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An increase in sudden unexpected cardiac deaths among young Swedish orienteers during 1979–1992

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Background Sixteen cases of sudden unexpected cardiac death, 15 males and one female, are known to have occurred among young Swedish orienteers from 1979 to 1992, of which seven cases occurred between 1989 and 1992. This is considered to be indicative of an increased death rate.

Results Histopathological evaluation showed myocarditis in a higher than expected proportion of cases. In one such case, which we studied before the sudden unexpected death occurred, the victim had suffered a *Chlamydia pneumoniae* infection verified by serology, and a nucleotide sequence was found in the heart and lung by means of the polymerase chain reaction (PCR) that hybridized with a probe specific for that organism. Male Swedish orienteers do not, however, seem to have an increased rate of exposure to this agent. No further sudden unexpected deaths among young orienteers have occurred over the past 3·5 years. At the beginning of that period, attempts were made to modify training habits and attitudes.
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Key Words: Sudden unexpected death, athlete, myocarditis, arrhythmogenic right ventricular dysplasia, *Chlamydia pneumoniae*.

Introduction

Orienteering, the task of finding the fastest track through terrain between defined checkpoints by means of map and compass reading in the shortest possible time, has for a long time been a popular sport in Sweden. At present, some 160 000 people of virtually all ages, corresponding to 2% of the entire population, pursue this sport at various levels of competition. A top segment of 3000 are ranked, 70% of whom are men, reflecting the male-to-female ratio within the sport. About 200 of the 3000 compete at the international elite level.

In the summer of 1992, we reported a case of *Chlamydia pneumoniae*-associated myocarditis and sudden unexpected cardiac death in a young Swedish orienteer[1]. At that time we were aware of five more sudden unexpected cardiac death cases during the previous few years among young orienteers in Sweden, all belonging to the ranked elite and in whom inflammatory lesions had been found in the heart at autopsy[1]. In previously published studies of sudden unexpected cardiac death in young athletes, myocarditis was the cause in about 10% of the cases[2].

In November, 1992, another young Swedish elite orienteer succumbed to sudden unexpected cardiac death, and a retrospective analysis disclosed additional cases in earlier years. This prompted the Swedish Board of Health and Social Welfare to arrange a meeting of health experts to discuss the current situation. The microbial hypothesis was actualized by the fact that Swedish orienteering had been previously struck by an epidemic of hepatitis B, comprising 564 jaundiced cases in 1958–63[3]. The virus was presumed to be transmitted through skin lesions on the legs, resulting from scratches from twigs and brushwood, and common washing tubs which were used after races. It was concluded at the meeting that, although *Chlamydia pneumoniae* had been detected in the myocarditic heart of one case, where tissue sampling had been performed in order to allow for such testing, a broad search for all possible

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Table 1 Key notes in the history of 16 young orienteers struck by sudden unexpected cardiac death. All but case number 9 were male. The mean age for all was 25·0 (SD 3·9) years and for the males 25·4 (SD 3·7) years

Case number	Age	Elite level	Died during exercise	Prior heart symptoms	ECG at 18 years of age[3]	Examination of the heart prior to death	ECG at examination prior to death	Pathology group[4]
1	19	Yes	<30 min[2]		ST-T changes	Yes	T negative	C
2	18	Yes	Yes		0	No		A
3	25	Yes[1]	Yes		0	No		C
4	27	Yes[1]	No	Tachycardia	Pre-excitation	Yes	Pre-exitation	C
5	28	Yes	Yes		0	No		B
6	28	No	Yes		0	No		C
7	26	No	Yes		0	No		A
8	32	Yes	Yes	Tachycardia	0	Yes	ST-T changes	B
9	19	Yes	Yes		Not done	No		C
10	29	Yes	Yes		0	No		B
11	27	Yes	No		0	No		A
12	24	Yes	Yes	Chest pain	ST-T changes	Yes	Minor changes	C
13	20	Yes	Yes		ST-T changes	Yes	T negative	A
14	27	Yes	Yes	Fainted during exercise	0	Yes	ST-T changes	C
15	27	Yes	<30 min[2]	Fainted during exercise	0	Yes	T negative	A
16	24	Yes	<30 min[2]		0	No		B

[1]These men were never ranked. The ranking system was introduced 1979.
[2]Death occurred within 30 min from the end of exercise.
[3]All Swedish men undergo a general medical examination, including ECG, at the age of 18 in connection with recruitment into the Swedish Armed Forces.
[4]Cases were classed into one of three groups (A, B or C). Group A; active myocarditis as the sole finding. Group B: arrhythmogenic right ventricular dysplasia-like alterations. Group C: remaining cases (see text).

causes and underlying conditions should be undertaken along with epidemiological studies. Furthermore, a peak-performing group within the orienteering elite was identified as a tentative risk group that would undergo in-depth medical examination. A total of 187 elite orienteers were subsequently examined at Uppsala University Hospital, and results are currently being compared to those of elite middle-distance runners and cross-country skiers having similar physiological profiles.

The Board of the Swedish Orienteering Federation decided to recommend that their elite members refrain from training and competition for a period of 6 months (December 1992–May 1993). No elite classes were included in any of the races organized during that period. A similar intervention had been imposed in connection with the hepatitis B epidemic 30 years earlier. This general intervention was deemed necessary as several of the deceased had reported no heart symptoms prior to death (Table 1). Elite orienteers, like other elite sportsmen, tend to participate in races even when suffering respiratory-tract infections. In orienteering, technical skills and careful map reading can, to some extent, compensate for an impaired physical performance.

The following is a brief report of results from the ongoing investigations.

Material

The present study group of 16 deceased orienteers consists of two subgroups, I and II. Subgroup I (case numbers 1–14) could be studied only retrospectively. These cases had previously been autopsied at different forensic medical centres, and variable amounts of formalin-fixed and paraffin-embedded tissues were available for re-evaluation of the cardiac histopathology. Serum had been saved in only three cases of subgroup I (numbers 10, 13 and 14), and kept at − 18 °C. The cases of subgroup II (numbers 15 and 16) were autopsied by us, and we could thus obtain ample tissues and serum, making a variety of analyses possible.

In addition, sera were collected for serology from 1790 members of the ranked elite, 1194 males and 596 females.

Methods

Epidemiology

In 1979 the Swedish Orienteering Federation introduced a ranking system of their members. Since then, records have been kept on all members, including the ranked elite and their individual ranking numbers. These records were used in the present study.

History

For all of the deceased, a history was retrospectively obtained through interviewing family members, coaches

and fellow orienteers. Furthermore, information was obtained from doctors' notes and hospital files from previous medical examinations. In addition, following the death of the 15th orienteer, a questionnaire was distributed to 316 highly active young members of the Swedish Orienteering Federation, including the man who subsequently died (case number 16), to gain information about their health status.

Serology

The sera available in five of the deceased cases were analysed for specific IgM and/or IgG antibodies to a variety of microbial agents by means of the following methods: enterovirus-IgM-SPRIST[4], enterovirus-IgM-RIA[5], and Coxsackie B1-5-IgM-RIA[6]; complement fixation tests (IgG) for influenza virus A and B, adenovirus, respiratory syncytial virus, *Mycoplasma pneumoniae*, parainfluenzavirus 1, 2 and 3, and *Chlamydia* group antigen; ELISA tests (IgG and IgM) for herpes simplex, herpes virus 6, cytomegalovirus, Epstein–Barr virus, Sindbis virus, Puumala virus, *Francisella tularensis, Borrelia burgdorferi, Coxiella burnetii* phases 1 and 2, *Brucella abortus*, and staphylococcal teichonic acid and alphatoxin; neutralization tests for antistreptolysin O and antistreptococcal DNase B; direct agglutination tests (IgG) and immunofluorescence tests (IgM+IgG) for *Toxoplasma gondii*, and, finally, micro-immunofluorescence IgG tests for *Chlamydia psittaci, Chlamydia trachomatis* and *Chlamydia pneumoniae*.

The 1790 sera from the ranked elite, and from 254 blood donors as controls, were analysed by means of micro-immunofluorescence tests for IgG antibodies to *Chlamydia pneumoniae*, and, for methodological comparison, to *Chlamydia trachomatis* and *Chlamydia psittaci*.

Microbiological examination

From cases 15 and 16 (subgroup II), a variety of tissues, including five different samples of myocardium, and samples of pericardium, coronary arteries, lung, bronchi, tonsil, thoracic lymph nodes, liver, intestine, pancreas, kidney, intercostal muscle, quadriceps muscle, blood, faeces, and urine were cultured for viruses by means of routine methods. Cell cultures of human lung fibroblasts, A-549, green monkey kidney and RD cells were used in triplicate. Routine bacterial cultures were performed from blood, airways, liver, spleen and kidney. Cultures for *Myocoplasma* were performed on samples from myocardial septum, pericardial fluid and lung, and for *Chlamydia* on samples from these locations, as well as from liver, spleen, kidney and several other tissues.

Culturing for *Chlamydia* was performed with homogenates of samples, which were inoculated on human lung cells (HL cells), incubated for 3 days, and stained with genus-specific monoclonal antibodies (Pathfinder, Kallestad) and species-specific anti-*C. pneumoniae* antibodies (Clone rr-402, Washington Research Foundation).

For polymerase chain reaction (PCR) tests of *C. pneumoniae*, DNA was extracted from homogenized tissue by digestion with Proteinase K, phenol/chloroform/isoamyl alcohol extraction and ethanol precipitation. Primers and cycle conditions have been previously described[7]. The extracted DNA was amplified with two different sets of primers. One of the primer sets amplifies a part of the 16S rRNA gene[7], whereas the other primer pair recognises a fragment of *C. pneumoniae* DNA which had been cloned[8]. For dot blot hybridization, probes were made by PCR amplification of the type strain TW 183 of *C. pneumoniae* with each set of primers and random primer labelling with digoxigenin (Boehringer Mannheim) according to the manufacturer's instruction. For a nested enterovirus PCR, the nucleic acids were extracted in a similar manner as described above. cDNA synthesis and the first PCR was performed with primers within the 5′ non-coding region according to Hyypiä[9]. The second PCR utilized internal primers corresponding to positions 274–293 and 410–430 of the Coxsackie B3 virus genome.

Histopathology

In cases 15 and 16 (subgroup II) macroscopical evaluation, as well as histopathology, were performed for virtually all organs in connection with our autopsies. Tissue samples were taken from various parts of the heart, including right and left ventricle septum, atria and coronary arteries. The cardiac histopathology was subsequently re-evaluated by a group of recognised cardiac pathologists along with cases 1–14 (subgroup I).

Investigation of hygienic routines and sanitation equipment

The hygienic routines and sanitation equipment were investigated in 100 of 771 clubs organized within the Swedish Orienteering Federation.

Results

Epidemiology

From 1979 to 1992 there has been, to our knowledge, 16 cases of sudden unexpected cardiac death among young Swedish orienteers, seven of which occurred between 1989 and 1992. All but two cases (numbers 6 and 7) belonged to the high-performing elite. Their mean age was 25·0 years (range 18–32 years), and all but one were male. We cannot exclude the possibility that additional sudden unexpected cardiac death cases among young orienteers may have occurred during the study period, especially during its early years.

From information obtained through the national registry of the Swedish Orienteering Federation, the

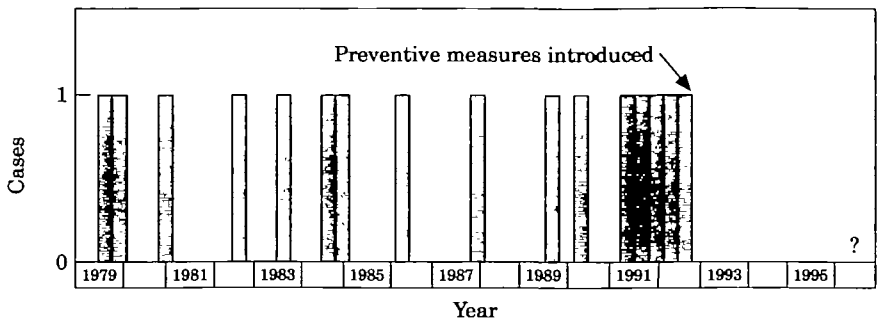

Figure 1 **Occurrence of sudden unexpected cardiac deaths in young Swedish orienteers 1979–1995 by year (n=16).**

average annual sudden unexpected cardiac death rate in ranked male orienteers was calculated to be one case per 2900 for the whole period, and one case per 1900 for the last half of the period. The reported sudden unexpected cardiac death rate among American sportsmen below the age of 30 is 1–2 per 200 000[2]. An increased mortality rate among young Swedish orienteers seems to exist and apppears to be increasing over the past few years (Fig. 1).

No accumulation of sudden unexpected cardiac death cases among the elite of other endurance sports in Sweden, such as cross-country skiing, running or cycling, could be disclosed through communication with the national leaderships of these sports.

History

Key facts from the histories of the deceased are summarized in Table 1. None of the cases had a positive family history of sudden unexpected cardiac death. All but two sudden unexpected cardiac deaths (cases numbers 4 and 11) occurred during or within 30 min of an exercise session. In all the cases, except in the female (case number 9), an ECG had been recorded, along with a general medical examination, at the age of 18 years in connection with the draft to the Swedish Armed Forces.

In four cases, the ECG at the recruitment to the Armed Forces was considered to deviate from normal, and these cases underwent a heart examination by the military. In one of these cases (case number 4), the ECG showed pre-excitation. This man had experienced at least one previous attack of tachycardia. Several years later, he experienced palpitations and sought two doctors' advice. His symptoms were considered to be of vegetative origin by one of the doctors, and Tietze's syndrome was suggested by the other. He died suddenly later, while abroad.

In another of these four cases (number 12), S-T interval and T wave changes were present in all chest leads at enrolment. It was noted that he had reported an episode of acute, transient chest pain during exercise 1·5 years before. At that time similar ECG changes had also been recorded. At a later check-up the changes were considerably less pronounced. He was assessed as having had previous myocarditis and was recruited into the Army. In the remaining two cases (numbers 1 and 13) S-T interval and T wave changes were found at the draft but these men reported no previous or current heart-related symptoms to the military doctor.

All four of these men were drafted. Three of them completed their military service and died one to several years later. One man (case number 1) died before entering the service.

In addition to the two above-mentioned men with symptoms prior to death (case numbers 4 and 12), three more men (case numbers 8, 14 and 15) had sought a physician's advice on at least one occasion due to heart-related symptoms. One of these men (case number 8) had experienced tachycardia when exercising and had undergone a medical examination, including ECG showing S-T interval and T wave changes; at an ECG exercise test he developed ventricular tachycardia, reverting to sinus rhythm after 1 min. He was prescribed beta-blocking medication. A subsequent exercise ECG test and echocardiogram were considered normal. Two months later he turned up at the emergency room because of a brief attack of tachycardia. The medical examination, including a resting ECG, was then normal. He was considered to have had myocarditis. He died while running 1 year after the first medical examination and 2 years after having first experienced tachycardia.

The two other men (case numbers 14 and 15) had fainted once during exercise. These episodes occurred 4 and 6 months, respectively, prior to death. The fainting episode lasted for about 1 min in case number 15, whereas, in case number 14, the man was out running alone when he fainted but could return home by himself although slightly mentally confused. He later recalled that he had felt dizzy while running in daylight, and when he later woke up lying on the ground it was dusk. Both men underwent a subsequent medical examination, the ECG showing minor S-T interval and T wave changes, as well as ventricular extra systoles in both cases. Both men performed an exercise ECG test showing ventricular extra systoles that disappeared with the higher loads. One of the men (case number 15) also underwent echocardiography that was considered virtually normal, as well as a 24-hour Holter-ECG showing 6% ventricular extra systoles. In this case, symptoms of a respiratory

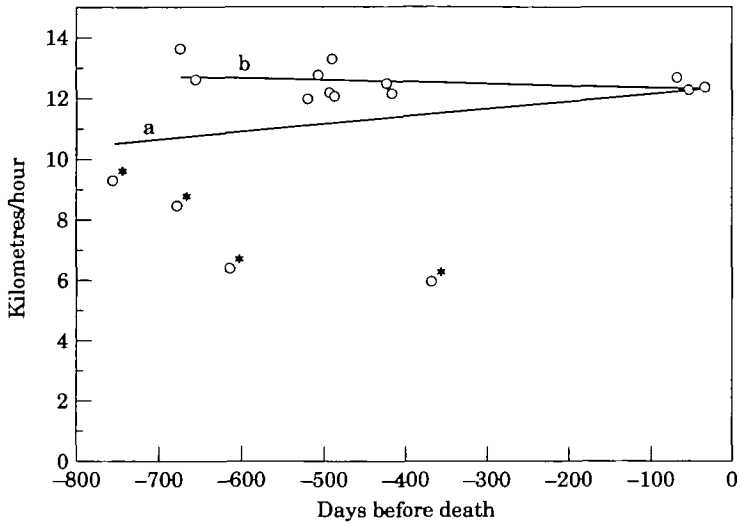

Figure 2 **No sign of impaired performance was seen even shortly before death in this case. Average running speed was calculated as the ratio of the length of the shortest distance between checkpoints and the total time used in orienteering. The observations had been noted in a diary. Linear regression lines are shown (a) for all recordings and (b) excluding four occasions of slower speed (marked with *) when the covered distance was longer because of inferior map reading.**

infection, including a month-long episode of bronchitis, appeared shortly afterwards, and he was prescribed a course of an antibiotics, probably penicillin. He was still advised to avoid strenuous exercise when he subsequently died within 30 min of a jogging session at a high altitude.

In the remaining nine cases (numbers 2, 3, 5, 6, 7, 9, 10, 11 and 16) no heart symptoms or signs could be disclosed retrospectively. One of these cases (number 16) had completed and returned a questionnaire regarding his health status about 4 months prior to his death, where he stated that he was healthy.

In none of the cases were signs of impaired physical performance found in the months before death. An example of this is given in Fig. 2. Some of them were placed among the first five in national competitions shortly before sudden unexpected cardiac death occurred.

Serology

IgG antibodies to *Chlamydia pneumoniae* were found in all the available sera from five of the deceased (case numbers 10 and 13–16) (Table 2). In four of these five cases the reciprocal titre was 512 or higher, which has been considered to indicate a current or recent infection[10]. In addition, IgG antibodies to *Toxoplasma gondii*, indicating previous infection with that agent, were found in three of these five cases (case numbers 10, 15 and 16). Antibody levels to the other tested agents were not significantly elevated. In case number 15 serology was performed in three different sera: one was obtained after an attack of fainting, another during convalescence after bronchitis, and the third at autopsy. High IgG levels of *C. pneumoniae* antibodies were found in all these sera, indicating a recent or ongoing infection with that agent.

Table 2 Prevalence of antibodies to **Chlamydia pneumoniae** *in orienteers and blood donors (percentages in parentheses)*

IgG reciprocal titre	Deceased orienteers (n=5)	Elite orienteers (n=1790)	Blood donors (n=254)
≥64	5 (100)	947 (53)	151 (59)
≥512	4 (80)	160 (9)	18 (7)

The newly collected sera from the 1790 elite orienteers and a control group of 254 blood donors were analysed for antibodies to *C. pneumoniae* (Table 2). No significant differences were found regarding exposure to this agent (frequency of IgG antibodies). This is in agreement with the results of Gnarpe *et al.*[11], who performed a similar study of sera collected in 1990. Furthermore, as in that study, no significant difference between sexes was found among the orienteers. In most other *C. pneumoniae* antibody prevalence studies, as with the blood donors of the present study, a lower prevalence has been recorded in females than in males of comparable ages[12].

Microbiological examination

All cultures and PCR for viruses, performed in case numbers 15 and 16 (subgroup II), were negative.

Similarly, cultures for *Mycoplasma* and *Chlamydia* were negative. Ordinary bacterial cultures showed insignificant findings. In case number 15, PCR with the primers directed to the rRNA gene of *C. pneumoniae* showed a discrete band of expected size in lung tissue, as well as in several specimens of the myocardial septum but not in specimens from several other organs. Furthermore, this PCR product hybridized to the *C. pneumoniae*-labelled probe. No amplification was achievable with the other *C. pneumoniae* primer set.

Histopathology

As a result of the re-evaluation of the histopathology of available heart tissues from the 16 cases, these were allocated into one of the three groups (A–C, Table 1) as follows:

Group A (five cases): Active myocarditis according to the Dallas criteria[13] was the sole finding (Fig. 3a).

Group B (four cases): Cases, where arrhythmogenic right ventricular dysplasia-like alterations were found as part of the picture were assigned to this group. In two cases (numbers 10 and 16), changes in the left ventricule were grave and similar to those in arrhythmogenic right ventricular dysplasia[14] (Fig. 3b) along with signs of active as well as healing myocarditis in one case (number 16) and probably of healed myocarditis in the other (number 10). Changes in the right ventricle were not diagnostic for arrhythmogenic right ventricular dysplasia in these cases. In the third case (number 5) the microscopic appearance was one of healing myocarditis, and the autopsy protocol described a macroscopic picture compatible with arrhythmogenic right ventricular dysplasia. In the fourth case (number 8), one out of three myocardial specimens was considered by appearance to originate from the right ventricle and showed serious arrhythmogenic right ventricular dysplasia alterations, whereas in the other specimens a picture of healed myocarditis was found.

Group C (seven cases): This group consisted of the remaining cases (numbers 1, 3, 4, 6, 9, 12 and 14). They could not be classified into one of the previous groups. In case number 1, the histopathological picture was compatible with hypertrophic cardiomyopathy in one out of three myocardial specimens, and the other two showed a picture of healed myocarditis. In case number 3 the available material was insufficient for diagnosis; fibrosis, fatty infiltration and hypertrophy were present. No degenerative changes were seen in case number 4 but an anatomical basis for pre-excitation had been found by the pathologist at autopsy. Case number 6 had been autopsied 6 days after death, and autolytic changes and fibrosis were found. In case number 9 early healing myocarditis was the sole histopathological finding but the macroscopic description was compatible with hypertrophic cardiomyopathy. In case number 12 pronounced histological alterations were seen in both ventricles, being not diagnostic for arrhythmogenic right ventricular dysplasia or hypertrophic cardiomyopathy. It was presumed to be, but was not diagnostic for, an earlier myocarditis.

No coronary artery disease, or anomalies, were reported in any of the 16 cases. In five cases active myocarditis with signs of concomitant healing was the sole finding. Histopathological myocarditis was found in altogether 12 of the 16 cases (75%), being unequivocal in ten cases (62%). Fibrosis was a conspicuous finding, suggesting a subacute to chronic condition.

Investigation of hygienic routines and sanitation equipment

The hygienic routines, as well as the sanitation equipment used in connection with training and competition over the country, were found to be of a high standard and not suspected of transmitting any communicable disease. After transmission of hepatitis B during the early 1960s the importance of high standards in hygienic routines during competitions were stressed. A thorough revision was then made and mobile sanitation units capable of handling large number of participants were developed.

Discussion

Young Swedish elite orienteers have evidently had an increased sudden unexpected cardiac death rate in recent years. Such a death rate did not occur within other endurance sports in Sweden during the same period of time. In one of two prospectively studied cases, who had suffered a prolonged respiratory tract infection prior to death, a nucleotide sequence that hybridized with a *Chlamydia pneumoniae*-specific probe was found by means of polymerase chain reaction in the myocarditic heart as well as in the lung, and antibodies to this agent were found in consecutive sera at levels indicating recent or current infection[10]. *C. pneumoniae* has previously been associated with myocarditis[15,16]. Until now, in our retrospectively studied sudden unexpected cardiac death cases, a role for *C. pneumoniae*, or a cross-reacting organism, has not been confirmed; however, the finding of high-level antibodies to this agent in four out of five cases where serum samples were available is suggestive evidence. Further research is needed to evaluate these findings, since it has recently been shown that findings in cases of *Chlamydia*-associated endocarditis[17] might be compatible with infection with *Bartonella*[18] and, according to the present as well as a previous study[11], Swedish male orienteers do not seem to have an increased rate of exposure to *C. pneumoniae*.

The histopathological alterations in the hearts of the present cases were advanced, with one exception, and included fibrosis, suggesting a subacute to chronic condition. In those cases with cardiac symptoms, i.e

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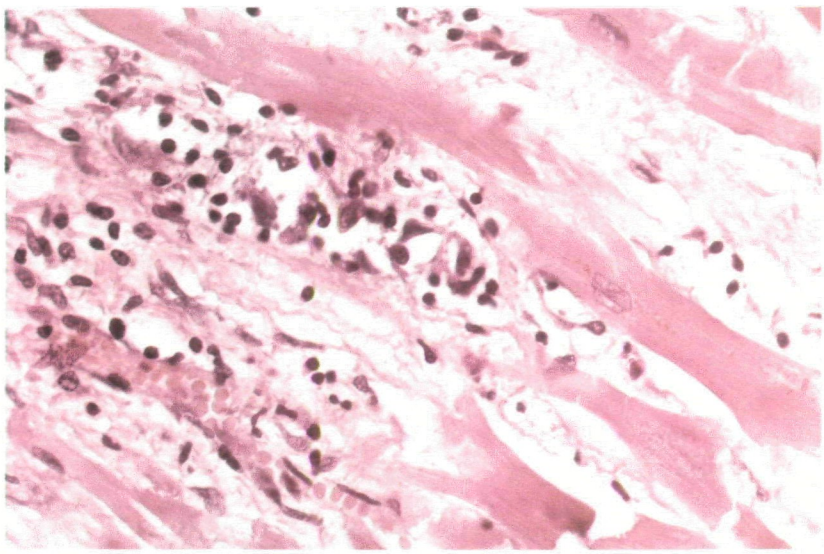

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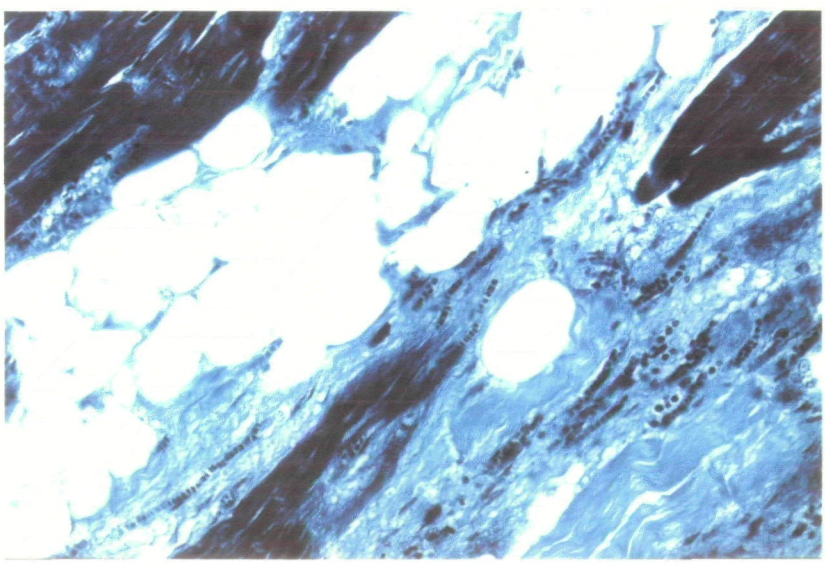

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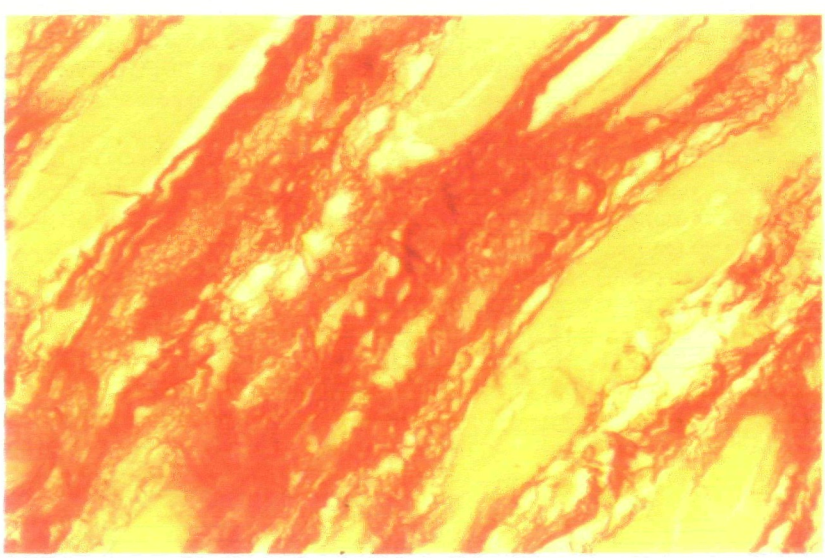

fainting during exercise or tachycardia, an underlying condition causing arrhythmia seems to have been present for at least 4 months to 2 years prior to death, as estimated from the time of onset of these symptoms. In one case, the myocardium was normal at autopsy, except for alterations found in the atrio-ventricular conduction tissue suggestive of causing pre-exitation. This had also been present in previous ECG recordings and palpitations began several years before death. Histopathological myocarditis was found in a considerably higher proportion of cases than the approximate 10% reported in previously published studies of sudden unexpected cardiac death in young athletes[2]. Some 62–75% showed myocarditis at different stages, and in 31% of all the cases, active myocarditis was the sole finding. Among the remaining 31–44% of the cases, myocarditis foci were present along with other alterations. The histopathological picture of myocarditis did not give any clue to the aetiology.

There are no indications that Swedish orienteers have been exposed to doping or other toxic agents. Thus, other causative factors must be considered.

Arrhythmogenic right ventricular dysplasia is a recognised cause of sudden unexpected cardiac death[14]. In two of the present cases, severe changes of this kind were seen in the left ventricle but could not be confirmed in the right ventricle. It has previously been reported in arrhythmogenic right ventricular dysplasia that histopathological arrhythmogenic right ventricular dysplasia-like changes may involve the left ventricle as well (G. Thiene, personal communication). In a third case of the present study, arrhythmogenic right ventricular dysplasia changes were found in the right ventricle, and in a fourth case, showing histopathological early-healing myocarditis, a macroscopic picture compatible with arrhythmogenic right ventricular dysplasia had been described in the autopsy protocol. Among these four cases histopathological myocarditis, at various stages, was a concomitant finding in at least three cases. The aetiology of arrhythmogenic right ventricular dysplasia remains unexplained. Various aetiological factors have been discussed, including a genetic predisposition, possibly in combination with exogenous factors including infection[14].

The histopathological re-evaluation was, for obvious reasons, restricted by the available tissue materials, which, in some cases of the retrospective group (subgroup I, cases 1–14), were sparse.

One conspicuous feature regarding all the present sudden unexpected cardiac death cases is the fact that they all performed in their sport at, or close to, their individual maximum even shortly before they died. This is noteworthy in the face of the pronounced alterations in the myocardia found at the autopsies (except in one case of pre-exitation).

The investigation into the sanitation equipment used by the different orienteering clubs, as well as a review of the hygienic routines used, did not provide any information that could explain the transmission of infectious agents.

After late 1992 there have been no new sudden unexpected cardiac death cases among young orienteers. It is too early to state whether or not the sudden unexpected cardiac death accumulation among young Swedish orienteers is now over. If so, it seems plausible that we are seeing a favourable effect of the 6-month intermission in training and competition in 1993, when all elite competitions were cancelled. We estimate that compliance with the recommendation not to train was over 80% among the elite orienteers. Furthermore, recommendations not to train while infected was strongly and systematically enforced. The risk of lethal arrhythmia decreases with rest in various heart diseases, and it is experimentally established that myocarditic lesions may become more severe with exercise[19,20]. It has also been established that several immune variables transiently deteriorate following intensive and prolonged exercise[21,22]. In fact, up to the end of 1992 the top segment of the male orienteers effectively trained for 350–700 h, mainly forest and track running, and participated in 25–50 competitions yearly. The figures for the females are somewhat lower.

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Figure 3 **Advanced histopathological pictures of the myocardium in sudden unexpected cardiac death victims as categorized according to predominant findings (see text).** ***(a) Active myocarditis only.*** **Myocytolysis and inflammatory reaction in left ventricle (haematoxylin–eosin stain).** ***(b) Arrhythmogenic right ventricular dysplasia (ARVD)-like pathology.*** **Fatty infiltration, fibrosis, and hypertrophy of remaining myofibres in left ventricle (May–Grünwald–Giemsa stain). The picture resembles that found, primarily in the right ventricle, in the syndrome of arrhythmogenic right ventricular dysplasia.** ***(c) Non-specific degenerative changes.*** **Extensive fibrosis in left ventricle (Sirius stain).**

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