Effects of Viral Upper Respiratory Illness on Running Gait

Thomas G. Weidner, PhD, ATC; Gale Gehlsen, PhD; Terry Schurr, PhD; Gregory B. Dwyer, PhD

Objective: To determine the kinematic changes that may occur during running with a cold of known etiology and to assess the impact of select accompanying upper respiratory illness symptoms.

Design and Setting: In this nonrandomized study, subjects with colds and subjects without colds were videotaped while exercising on a treadmill. Three weeks later, the trials were repeated.

Subjects: Eighteen young adults (5 females, 13 males; mean age = 20.4 ± 2.4 yr) with naturally acquired moderate to severe (total symptom score) colds were screened and selected for inclusion in the illness group (ILL). A control group (CRL) of 20 subjects (2 females, 18 males) was also examined. Virologic confirmation of specific viral infections, unprecedented in this line of research, revealed that 12 of the 18 subjects in the ILL group (67%) were infected with human rhinoviruses. None of the subjects had a fever.

Measurements: All subjects exercised on a treadmill for 5 minutes at a heart rate of approximately 85% of their age-predicted maximum. Both groups were videotaped kinematically during two running trials 3 weeks apart. All subjects in the ILL group displayed upper respiratory illness symptoms for the first running trial and were asymptomatic by the second.

Results: We identified significant differences in mean changes between the ILL and CRL group stride lengths ($p < .01$), stride frequencies ($p < .05$), and ankle maximum angle displacement ($p < .01$). Mean changes in stride length ($p < .03$) and in stride frequency ($p < .04$) were larger for ILL subjects who felt feverish.

Conclusions: Alterations in running gait during a rhinovirus-caused upper respiratory illness, and possibly increases in injury incidence, may be associated with feeling feverish. Gait alterations may increase injury incidence or decrease athletic performance, or both.

Key Words: kinematics, stride length, stride frequency, human rhinovirus

Viruses are the most common infectious agents affecting humans. Some authors contend that viral upper respiratory illness (URI) causes more frequent acute disability among athletes than all other diseases combined.[1] Disease patterns among Summer and Winter Olympic athletes are remarkably consistent, with respiratory infections heading the list, followed by gastrointestinal disorders and skin infections.[2] In the 1992 Winter Olympics, some of the world's greatest athletes were unable to compete or did not perform strongly because of a URI,[3] and several athletes were reportedly unable to compete in the 1988 Summer Olympic games due to infectious illness.[4] The average adult has from one to six colds each year,[5] with human rhinoviruses (HRV) accounting for 40% to 50% of these infections. Rhinovirus infections are most prevalent in the fall and spring months.[6] Athletes and exercise enthusiasts, however, commonly continue to participate in competitive and recreational sports during URIs. Unfortunately, little information related to kinematic changes that may occur during exercise with a rhinovirus-caused URI is available.

Detectable abnormalities in pulmonary functional capacity, such as forced expiratory volume and forced vital capacity, are known to occur during infectious illness, including URI.[7,8] The authors of these studies concluded that weakness in the inspiratory muscles may contribute to breathlessness during exertion. They added that this weakness may explain why athletic performance tends to be reduced during viral illness, suggesting that perhaps strenuous exercise should be avoided during such infections. Also, URI caused by rhinoviruses can produce transient peripheral airway abnormalities.[9]

Alterations in muscle ultrastructure and enzyme activity have been identified during viral and mycoplasma infections.[10] Roberts[11] suggested that a decrease in muscle glycogen utilization occurs during viral illness, while Ardawi and Newsholme[12] reported that a decrease in muscle glutamine release occurs with URI during prolonged physical training. Since viral infections can affect the heart, the lungs, and the muscles, it is important to quantify the effect that a URI may have on kinematic performance during exercise or labor. It behooves physically active individuals and those who provide health care and safety recommendations for them to realize that changes in kinematic performance may alter the mechanical and functional aspects of performance during exercise or labor and possibly increase the risk for musculoskeletal injury.

The purposes of this study were twofold. The main purpose was to examine the effects of a URI of known etiology on selected kinematic variables during treadmill running. A secondary purpose was to investigate if the effects of a URI on the kinematic variables were different for individuals reporting symptoms often associated with URI than for individuals not reporting the symptoms. The symptoms looked at in this study were feverishness, laryngitis, and aching muscles or joints.

Thomas G. Weidner, **Gale Gehlsen**, and **Gregory B. Dwyer** are affiliated with the School of Physical Education, and **Terry Schurr** is affiliated with University Computing Services, at Ball State University, Muncie, IN 47306.

METHODS

Physically active subjects with naturally acquired episodes of URI were recruited through the university student health center and through written announcements. This study could not be restricted to a homogenous group (eg, elite runners), because such a study, though interesting, would have required extending the period of the study indefinitely while waiting for a select group of individuals to become ill with a cold. Eighteen experimental subjects (5 females, 13 males) were screened and selected for inclusion in the illness (ILL) group for this study. Subjects met the following criteria: All were currently suffering (within 1 to 2 days of onset) from a moderate or severe upper respiratory illness. None reported symptoms of nausea, vomiting, or diarrhea. All were 18 to 28 years of age, moderate exercisers (five 30-minute aerobic exercise sessions per week), and nonsmokers with no history of alcohol or drug abuse. Subjects were not currently using medications (except oral contraceptives) or suffering from hypertension, heart murmur, hepatitis, kidney disease, hay fever or allergies, asthma, lung disease, chronic respiratory illnesses, or diabetes. Subjects' URI episodes were confirmed through laboratory diagnosis.[1,4,11,13]

All subjects agreed to refrain from self-treating their colds (eg, no over-the-counter medications) during the initial 3 days of evaluation of the URI. A control group (CRL) was also assembled, consisting of 20 subjects (2 females, 18 males) who met the selection criteria and guidelines except for the presence of URI. All subjects signed an informed consent form approved by the Ball State University Institutional Review Board, and those subjects with a URI who completed the study received modest remuneration for their efforts. Table 1 summarizes the physical characteristics of the study participants in each group.

Clinical and Etiologic Evaluation of URI

A symptom score questionnaire, used by Dick et al[14] for more than 20 years (Figure), assisted the physician in selecting subjects with moderate or severe colds. Nasal wash and blood specimens were taken from the subjects at the Ball State University Laboratories and were transported on ice by a delivery service to the Virus Section of the Wisconsin State Laboratory of Hygiene, usually arriving the day after collection. Viral cultures were performed as described in previous literature.[14,15] Human rhinoviruses were identified by cytopathic effect, cell spectrum, and acid lability, and they were serotyped by neutralization tests that used intersecting pools of monospecific antibody to 87 of the more than 100 known HRV types.[16] Pool HRV identification was confirmed by virus neutralization to high titer with serial dilutions of the indicated serotype. HRV serologic diagnosis (neutralization test) was performed on subjects' acute and convalescent sera obtained at least 3 weeks apart in a mycoplasma-free strain of Ohio State HeLa cells; virus challenge was 20 to 50 $TCID_{50}$ (tissue culture 50% infective dose).

COLD SYMPTOM REPORT SHEET

Name ____ Date ____

Subject Code Number ____

Score each symptom for each day as follows: 0 not present, 1 mild, 2 moderate, 3 severe

DAY	1	2	3
Cough	1	1	2
Nasal discharge	3	3	2
Sneezing	2	3	2
Stuffy nose	3	3	2
Sore throat	1	1	2
Headache	2	2	1
Malaise (tired out)	2	2	1
Chilliness	1	1	1
Shaking chills	0	0	0
Fever/feverishness	1	1	2
Laryngitis (hoarseness)	1	1	1
Aching joints or muscles	1	1	2
Watery or burning eyes	0	0	0
TOTAL SYMPTOM SCORE	18	19	18

A typical rhinovirus cold in this study. See Table 2 footnotes for definitions of mild, moderate, and severe colds.

Heart Rate

Heart rates were measured using a CM-5 electrocardiogram (ECG) lead setup with a Hewlett-Packard ECG Unit (model #1500B, Hewlett-Packard, Waltham, MA). All exercise sessions were conducted on a Quinton motorized treadmill (Quinton Instruments, Seattle, WA).

Kinematic Video Recording

Subjects ran on a treadmill, and each one's exercise gait was monitored using a stationary digital shuttered video camera. The camera was located perpendicular to the sagittal plane of movement at a distance of 10 meters. A wide-angle lens

Table 1. Physical Characteristics of the ILL and CRL Group Subjects (Means and Standard Deviations)

Group	Height (m)	Weight (kg)	Age (yr)	Heart Rate (bpm) Trial 1	Heart Rate (bpm) Trial 2
ILL	1.74	69.7	20.4	166.0	168.4
	(0.09)*	(11.6)	(2.4)	(13.2)	(16.3)
CRL	1.82	71.2	18.4	167.0	168.0
	(0.10)	(12.8)	(4.2)	(8.2)	(8.7)

* Standard deviations are shown in parentheses.

allowed the entire movement to be viewed. A 386 computer with a BCE-associated video control board was interfaced with a video playback system monitor and a video cassette recorder. A computer program (Peak Performance, Peak Performance Technologies, Inc, Englewood, CO) was used to encode sequentially every field on the tape. The time between any two given frames was determined with an accuracy of 1/60 second. To facilitate the location of segmental endpoints during the film analysis, contrasting markers were placed on the joint centers of each subject. Black and white plastic tape was placed on the following anatomical landmarks: 5th metatarsal, lateral malleolus, lateral condyle of the knee, and greater trochanter. An accelerometer was used to determine the number of running foot impacts per second (stride frequency). Stride length (SL) was calculated from impact frequency (IF) and treadmill velocity (V) parameters (SL = V/IF).

Experimental Design

Each ILL subject reported to the biomechanics laboratory for testing. The physician evaluated each subject's URI symptom severity inventory[17] and health history. The following measurements were taken for each subject: height, weight, resting heart rate, blood pressure, and oral temperature. In addition, a nasal wash was performed to isolate rhinoviruses, and blood was drawn from each subject to determine antibody titers against rhinoviruses. CM-5 ECG electrodes for the heart rate determination were then placed on each subject. The subjects were familiarized with the treadmill and began walking at 2 mph. Treadmill work loads were adjusted by 0.5 mph every minute to achieve a heart rate response of approximately 85% of age-predicted maximal heart rate (PMHR). The ECG was recorded every 30 seconds of exercise; heart rates were measured from these ECGs. The treadmill speed at 85% of PMHR was recorded and the subject ran at that speed for 5 minutes. During the 5-minute run at 85% of PMHR, the subjects were videotaped.

The ILL subjects reported to the laboratory at 7:30 AM the two mornings after the initial treadmill run for further cold evaluations (ie, nasal wash and URI symptom inventory updates). The exercise bouts and videotaping were repeated 3 weeks later when the subjects were asymptomatic and presumed to be healthy. The control group completed identical exercise bouts and accompanying videotaping for two trials 3 weeks apart.

Data Analysis

A multivariate analysis of variance (MANOVA) with repeated measures on the trial condition was used to determine the effect of URI on the kinematic variables. Two factors were specified in the MANOVA: an experimental/control between-subjects grouping factor and a within-subjects (ie, repeated-measures) trial factor. Trial conditions for ILL subjects were having and not having URI symptoms. Trial conditions for CRL subjects were two replications in the absence of URI symptoms. Results for the group-by-trial interaction were the foci of this study, that is, differences in mean values of the two groups under the two trial conditions.

Two separate MANOVA procedures were performed: one for stride length and stride frequency under the two trial conditions (values for the two variables under each condition) and one for the maximum and minimum ankle, knee, and hip joint angles under the two conditions (for a total of six variables under each condition). Bonferroni procedures were used to adjust the required R values for the group-by-treatment analyses of the separate variables in order to maintain an overall alpha level of 0.05. The p values were evenly divided between the two MANOVAs ($p = .025$), resulting in required Bonferroni univariate p values of .0125 for the stride data and .0042 for the joint angle data. Complete data for 18 experimental subjects were available for the stride frequency and length analyses. Complete joint angle data were available for 13 ILL subjects. The CRL group provided complete data for all 20 subjects in both analyses.

Separate ANOVAs with repeated measures on the trial condition were used to determine the effects that the URI feverish feeling, laryngitis, and aching muscles or joints symptoms had on the stride length and stride frequency kinematic variables. Only the experimental group was used in these analyses. Two factors were specified in the ANOVA: the presence or absence of a symptom during the illness trial (between-subjects grouping factor) and a within-subjects (repeated-measures) trial factor. Results for the group-by-trial interaction were the foci of the analyses, that is, differences in mean values of the kinematic variables for the two groups under the two trial conditions.

RESULTS

Episodes of URI

Rhinoviruses were cultivated from 6 of the 18 ILL subjects (Table 2). Because there are more than 100 individual HRV strains, it was not possible economically to thoroughly type the isolates. Using 87 strain-specific sera, three isolates were identified as HRV15, HRV23, and HRV43, and three were untypable. Serodiagnosis was performed with the isolates against a substantial segment of the paired (acute and convalescent) sera, and this yielded an additional six diagnoses for a total of 12 of the 18, a very high diagnosis rate (67%) despite the necessary diagnostic economies.[18,19]

Stride Length and Stride Frequency

The MANOVA yielded an F(2,38) of 4.55 for the interactive effect of group by trial on the stride length and frequency variables that had a $p = .017$. The p value was smaller than the required Bonferroni p value for both stride frequency and stride length. The mean differences for the groups under the two trials and the univariate ANOVA statistics are shown in Table 3.

The mean difference in stride length between the illness and control trials for the ILL group was −0.08 m. In contrast, the mean difference in stride length for the two trials of the CRL

Table 2. Severity of Symptoms and Etiology of Colds in 18 Regularly Exercising University Students

Subject	Severity of Cold*	Virus Isolated†	Serodiagnosis (Isolate)‡
A	+++	HRV-NT	A (HRV-NT)
B	++	. . .§	. . .‖
C	+++	HRV-NT	C (HRV-NT)
D	+++	. . .§	U (HRV43)
E	+++	. . .§	C (HRV-NT)
F	+++	HRV23	F, P (HRV-NT)
I	++	. . .§	C (HRV-NT)
J	++	. . .§	. . .‖
K	+++	. . .§	. . .‖
L	+++	HRV23	L (HRV23)
M	+++	. . .§	. . .‖
N	++	. . .§	. . .‖
P	++	HRV23	P (HRV23)
Q	+++	. . .§	C (HRV-NT)
R	+++	. . .§	C (HRV-NT)
S	+++	. . .§	V (HRV-NT)
U	+++	HRV23	U (HRV43)
X	++	. . .§	. . .‖

* Thirteen cold symptoms were scored as described in the text, and the symptom scores were totaled. Patients scoring 0–6 were deemed to have a mild cold (designated as +), 7–11 a moderate cold (++), and 12 or greater a severe cold (+++).

† Viruses were isolated from nasal washes as described in the Methods section and were identified by virus neutralization assays using 87 individual HRV-specific antisera. Those designated HRV-NT (nontypable) could not be identified using the available antisera. HRV = human rhinovirus.

‡ Patient convalescent sera were tested for a fourfold or greater rise in titer of antibody against rhinovirus. Controls were acute sera from the same patient. The viruses used to assay patient sera were those isolated from patients who participated in the study. For example, convalescent serum from patient D reacted with the virus isolated from patient U, which was independently identified as HRV23.

§ No virus isolated.

‖ No serodiagnosis obtained.

group (illness absent for both test periods) was 0.01 m. These differences for the two trials indicated that subjects had longer strides when they had URIs than when they did not have URIs ($F(1,39) = 7.32$, $p < .01$).

The mean difference in stride frequency between the illness and control trials for the ILL group was 0.18 Hz. In contrast, the mean difference in stride frequency for the two trials of the CRL group was −0.03 Hz. These differences for the two trials indicated that stride frequency was less when the subjects had URIs than when they did not have URIs ($F(1,39) = 8.93$, $p < .005$). Thus, the presence of URI was associated with longer and less frequent strides.

Joint Displacement

The MANOVA yielded an $F(6,26)$ of 2.70 for the interactive effect of group by trial on the six joint angle variables that had a $p = .036$. The p value was smaller than the required Bonferroni p value for only the maximum angle of the ankle joint ($F(1,31) = 13.38$, $p < .001$). The mean differences of the groups under the two trials and the univariate ANOVA statistics are shown in Table 3. The difference in the maximum ankle angle for the experimental group under the illness and control conditions was −5.6 degrees, while the difference for the two trials of the control group was 0.30 degrees. These differences indicated greater extension when URI was present.

Illness Group Symptoms and Stride Length and Frequency

Significant differences in the stride length and stride frequency means for the ILL group under the illness and control conditions were found for only the feverishness symptom. The mean value for the stride length of subjects reporting feverishness was 0.16 m higher under the illness condition than under the control condition, while the mean difference under the two conditions was only 0.04 m for subjects not reporting feverishness ($F(1,19) = 5.12$, $p < .03$). Concomitantly, the mean value for the stride frequency of subjects reporting feverishness was 0.34 Hz less under the illness condition than under the control condition, while the mean difference under the two conditions was only 0.08 Hz for subjects not reporting feverishness ($F(1,19) = 5.12$, $p < .03$).

DISCUSSION

This is the first study to measure kinematic changes during exercise with a URI. Also, to the best of our knowledge, this is the only study in sports medicine to confirm the etiology of URI episodes or to study the effects of a nonviremic viral URI on exercise performance. Furthermore, this study focused primarily on the effects of only a single virus (HRV) infection on exercise performance. We were able to determine the specific viruses that caused colds in 12 of our 18 subjects (67%). All these viruses were associated with HRV, a frequent cause of colds, especially in the early fall months.[13,20,21] The results of this study indicated that individuals suffering from a URI use longer and slower strides than do individuals not suffering from a URI. Additionally, when subjects in the ILL group recovered from their URIs, their gait was identical to that of well controls. Some caution should be used, however, in interpreting these results. It would be of interest to determine if a group of experienced runners who have had extensive practice on the treadmill before acquiring a URI would yield similar results.

Weidner[22] studied the reporting behaviors, activity levels, and perceived physical performance levels of 290 intercollegiate athletes (165 males, 125 females) with URIs. Respondents rated the severity of 14 cold symptoms and indicated to whom they reported their cold and within how many days. Respondents were asked to indicate whether they self-treated their illness, whether they missed a practice or game due to the cold, and whether the cold affected their performance. Of the illness episodes reported, 17.8%[20] caused the athlete to miss a practice and 5.1%[6] caused the athlete to miss a game. Athletes with URIs felt in some instances that their illness affected their performance. Again, the cold symptom of feverishness, along with laryngitis, aching muscles or joints, and nasal discharge, was significantly ($p < .05$) correlated with reporting behaviors, activity levels, and perceived physical performance.

Table 3. Stride Length, Stride Frequency, and Joint Angle Mean Differences Between Trial 1 and Trial 2

	Mean (Standard Deviation)		ANOVA*			
	ILL	CRL	Hypothesis MS	Error MS	F	*p*
Stride length (m)	−0.08 (0.3)	0.01 (0.02)	0.043	0.006	7.32	.010
Stride frequency (Hz)	0.18 (0.06)	−0.03 (0.04)	0.231	0.026	8.93	.005
Ankle joint maximum angle (deg)	−5.6 (6.8)	0.3 (5.2)	176.79	13.21	13.38	.001
Ankle joint minimum angle (deg)	−2.1 (10.0)	−0.9 (5.2)	2.70	24.53	0.11	.742
Knee joint maximum angle (deg)	1.1 (6.3)	−2.8 (6.9)	93.07	19.27	4.83	.036
Knee joint minimum angle (deg)	1.5 (9.7)	0.6 (4.8)	23.84	29.15	0.82	.373
Hip joint maximum angle (deg)	−1.4 (5.6)	0.6 (4.2)	16.81	13.87	1.21	.280
Hip joint minimum angle (deg)	−4.2 (8.9)	−0.3 (4.8)	16.14	12.31	1.31	.261

* ANOVA statistics are for the group-by-trial interaction. Df for the stride length and frequency variables were 1,39. Df for the ankle, knee, and hip joint variables were 1,31.

Results of Weidner's[22] study appear to be supported by our study. The ILL subjects altered their freely chosen stride length and stride frequency (ie, increased stride length and decreased stride frequency) between illness and convalescent running trials. Stride length and stride frequency are generally described in terms of velocity and physical stature, both of which were controlled in this study. It is reasonable to believe that a general feeling of feverishness, as perceived by the subjects, may have affected kinematic performance. When the subjects were ill, they chose to move the lower limbs more slowly, while increasing the range of the ankle and hip. However, a general feeling of fatigue also may have influenced performance. Other studies have similarly indicated that the stride rate of sprinters decreases as a result of fatigue,[23,24] sometimes with an increase in stride length. Further research is necessary to substantiate our results.

CONCLUSIONS

In this study, kinematics of running were altered during an HRV-caused URI. In particular, stride length and stride frequency changed significantly between illness and convalescent running trials. Perhaps due to these changes and others that may occur but were not measured (eg, endurance, power), athletes may be susceptible to injuries or declines in athletic performance, or both, during a URI. Although the perception of feverishness may be an important indicator for alterations in kinematic performance, further examination of kinematic performance during an HRV-caused URI, as well as from other etiologies, such as enteroviruses, appears to be a fertile field for research.

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