Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

sound to date. A further 310 operations have been performed in the years 1975–79, inclusive, with no recurrences to date.

These results confirm the clinical efficiency of the Shouldice operation and support our earlier enthusiastic recommendation of the technique.

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SCANDINAVIAN EPIDEMIC NEPHROPATHY AND KOREAN HÆMORRHAGIC FEVER

SIR,—Last year in *The Lancet* Svedmyr and Lee and their colleagues[1,2] described antibodies to the Korean hæmorrhagic fever (KHF) agent in sera from patients with Scandinavian epidemic (endemic) nephropathy (SEN). Significant titre rises were recorded in several patients. These workers used an indirect immunofluorescence method[3] with lung sections from infected rodents *(Apodemus agrarius Coreæ)* as antigen. Recently one of us (G.R.F.) successfully propagated the KHF agent on A-549 human lung carcinoma cells, and a spot slide indirect immunofluorescence method has been developed for the detection of antibodies to KHF virus. This method was used in a prospective study of patients with SEN, and we report here results from our first 13 patients who fell ill during the latter half of 1978.

KHF ANTIBODY TITRES (RECIPROCAL) IN PATIENTS WITH SEN AND IN CONTROLS

Total study group	SEN (n=13)	Controls (n=40)
No. of sera	44	40
Max. titre (median and/or range)	160 (40–2560)	<20
Seropositive patients	13/13	0/40
⩾4-fold titre rise	5/13	. .
⩾4-fold titre drop	2/13	. .

All patients lived in the county of Västerbotten in northern Sweden where SEN is prevalent, and they had been referred to Umeå University Hospital. Patients were included if they fulfilled clinical criteria of SEN.[4] All patients typically had an initial oliguric phase accompanied by high serum creatinine levels followed by a polyuric phase. All the patients had suggestive evidence of indirect contact with small rodents a few weeks before symptoms appeared. Two to five serum samples were drawn from each patient at different times, 4–319 days after the onset of the illness.

Control sera were obtained from 40 patients with other illnesses who were similar in age to the SEN patients and lived in the same county.

Patients' sera were stored at −70°C and control sera at −25°C until analysis, except during the 36 h transport on ice to Fort Detrick in the United States. Sera were coded before assay.

All 13 SEN patients had KHF antibodies, while control sera were negative. 7 patients showed significant changes in titres (see table). In no case did the antibodies disappear. 5 patients investigated more than six months after the acute disease still had an antibody titre of 80 or more. In 1 patient no antibodies were detected in the first serum, taken on the fourth day of symptoms. In all the others the first serum was taken on days 5–12. This early presence of antibodies accords with the findings of Lee et al.[2] The antibody titres recorded with our spot slide method are of the same order of magnitude as those recorded with the lung section method.[1,2]

Our results support recent findings[1,2] indicating a close antigenic similarity between the KHF virus and the agent causing SEN. The fact that age-matched controls from the same endemic area as that of the SEN patients were seronegative and the finding of apparently long-lasting antibody titres after SEN indicate that subclinical SEN with humoral immune response is rare.

Finnish workers,[5] using Lee immunofluorescent technique,[3] have found the SEN antigen in frozen sections of bank vole *(Clethrionomys glareolus)* lung tissue. Thus it is now possible to compare directly the two antigens—i.e., that associated with SEN in infected bank vole lung and KHF antigen in infected *A. agrarius Coreæ* lung or in infected A-549 cells. We now have 6 mm diameter spot slides and KHF infected A-549 cells in the laboratory at Uppsala and hope to be able to collaborate with the Finnish group in a joint effort to determine the degree of antigenic relatedness of the two agents.

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CARDITIS ASSOCIATED WITH CORONAVIRUS INFECTION

SIR,—Symptomatic myopericarditis is not uncommon in enterovirus infections and electrocardiographic changes suggestive of myocardial damage are often seen in association with mumps and certain other viral infections. Coronaviruses normally cause only mild upper respiratory infections in man.[1] We describe here a case of carditis related to human coronavirus infection.

The patient was a 43-year-old previously healthy man. Early in January, 1980, he had an upper-respiratory-tract infection which turned into prolonged fever, tiredness, and, later on, chest pain. On Jan. 9 chest X-ray revealed an enlarged heart and he was admitted to hospital. Over the following fortnight heart failure ensued. The ECG showed raised ST-segments and inverted T-waves, and a soft apical systolic murmur became audible. Pleural and pericardial effusion was noted on X-ray and echocardiography. Continuous fever and high erythrocyte sedimentation rate (120 mm/h) suggested bacterial infection but repeated blood cultures for bacteria were negative and there was no response to intravenous penicillin G. Clinical recovery began at the beginning of February, and on March 19 both physical and laboratory investigations suggested complete recovery although the patient still complained of fatigue.

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Significant increases in coronavirus OC43 antibodies were demonstrated in blood specimens during the disease followed by a decrease during convalescence. Complement fixation (CF) and radial hæmolysis-in-gel (HIG[2]) gave parallel results:

Date	*CF titre*	*HIG diameter (mm)*
Jan. 15	8	10
Jan. 23	128	15
Feb. 4	64	14
March 19	32	12

No changes in CF antibody titres could be found against adenovirus, mumps, measles, influenza A and B, parainfluenza types 1 and 3, Coxsackievirus B5, polio type 3, respiratory syncytial virus, herpes simplex, varicella zoster, cytomegalovirus, *Mycoplasma pneumoniæ, Chlamydia trachomatis,* or *Toxoplasma.*

We have evidence (unpublished) to suggest that coronaviruses can be associated with diseases more severe than the common cold and it now seems justified to investigate their potential for causing carditis.

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MULTIVITAMIN PROPHYLAXIS AS A CAUSE OF NEURAL TUBE DEFECT

SIR,—In the context of periconceptual vitamin supplementation to prevent neural tube defects (NTD) your correspondents have raised the question of possible teratogenic effects of vitamins (vitamin A has long been used experimentally to produce NTDs). It seems worth pointing out that there is a case on record of an anencephalic fetus, aborted at 17 weeks, associated with maternal megavitamin therapy for psychiatric reasons during pregnancy.[1]

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Commentary from Westminster

Complaints about Clinical Judgment

POLITICIANS and doctors look set to agree upon a compromise on the vexed issue of how to investigate patients' complaints against alleged clinical misjudgment. But the compromise may not satisfy everyone at Westminster. An agreement would, however, effectively end a year's wrangling over the role the office of the Health Ombudsman should play in examination of complaints about the treatment offered by doctors. It was the powerful back-bench Commons select committee overseeing the Ombudsman's work that suggested he should be empowered to investigate clinical matters, rather than simply covering administrative complaints against health authorities. The suggestion found ready support among M.P.S, with some 80 members putting their names to a Commons motion reiterating the select committee's advice.

The Social Services Secretary, Mr Patrick Jenkin, took the line that nothing could be accomplished in this area without the agreement of the medical profession, and he invited the Joint Consultants Committee to make their own proposals. The system submitted by the J.C.C. found little favour at Westminster. The consultants proposed starting with informal discussions between complainant and the doctor complained against. If these did not solve the problem, it should be referred to the regional medical officer. Failing a resolution then, the patient would be offered an investigation by two independent consultants, arranged by the regional medical officer. One investigator would be named by the appropriate Royal College; the other would be in the same specialty as the doctor complained about. The doctor whose treatment was under investigation would have the right to veto either or both of the proposed investigators, though the J.C.C. has not elaborated any specific grounds on which the veto should be based. "If no satisfactory opinion could be achieved, there would be nothing more that the second opinion could offer," runs the text of the J.C.C.'s submission. "No formal report would be submitted, but the regional medical officer would be informed in writing that the patient appeared to be satisfied or not to be satisfied following the consultation with the second opinions."

This formula proved totally unacceptable to most M.P.S, and there was widespread support for Mr Jack Ashley (Lab.) when he described the proposals in the Commons as "cheek". Introducing a private Bill under the ten-minute rule, Mr Ashley said the J.C.C. was making "an incredible proposal for seeking to alleviate the anxieties of people with legitimate fears". It amounted to saying "shut up or sue" to the patient. The whole J.C.C. scheme, he went on, was "a travesty of justice, designed simply to get doctors off the hook". Mr Ashley's Bill, to give the Ombudsman the powers suggested by the select committee, is destined for the Commons dustbin, along with the great mass of private Bills. But that is a result of lack of spare legislative time, not lack of sympathy from colleagues. Mr Jenkin made the position clear when he rejected the J.C.C. scheme, writing to the J.C.C. chairman: "there are several features of the proposals which I find difficult to accept, and which would be difficult to defend in Parliament. The most important of these are that the consultant concerned would have sole responsibility for deciding how a complaint should be initially handled; that the consultant could, if he wished, effectively prevent the proposed machinery for an independent review from being brought into operation; and that it is not clear how the proposals would operate if the 'second opinions' should conclude that there are grounds for criticism of the consultant concerned. I also see difficulty if, as I read the proposals, there would be no role for the health authority." In his letter Mr Jenkin spoke of mounting pressure in Parliament for a lasting solution.