Q fever in Maritime Canada

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Only nine cases of Q fever were recorded in Canada in the 20 years prior to 1978. In the 18 months from August 1979 to January 1981 the disease was diagnosed serologically in six patients from the Maritime provinces. All were epidemiologically unrelated and none had been exposed to animals. Five had pneumonia and one had chronic Q fever with probable prosthetic valve endocarditis. Three of the five pneumonia patients presented with signs and symptoms of an acute lower respiratory tract infection and were indistinguishable clinically from other patients with atypical pneumonias. The other two with pneumonia presented with nonresolving pulmonary infiltrates and complained of decreased energy. Four of the five pneumonia patients responded well to treatment with erythromycin; the fifth required two courses of tetracycline. The patient with chronic Q fever had a large amount of cryoglobulins in his serum and evidence of immune complex disease. These cases indicate that Q fever should be considered as a possible cause of atypical pneumonia in Canada.

Seulement neuf cas de fièvre Q ont été enregistrés au Canada dans les 20 années antérieures à 1978. Au cours des 18 mois de août 1979 à janvier 1981 cette maladie a été diagnostiquée sérologiquement chez six patients des provinces maritimes. Aucun rapprochement épidémiologique n'a été établi et aucun patient n'avait été exposé aux abbatoirs. Cinq souffraient de pneumonie et un avait une fièvre Q chronique avec endocardite valvulaire prosthétique probable. Trois des cinq patients atteints de pneumonie montraient les signes et symptômes d'une infection aiguë des voies respiratoires inférieures et n'avaient aucune particularité clinique les distinguant d'autres patients souffrant de pneumonie atypique. Les deux autres patients ayant une pneumonie présentaient des infiltrats pulmonaires tenaces et se plaignaient d'un manque d'énergie. Quatre des cinq patients souffrant de pneumonie ont bien répondu à un traitement à l'érythromycine, le cinquième nécessitant deux cures à la tétracycline. Le patient atteint de fièvre Q chronique avait de grandes quantités de cryoglobulines dans son serum et montrait des signes de maladie des complexes immuns. Ces cas indiquent que la fièvre Q doit être considérée comme une cause possible de pneumonie atypique au Canada.

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The term Q (or query) fever was coined by Derrick[1] in 1937 as a result of his investigation of a febrile illness that had affected 20 of 800 employees of a large Brisbane meat works. During study of another nine patients he isolated an organism that was identified as rickettsial by Burnet and Freeman[2] in 1937. Almost simultaneously Davis and Cox[3] isolated an organism in the United States from ticks (*Dermacentor andersoni*) and named it *Rickettsia diaporica*. The two organisms were subsequently shown to be identical and are now known as *Coxiella burnetii*. Infections due to this organism have been recognized worldwide.[4]

In 1956 Marc-Aurèle and associates[5] described the first case of Q fever in Canada; the first outbreak occurred that year in a group of 62 slaughterhouse workers in Princeville, PQ.[6] Since then there have been very few reports of Q fever in Canada.[7-10] Indeed, an editorial comment in the *Canada Diseases Weekly Report* stated that only nine cases of Q fever had been reported in the 20 years after that first outbreak.[11]

In the 18 months from August 1979 to January 1981 we diagnosed six cases of Q fever — five in patients from Nova Scotia and one in a patient from Prince Edward Island. In this report we discuss the diagnosis of Q fever, outline its course in these six patients and summarize its treatment.

Materials and methods

Four of our patients (nos. 1 to 4) were identified as part of a study of atypical pneumonia. Blood samples were taken from all six patients during the acute and convalescent stages of their illness and tested for antibodies to *Legionella pneumophila* serogroups 1 to 4, *Chlamydia*, *Mycoplasma pneumoniae*, adenovirus, and respiratory syncytial, influenza A and B, and parainfluenza 1, 2 and 3 viruses, as previously described.[12]

Titres of complement-fixing (CF) antibody to *C. burnetii* were determined with the use of phase II organisms (standards laboratory, Central Public Health Laboratory, London, England) as antigens. Titres of antibodies to individual phase I and II antigens were determined with a microagglutination test as follows:

The serum was inactivated at 56°C for 30 minutes, duplicate 0.025-ml aliquots of a 1:8 dilution were pipetted onto microtitre plates, and serial twofold dilutions were prepared with 0.85% saline. Phase I antigen (0.025 ml) was added to one row of dilutions and phase II antigen (0.025 ml) to the other row. Positive and negative controls were included with each run. Serum and antigen were mixed and incubated in a moist environment at room temperature (approximately 21°C) for 24 hours. Antigens and control sera were supplied by Dr. R.N. Philip, Rocky Mountain Laboratory, Hamilton, Montana.

Cryoglobulin preparation and analysis

Each blood sample (10 ml) was collected in a plain glass tube prewarmed to body temperature, allowed to clot and kept at 37°C until clot and serum separated. Aliquots (2 ml) were placed in three glass tubes and one Wintrobe tube; one glass tube was kept at 37°C (control) and the other three tubes were kept at 4°C for up to 7 days. The result of the test for cryoglobulins is positive if a whitish precipitate is present in the bottom of the tubes at 4°C and disappears when the tube is warmed to 37°C.

After 7 days the Wintrobe tube was spun in a refrigerated centrifuge for 10 minutes at 750 × *g*. The amount of precipitate was measured and reported as a percentage of the total volume. The precipitate was then washed six times in cold (4°C) saline, resuspended in 0.5 ml of saline and dissolved at 37°C. The components of the cryoprecipitate were determined by placing 10-μl aliquots in the central wells of two Ouchterlony diffusion plates (Hyland Laboratories, Deerfield, Illinois) and the following specific antisera in the peripheral wells: anti-IgG, anti-IgM, anti-IgA, anti-κ chain, anti-λ chain (Atlantic Antibody, Scarborough, Maine), anti-C1q (the first component of complement), anti-C3 (the most abundant and important component), anti-α_1-lipoprotein, anti-β-lipoprotein and antihuman antiserum (Behringwerke, Marburg, West Germany). The plates were placed in a moist chamber at 37°C for 24 hours and examined for the presence of a precipitant arc with a calibrating viewer (Transidyne General Corporation, Ann Arbor, Michigan). The antihuman antiserum was used to ensure that the cryoprecipitate had not been lost in the washing process.

Case reports

Case 1

A 55-year-old woman had become ill, with a throbbing headache on both sides of her forehead, nausea and fever, 4 days before she was admitted to hospital. Two days before admission she had begun to have chills and a cough that produced a brownish-green tenacious sputum. Amoxicillin had no effect, and she was admitted to hospital, where she was noted to be moderately ill. Her oral temperature was 39.4°C and her respiratory rate was 26/min. Dullness to percussion and rales were present over the upper lobe of the right lung. A lumbar puncture revealed normal cerebrospinal fluid. A chest roentgenogram showed an infiltrate in the middle and lower lobes of the right lung (Fig. 1). She was still febrile 48 hours after admission, and progression of her pneumonia was evident. Erythromycin therapy was instituted, and within 36 hours she was afebrile.

Laboratory data at the time of admission for this and the other five patients are shown in Table I. At 60 days after the onset of symptoms the titre of CF antibody to *C. burnetii* was 1:32 768; on day 88 it was 1:131 072. Microagglutination tests gave antibody titres of 1:512 and 1:1024 against phase II antigen and 1:64 and 1:32 against phase I antigen on these 2 days. She had not been exposed to animals.

Case 2

A 67-year-old man with rheumatoid arthritis who had been treated with prednisone (30 mg/d) for several years noted a dull headache and "sore eyes", then a day later began to vomit and to have diarrhea and abdominal pain. On the third day rigors and a productive cough appeared, and on the fourth day he was admitted to a local hospital febrile and in hypovolemic shock. He received fluids, penicillin G and clindamycin, and was transferred to our hospital on the seventh day, at which time he was afebrile but critically ill. His respiratory rate was 30/min and he had marked dyspnea. Bronchial breathing, rales and rhonchi were present on the right side, with signs of a pleural effusion at the base of the right lung. The following blood gas values were obtained while he was breathing 40% oxygen: pH 7.23, partial pressure of carbon dioxide 45 mm Hg, partial pressure of oxygen 56 mm Hg, bicarbonate level 19

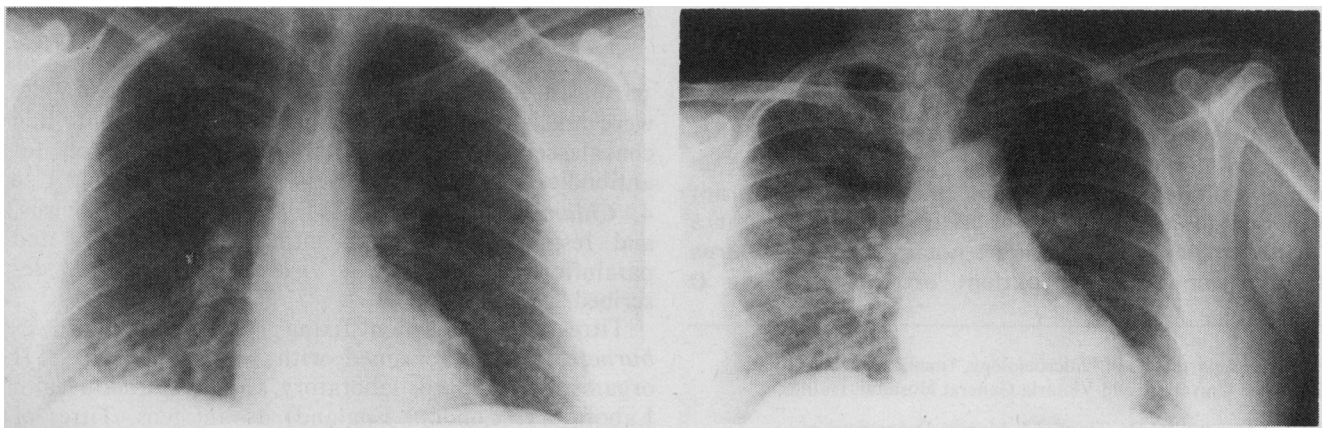

FIG. 1—Between time of admission to hospital (left), 4 days after onset of illness, and 3 days later (right), infiltrate in right lung of patient 1 increased in density and progressed from lower and middle lobes to upper lobe.

mmol/l and base excess −9 mmol/l.

Assisted ventilation was required for 4 days. Erythromycin, 500 mg administered intravenously every 6 hours, was added to his antibiotic regimen, and his condition gradually improved. *Enterobacter* bacteremia occurred secondary to infection of an arterial line. On the 13th hospital day he was discharged back to his local hospital. *C. burnetii* phase II antibody titres determined by the microagglutination test on days 7 and 15 after the onset of symptoms were 1:16 and 1:256 respectively. There was no CF antibody to *C. burnetii*. Fourfold rises in the titre of CF antibody to respiratory syncytial virus (from less than 1:8 to 1:128) and herpes simplex virus (from less than 1:8 to 1:64) occurred between days 7 and 24 of his illness. Stool cultures were negative for *Salmonella*, *Shigella*, *Yersinia*, *Campylobacter* and viruses. Herpes labialis was evident, and herpes simplex virus was isolated from his oral secretions.

Case 3

A 47-year-old man became ill, with abdominal pain, diarrhea and vomiting, 1 day after eating refrigerated Chinese food. Two days later a fever and a cough that produced yellow sputum developed. These symptoms progressed, and on the seventh day of the illness he was admitted to a local hospital, where a temperature of 40°C, a respiratory rate of 24/min and bilateral basal rales were noted. Tests of his serum showed the following levels: glutamic oxaloacetic transaminase 167 (normal 8 to 29) IU/l, glutamic pyruvic transaminase 69 (normal 1 to 41) IU/l and total bilirubin 60 (normal 0 to 16) μmol/l. A chest roentgenogram revealed an infiltrate in the lower lobe of the left lung. Erythromycin was given intravenously starting on the third hospital day, and within 48 hours he was afebrile. Stool cultures were negative for *Salmonella* and *Shigella*. Titres of CF antibody to *C. burnetii* on days 7 and 21 were less than 1:8 and 1:1024 respectively.

Table I—Laboratory data for six patients with Q fever at the time of admission to hospital*

Measures	Patient no. 1	2	3	4	5	6
Hemoglobin level, g/dl (normal for males 13–17.5, for females 11.5–15.5)	14.1	12.9	15.0	14.9	15.0	10.8
Leukocyte count, × 10^9/l (normal 4.5–10.5)	9.6	13.3	11.7	11.0	6.4	4.2
% polymorphonuclear leukocytes	84	ND	72	74	49	73
% lymphocytes	8	ND	17	16	44	22
Erythrocyte sedimentation rate, mm/h (normal for males 0–5, for females 0–7)	80	ND	55	75	9	35
Serum glutamic oxaloacetic transaminase level, IU/l (normal 8–29)	42	ND	167	31	24	85
Serum glutamic pyruvic transaminase level, IU/l (normal 30–104)	30	ND	69	56	ND	90
Serum alkaline phosphatase level, IU/l (normal 30–104)	ND	ND	ND	68	74	558
Total serum bilirubin level, μmol/l (normal 0–16)	ND	ND	60	30	12	ND

*ND = not determined.

Case 4

A 44-year-old man became ill, with fever and a severe headache, 1 day after cutting brush near his country cottage. Over the next 10 days he felt feverish and lethargic in the early afternoon and had a headache in the very early morning. On the 10th day a cough that produced yellow sputum began. The only physical abnormalities were petechial hemorrhages on the soft palate and rales throughout the lower lobe of the right lung. Roentgenographic examination revealed an infiltrate in this lobe that took 55 days to clear (Fig. 2). Shortly after he started taking erythromycin he became afebrile and the headache disappeared, although his cough and rales persisted for 100 days. Antibody titres could not be determined by the CF method because his serum was anticomplementary. The microagglutination test gave titres of antibody to *C. burnetii* on days 14 and 45 of 1:32 and 1:128 respectively against phase II antigen and less than 1:8 against phase I antigen.

Case 5

A 32-year-old man who worked as a dishwasher became ill with a cough and a tight feeling in his chest. Roentgenographic examination showed an infiltrate in the upper lobe of his left lung that persisted despite 2 weeks of therapy with tetracycline. Over the next month he noted increasing fatigue, dyspnea on exertion and intermittent fever. When admitted to our hospital he had rales in both lungs, but particularly in the left upper lobe (Fig. 3). On days 23 and 46 of his illness his titre of CF antibody to *C. burnetii* was 1:1024. Sucrose-gradient fractionation of the first serum sample showed that the IgM titre was 1:64 or higher. He was again given tetracycline — 500 mg orally every 6 hours for 2 weeks. His symptoms disappeared within 3 weeks and his pulmonary infiltrate cleared. He had not been exposed to farms or to animals.

Case 6

A 34-year-old man was admitted to our hospital 10 months after he had had a porcine mitral valve inserted

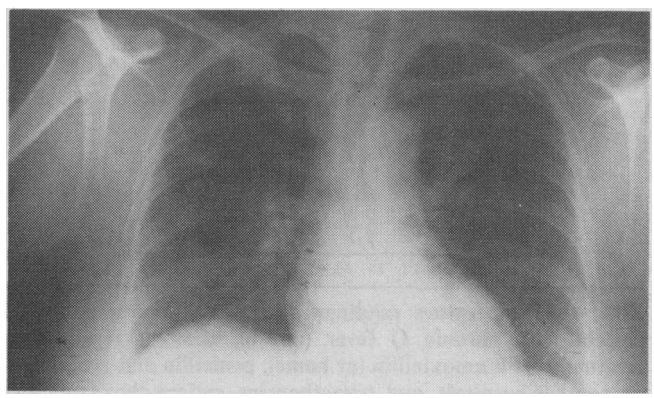

FIG. 2—Infiltrate in lower lobe of right lung of patient 4, 19 days after onset of illness.

because of mitral stenosis. His postoperative course had been uneventful and he had returned to work as a highway flagman 4 months later. However, 6 months after his operation he began to suffer fever, chills and vomiting that recurred every few days. One month later drenching night sweats started. His energy and appetite decreased, and he lost 11.3 kg in weight over 3 months. In the ninth postoperative month he began receiving amoxicillin therapy (Fig. 4), but shortly thereafter pain in the right upper quadrant of the abdomen developed and he was admitted to hospital. A laparotomy, performed because of suspected acute cholecystitis, revealed a normal gallbladder, a large firm liver and splenomegaly. A liver biopsy showed nonspecific hepatitis. He remained febrile, all cultures gave negative results, and antibiotic therapy was unsuccessful.

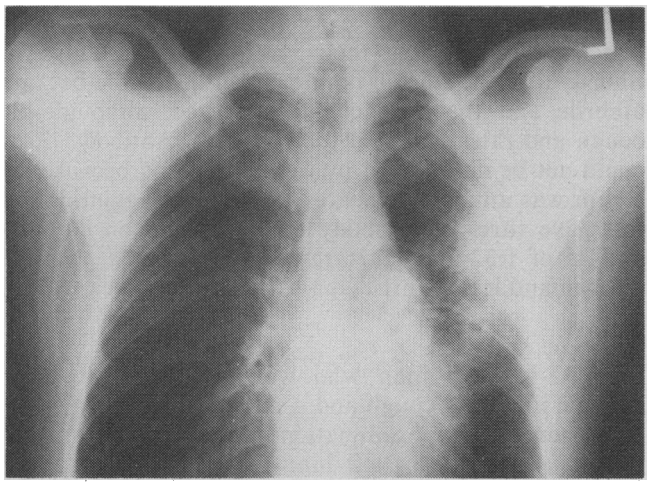

FIG. 3—Infiltrate in upper lobe of left lung of patient 5, 56 days after onset of illness.

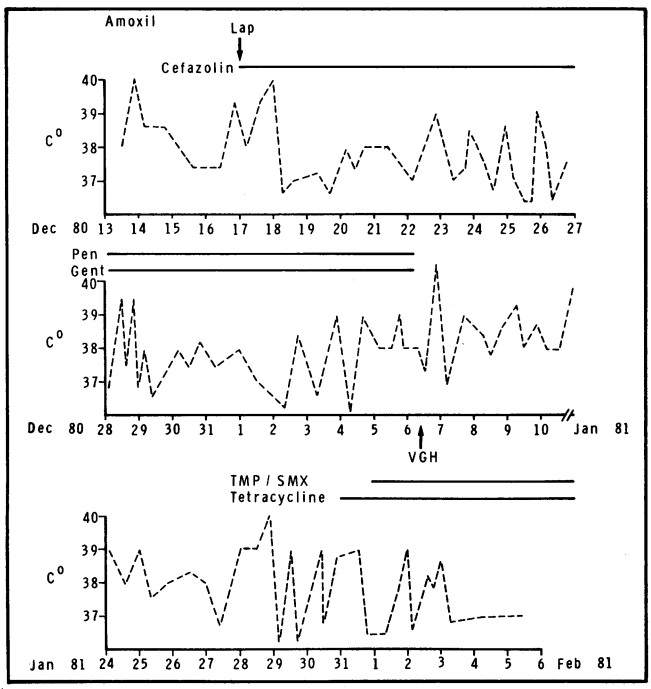

FIG. 4—Temperature readings during a 2-month period in patient with chronic Q fever (no. 6), showing response to treatment with amoxicillin (at home), penicillin and gentamicin (in another hospital), and trimethoprim–sulfamethoxazole and tetracycline (in our hospital). Lap = laparotomy; VGH = (admission to) Victoria General Hospital.

When admitted to our hospital he had an embolic lesion in his right fundus, rales at both lung bases and a pleural friction rub at the level of the right fifth intercostal space. There was no evidence of cardiac failure, although a grade 3/6 midsystolic murmur was heard over most of the precordium. His liver had a vertical span of 16 cm, and the spleen was palpable 4 cm below the left costal margin. There was marked clubbing of his fingers and toes, and splinter hemorrhages were present. The urine sediment contained erythrocytes, leukocytes and casts.

Extensive testing gave mostly negative or normal results. Two bone marrow biopsies revealed generalized hyperplasia but gave negative results on culture. A second liver biopsy showed nonspecific hepatitis. No vegetations were seen by echocardiography. Anemia and thrombocytopenia were present. Tests for cryoglobulins gave markedly positive results, and the total hemolytic complement titre was less than 1:4 (normal 1:12 to 1:24). The C3 and C4 levels were normal. Rheumatoid factor was detected in the serum. Tests for hepatitis B antigen and for antibody to cytomegalovirus and *Histoplasma capsulatum* gave negative results. Serum immunoglobulin levels were as follows: IgG 1740 (normal 565 to 1460) mg/dl, IgA 330 (normal 50 to 285) mg/dl and IgM 630 (normal 35 to 200) mg/dl. The titre of CF antibody to *C. burnetii* was 1:16 384 initially, then rose further, to 1:32 768. The titre of antibody to *C. burnetii* phase I antigen by the microagglutination technique rose from 1:64 on his third day in our hospital to 1:1024 2 months later. A test for cryoglobulins in a serum sample obtained 2 months after admission again gave strongly positive results; the cryoglobulin was composed of IgG, IgA, IgM, κ and λ chains, C1q and C3.

A diagnosis of probable Q fever endocarditis was made during his first month in our hospital, and therapy with tetracycline, 500 mg four times a day, and trimethoprim–sulfamethoxazole (TMP–SMX), two tablets (each containing 80 mg of TMP and 400 mg of SMX), was started. He became afebrile, the clubbing and hepatosplenomegaly regressed, and his complement levels returned to normal.

Discussion

The clinical features of Q fever are diverse. As was the case with most of our patients, the onset is sudden, with fever, myalgia and a severe headache.[13-15] Because of the headache with fever, clinicians often request a lumbar puncture, as in our case 1; the cerebrospinal fluid is invariably normal. Cough was present in 24% of the patients studied by Clark and collaborators[14] and in 46% of those described by Powell.[13] Pneumonia was present in 34% and 6% respectively of the patients in these two series. That five of our six patients had pneumonia probably reflects our selection criteria since patients had to have a pulmonary infiltrate for entry to our atypical pneumonia study.[12] Thus, we surmise that many other patients in Nova Scotia may have experienced a self-limited febrile illness that was Q fever over the 2 years in which our patients were identified.

Eshchar and colleagues[16] found that 6 of 119 patients with short-term fever had Q fever, and Carilli and coworkers[17] showed that 2 of 30 patients with chronic bronchitis had had a flare-up of their disease due to Q fever.

One of our patients (no. 2) had severe diarrhea as the presenting manifestation of Q fever. The frequency of diarrhea varies in the reported series, but recently Lim and Kang[18] described two patients with Q fever presenting with gastroenteritis.

Multiple round segmental consolidations of the density of ground glass are the most common findings in chest roentgenograms. Other findings include linear atelectasis, lobar or partial lobar consolidations, and a slight pleural reaction.[19] Resolution time has ranged from 10 to 70 days, with an average of 30 days.[19]

We had begun giving erythromycin to four of our patients with pneumonia before Q fever was diagnosed, and three had responded rapidly to this drug. While tetracycline is the antibiotic of choice in this illness,[20] D'Angelo and Hetherington[21] have described five patients with Q fever who responded favourably to treatment with erythromycin. Since the identification of Legionnaires' disease, erythromycin has become the antibiotic of choice for patients with atypical pneumonia of unknown cause. Because in our area at least some of these patients may have Q fever, it is reassuring to know that they will respond to this drug.

While most cases of Q fever are of sudden onset and usually subside within 2 to 3 weeks, some patients may have a fever for 4 to 9 weeks;[14] recovery in such cases is usually without permanent sequelae. However, a persistent form of Q fever has been recognized that is far more serious and usually indicates endocarditis.[22-27] All of the patients of Turck and colleagues[22] had valvular heart disease, and 14 of the 16 presented with features of infective endocarditis and negative results of blood cultures. Our patient with chronic Q fever (no. 6) had these features by the time he arrived at our hospital, although his initial symptoms had been fever and pain in the right upper quadrant of the abdomen. He underwent a laparotomy for suspected acute cholecystitis. Dupont and associates[28] described a patient with Q fever hepatitis who had also been thought to have acute cholecystitis, for which he underwent a laparotomy. Hepatitis occurs frequently during the course of acute Q fever; 78% of the patients in one series had abnormal results of liver function tests.[13] Indeed, four of our patients with acute Q fever had raised serum levels of liver enzymes. Patient 6, however, had hepatomegaly and abnormal results of a liver biopsy. Although we did not find the distinctive features of granuloma formation plus a fibrinoid ring described by Pellegrin and coworkers,[29] this patient had many of the other features of chronic Q fever, including hepatosplenomegaly, finger clubbing, persistent fever and sweats, anemia, thrombocytopenia, positive results of tests for rheumatoid factor and raised serum levels of immunoglobulins.[22-27] It is important to ask if patient 6 had Q fever endocarditis; the mortality of this condition is very high,[22] and many patients may need to have valves replaced. Indeed, Q fever endocarditis seems to occur preferentially on artificial valves.[22,26,27]

A titre of CF antibody to phase I antigen of 1:200 or higher is supportive evidence of chronic Q fever.[22] In nature and in laboratory animals *C. burnetii* exists in the "phase I" state; the organism reacts readily to guinea pig serum obtained late in convalescence (at 45 days) and only slightly to that obtained earlier (at 21 days). Repeated passage of phase I organisms in embryonated chicken eggs has led to gradual conversion to phase II. Most isolated strains have been found to contain a mixture of phase I and II antigens, with one or the other predominating. Further, there is no morphologic difference between the two antigens, although their buoyant density in cesium chloride and their affinity for hematoxylin and basic fuchsin differ.[30]

The duration and treatment of chronic Q fever are not well defined. Therapeutic regimens have included tetracycline and lincomycin,[22] as well as doxycycline and TMP–SMX.[23,25] It has been suggested that therapy may be monitored by serial measurements of antibody titres; levels of phase I antibodies may take up to 1 year to fall.[26]

Although cryoglobulins have been associated with a large number of infections, including viral, bacterial, fungal and parasitic,[31] their association with Q fever has not been specifically mentioned.

Finally, the question of the source of Q fever in Nova Scotia arises. None of our patients were farmers or had been exposed to animals, and there was no geographic clustering. The American dog tick, *Dermacentor variabilis*, is present in Nova Scotia.[32] *C. burnetii* infects ticks associated with a variety of rodents, cattle and sheep, and Hart[4] reported that in 1944 Derrick suggested that human infections were the result of inhalation of infected tick feces. The most common route of infection in humans, however, has been spread from infected cattle and sheep by inhalation of dust.[15] Unusual routes of infection have included spread by droplet spray from an infected patient and by exposure during an autopsy of an infected patient.[15] Exposure to infection in sheep and other animals during the course of research has also resulted in infections.[33,34] Whatever the source of infection, the finding of six cases since 1979 demonstrates that Q fever is comparatively common in Nova Scotia; it accounted for 4 of the 47 cases of atypical pneumonia that we have studied prospectively, some of which we described in the Journal last year.[12]

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Utilité de l'épreuve d'effort sur tapis roulant peu après un infarctus du myocarde

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Un programme de reconditionnement par la marche a été prescrit pour 130 patients à la suite d'une épreuve d'effort sur tapis roulant 3 semaines après un infarctus du myocarde. À 8 et à 12 semaines les patients ont subi une nouvelle évaluation. Le protocole est sécuritaire et permet de déceler l'angine, les arythmies et la dyspnée pendant l'exercice, évitant ainsi des délais thérapeutiques. On a mesuré la fréquence cardiaque et la tension artérielle systolique à la fin de chaque étape d'une épreuve et après 3 minutes de récupération.

Environ 75% des patients ont atteint le niveau énergétique cible des deux épreuves sous maximales (4 et 7 mets à 3 et à 8 semaines respectivement); un niveau de 7 mets permet à un patient de reprendre ses activités quotidiennes habituelles. Les résultats des épreuves de 3 et 12 semaines (celle-çi maximale) ont montré que la probabilité d'une capacité aérobique de 7 mets ou plus à 12 semaines est de 86% si l'épreuve de 3 semaines est complétée. Les observations cliniques seules n'avaient pas la même valeur pronostique 3 semaines après l'infarctus.

A program of reconditioning through walking was prescribed for 130 patients following an exercise test on a treadmill 3 weeks after a myocardial infarction. At 8 and at 12 weeks the patients again underwent an exercise test. The protocol is safe and permits the detection of angina, arrhythmias and dyspnea during the exercise, thus avoiding delays in treatment. The heart rate and the systolic blood pressure were measured at the end of each stage of the test and after 3 minutes of recuperation.

About 75% of the patients attained the target energy output of the two submaximal tests (4 and 7 mets at 3 and 8 weeks respectively); an output of 7 mets permits a patient to resume his or her usual daily activities. The results of the tests at 3 and 12 weeks (the latter a maximal test) showed that the probability of an aerobic capacity of 7 mets or greater at 12 weeks is 86% if the 3-week test is completed. Clinical observations alone did not have the same prognostic value 3 weeks after the infarction.

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La mobilisation précoce du patient après un infarctus du myocarde est maintenant acceptée,[1,2] car la reprise de l'activité physique permet d'éviter le déconditionnement[3] et facilite le retour aux occupations antérieures.

L'épreuve d'effort sur tapis roulant après un infarctus s'avère un bon moyen d'intervenir dans la reprise de l'activité et d'identifier les patients à haut risque. La valeur pronostique de certaines observations faites lors