguably, these drugs, even if effective, should not be given during that period, because of the possible sudden release of antigens that are believed to induce the acute syndrome. Steroids have been tried but proper investigation is hampered by the few patients available for a clinical trial.[4]

Praziquantel, the newest drug in the treatment of schistosomiasis, is highly effective against all human schistosome species. It can be given in a single oral dose of 40 mg/kg body weight for *S mansoni* and *S haematobium*; for *S japonicum* the present recommended regimen is 2×30 mg/kg at an interval of four hours in one day or 3×20 mg/kg in one day. Side effects are generally mild, such as abdominal pain, nausea, diarrhoea, and headache.[7]

The three cases of acute schistosomiasis described here are an example of the impact of mass tourism on the pattern of imported tropical diseases.

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