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The Predictive Value of Cardiovascular Disease for Shoulder Injuries in a Retrospective Cohort Study

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Clinical Significance

This article examines and quantifies the strong relationship between an accepted cardiovascular risk factor metric and rotator cuff injuries in a retrospective cohort study of commercial truck drivers. Identification of cardiovascular risk factors as potential rotator cuff risk factors may highlight potential etiological pathways and prevention tools.

Abstract

Objective: To assess predictive relationship that cardiovascular disease (CVD) has on work-related shoulder injuries.

Methods: This retrospective cohort study quantifies relationships between CVD risk and subsequent incident cases of shoulder-related worker compensation (WC) claims in a large cohort of 34,369 commercial drivers. CVD risk was assessed using an expanded Framingham Risk score. Incident shoulder WC cases were identified. Relative risk (RR), hazard ratio (HR) and their associated 95% Confidence Intervals (95% CI) were calculated.

Results: Significant relationships between CVD risk scores and subsequent shoulder WC claims were observed. Participants in the highest CVD risk were significantly more likely to have a shoulder-related WC claim (RR=6.70, 95% CI 2.03, 22.12), after adjustment for age, gender and Body Mass Index.

Conclusion: There may be a relationship between CVD risk and shoulder injuries and disorders.

Keywords: Shoulder disorder; Rotator Cuff; Cardiovascular disease; Cohort study; Occupational Injury Prevention; Occupational Epidemiology; Shoulder MSD; Truck Driver; Commercial Motor Vehicle Driver; Risk Factor;

Learning Outcomes:

- Readers will be able to quantify the predictive association between cardiovascular disease (CVD) risk and incident shoulder-related workers' compensation (WC) claims within a 7-year retrospective cohort of 34,369 commercial drivers.
- Readers will be able to evaluate the utility of the Expanded Framingham Score (EFHS) in forecasting shoulder injuries and disorders among commercial motor vehicle drivers, using relative risk and hazard ratio estimates.
- Within the context of occupational medicine, readers will be equipped to interpret how modifiable CVD risk factors may contribute to musculoskeletal injury prevention strategies aimed at reducing shoulder-related WC claims over a 10-year risk horizon.

Introduction

A 2014 Bureau of Labor Statics (BLS) study of non-fatal work injuries in the general working population reported that upper extremities affected by an injury or illness accounted for 346,170 total cases [1]. Of those cases, 88,980 (26%) of all upper extremity illnesses or injuries were shoulder related. The median days away from work for shoulder injuries was 26 days, 11 days more than the second highest cause of days away from work [1]. Shoulder injury-related worker compensation (WC) claims, ranging from minor treatment to invasive surgical intervention and permanent work restrictions, can cost WC insurers $10,000-$100,000 to settle [2]. Research on shoulder risk factors is sparse, and risk factors for shoulder injuries are not well established, though a few cross-sectional and case-control studies have pointed to cardiovascular disease (CVD) as a possibility [3-6].

Atherosclerotic cardiovascular disease (CVD) is characterized by the premature and progressive hardening of the body's blood vessels [7]. In the occupational setting, evidence suggests that approximately 53% of workers with CVD are less than 60 years old [8]. Known risk factors for CVD include hypertension (HTN), diabetes (DM), hyperlipidemia, obesity, poor diet, physical inactivity, age, and sex have been identified as risk factors for CVD [7, 9, 10]. The Framingham Heart Study, a 50-year prospective cohort study of CVD, has shown that a combination of cardiovascular (CV) risk factors can predict an individual's likelihood of a CV health event in the following ten years [7].

The unique features of the shoulder subsequently place a high demand for the shoulder's microscopic vasculature to accommodate the perfusion of muscles, tendons, ligaments, cartilage, and nervous tissues [11]. The vasculature of the shoulder is known to be

small, delicate, and commonly insufficient in otherwise healthy individuals [11]. It would go to reason that disruptions in the health of these blood vessels, such as those caused by CVD, would result in accelerated shoulder injuries. Given the known effects of CVD on the blood vessels of the body [7, 10, 12] and the vasculature of the shoulder, we hypothesize that components of CVD are risk factors that contribute to shoulder injuries and disorders.

A population known for its increased prevalence of CV risk factors is truck drivers [13-16]. Gender, poor cholesterol, smoking, DM, and hypertension are all common CV risk factors in the CMV drivers (CV) health [7, 17-19]. Each of these risk factors negatively impacts the microvasculature of the CV system [10, 12]—the primary vasculature in the shoulder joint. Truck drivers also have an increased exposure to biomechanical risks for MSDs while performing non-driving-related work activities [20, 21]. Drivers regularly experience minor to serve musculoskeletal injuries during cargo handling, ingress and egress of the cab, and connecting and removing the trailer from the fifth wheel. Truck drivers are 3.5 times as likely to experience musculoskeletal injuries than the general population [20].

A growing body of evidence supports a significant relationship between cardiovascular disease (CVD) risk factors and shoulder disorders, particularly rotator cuff pathology and adhesive capsulitis. Multiple occupational and clinical studies have shown that individuals with higher Framingham CVD risk scores have markedly increased odds of glenohumeral joint pain and rotator cuff tendinopathy, with dose-response patterns suggesting potential causal mechanisms [22]. Longitudinal data further indicate that elevated CVD risk predicts future development of multiple musculoskeletal disorders, including shoulder conditions, over nearly a decade of follow-up [23] Other publications of cross-sectional and case control studies

identified associations between cardiometabolic risk factors and rotator cuff tendinopathy or glenohumeral pain [22, 24, 25]. Specific cardiometabolic components such as obesity, hypertension, hyperlipidemia, and diabetes mellitus have also been independently associated with larger or more severe rotator cuff tears and higher incidence of adhesive capsulitis [26-29] Systematic reviews and genetic analyses reinforce these findings, showing consistent links between metabolic syndrome traits and rotator cuff-related shoulder pain [30, 31]. Imaging studies have demonstrated correlations between systemic atherosclerosis and rotator cuff degeneration, suggesting vascular insufficiency as a plausible biological pathway [32]. These potential relationships are consistent with biological models that propose a role for atherosclerotic microvascular insufficiency in tendon degeneration and impaired tissue repair [33, 34]. Collectively, these prior publications suggest that cardiometabolic dysregulation is related to, if not contributes to, impaired tendon perfusion, collagen integrity, and healing capacity, offering a mechanistic bridge between cardiovascular and shoulder musculoskeletal disease.

As an occupation, truck driving is marked by low physical activity, poor diet, increased use of substances, and poor sleep [17, 35, 36]. According to the Job Demands-Resource model [37], these job demands increase the truck drivers' risk of physical injury due to limited resources provided by trucking employers to mitigate the negative physical effects of the job demands on drivers. This retrospective cohort study aims to quantify relationships between CVD risk and incident cases of shoulder-related worker WC claims. We hypothesize that the increased job demands and limited resources to address the physiological effects—CVD—of those demands place our sample at increased risk of a shoulder injury.

Methods

Study Design

These analyses continue one of the most extensive cohort studies of CMV drivers completed in the US. Previous publications from this dataset outline the methodological details of data collection; as such, only details concerning the completion of this analysis will be presented [13]. Institutional Review Board approval was obtained from the University of Utah (IRB# 35889 and 35741).This retrospective cohort study examines data collected over a span of seven years. A retrospective cohort study is the appropriate study type because the data permits the establishment of a temporal relationship.

Study Participants

The sample of CMV drivers includes *n* = 49,633. Driver data was collected from 2005 – 2012 from drivers employed by a large trucking company. The data is from the unification of three large data sources; commercial driver medical exams (CDME) data from a clinical system, employment data, including hire and termination dates, and internal workers' compensation data from a large trucking company. These exams are required for drivers to operate a commercial vehicle across state lines, and there are many medical conditions which prevent drivers from being medically qualified [38]. Data were merged by name, driver's license number, and birth date to ensure correct linkage between the three data sources. The data was then stripped of all personally identifiable information. We excluded participants who were employed by the company for less than 7 days. We followed the STROBE checklist for cohort

studies throughout this study and manuscript (see STROBE Checklist in Supplemental Digital Content, http://links.lww.com/JOM/C338).

Measuring Cardiovascular Disease

The Framingham Heart Study is a large, multigenerational prospective cohort study assessing the predictive nature of atherosclerotic cardiovascular disease (CVD) in men and women [7]. The study focused on creating a tool that allows physicians to identify CVD risk based on a patient's gender, age, HDL, total cholesterol, treated and untreated systolic blood pressure (SBP), smoker status, and diabetes (DM) status. Each of these factors is assigned point values based on presence, absence, or severity and are summed to create an output value indicating a patient's risk of cardiovascular (CV) health event in the next ten years.

Not all of the measures used in the Framingham CVD prediction calculator are collected as part of a CDME. Therefore, we have created a scoring system based on the Framingham risk scale that we used to measure the CV risk factors among our sample of CMV drivers, called the Expanded Framingham Score (EFHS). This EFHS utilized the Framingham measures which were collected, and incorporated the additional CV health information captured during CDME for each driver. These data are gender, age, history of heart disease, history of heart surgery, treated and untreated SBP, DM status, HTN meds, smoking status, and treated and untreated cholesterol. The additional categories we identified that were not included in the original FHS, are cardiovascular history, history of heart surgery, number of prescribed HTN medications, and medications to treat poor cholesterol. Many of these factors were identified by searching through physician comments in the CDME records. These variables were selected as markers

which provide additional insight into an individual's CV health. Like the Framingham score, these variables were used to create a score for each driver. The sum of the points outputs a value we used to measure the CVD risk for each driver in our sample.

Because commercial driver medical exam records did not consistently contain lipid measurements or smoking status, we used an adapted cardiovascular risk index that followed the structure of the Framingham general cardiovascular disease profile while substituting variables available in the commercial driver medical exam and adding pre specified cardiovascular history and medication indicators. This strategy is consistent with prior work that provides a body mass index based Framingham formulation for use when lipid data are not available and with validated office based nonlaboratory risk tools that substitute body mass index for cholesterol while maintaining acceptable predictive performance [7, 39]. We report both discrimination and calibration for our adapted score and compare results with models that use only the original Framingham variables available in our data.

Moreover, we created two similar scoring systems. The first is a Modified Framingham Score, using all of the original Framingham measures that were available in the CDME; age, gender, treated and untreated SBP, diabetes, and smoker status. The second is a CV risk score using only the history-based components of the EFHS, therefore excluding SBP measurements. A Pearson Correlation Coefficient was calculated with all three scoring systems. See Appendix 1 for all three scoring tables (http://links.lww.com/JOM/C339).

Search Methods

We searched the dataset using SAS to identify components related to CV health. We searched common synonyms for the selected risk factors pertinent to CV risk. The progression of CVD leading to cardiac and cerebrovascular events is a well-established disease progression [40]. As such, an individual with multiple risk factors only receives the highest point of the two risk factors. E.g., a male driver with a history of CAD and MI gets only 12 points for the history of MI.

The CV History column in the EFHS the scored was based on a history of coronary artery disease (CAD), stroke, and myocardial infarction. The Heart Surgery column gave points for a history of angioplasty, cardiac stent, and coronary artery bypass graft (CABG). For hypertension medications (HTN), individuals received a score of zero for taking one medication because at least one medication is implied in the SBP (treated) column from the original Framingham score. Since total cholesterol (TC) and high-density lipoprotein (HDL) lab values, are components of the original Framingham score, we captured attempted to capture them as well. We searched for cholesterol in the exam and physician comments and scored based on the number of reported cholesterol medications for a given individual. All these components were identified a priori.

Because we captured CV history, history of heart surgery, number of HTN meds, and cholesterol medications from exam and physician comments, we searched for common synonyms and misspellings for each disease, surgery, and medication. We then reviewed all relevant comments to ensure the omission of non-CV-related comments. For example, a driver ID with the exam comment of “heat *stroke*” was identified and omitted from the driver ID

codes with "*stroke*." This search procedure was applied to all the terms searched. See Appendix 2 for a complete list of phrases searched (http://links.lww.com/JOM/C339).

To establish temporality between cardiovascular risk and shoulder injury, the Expanded Framingham Score (EFHS) was calculated from cardiovascular health data recorded at each driver's initial commercial driver medical examination (CDME). This examination occurred before the start of observation for workers' compensation outcomes. Each driver's time zero was defined as the date of the first CDME or the hire date, whichever occurred later. Only shoulder injury claims that were filed after this baseline date were considered incident cases. Drivers who had a shoulder claim prior to their first CDME were excluded. This design ensured that cardiovascular risk factors were measured before the occurrence of the outcome and permitted assessment of temporal relationships between baseline cardiovascular health and subsequent injury events.

Shoulder Injury Claim

A shoulder injury claim was defined as any new workers' compensation (WC) claim in which the primary body part affected was the shoulder, including the rotator cuff, glenohumeral joint, acromioclavicular joint, or associated musculotendinous and soft tissue structures. Claims were identified from the company's internal WC database using standardized body-part and nature-of-injury codes, supplemented by text searches of physician and adjuster notes for the terms "shoulder," "rotator cuff," "labrum," or "glenohumeral" Only incident claims that occurred after the baseline commercial driver medical examination or hire date

were included, ensuring that all injuries represented new occupational events. Claims associated with follow-up visits, rehabilitation, or sequelae of prior injuries were excluded.

This operational definition aligns with approaches used in prior occupational and musculoskeletal epidemiology studies that relied on administrative WC data to identify clinically relevant and work-related shoulder outcomes. Comparable definitions have been used in research on shoulder disorders among working populations, rotator cuff disease in compensation claim datasets (Miranda 2008), and upper extremity musculoskeletal injury surveillance among commercial motor vehicle and industrial workers [23, 41-43]. These studies similarly used standardized WC body-part codes, medical diagnosis text fields, or physician review to define cases, ensuring that injury ascertainment reflects validated occupational exposures and outcomes.

Statistical Methods

The unit of measurement is the individual driver. The outcome was the incidence of filing of a new shoulder injury-related WC claim. The primary exposure was cardiovascular disease risk, estimated using the Expanded Framingham Score. Descriptive statistics include mean and standard deviation for continuous or discrete variables. Categorical variables are described using frequency and percentage for each category. The normality of continuous and discrete variables was assessed. Initial comparisons will be evaluated using chi-squared calculations for categorical variables and a t-test or Wilcoxon rank-sum test for continuous variables. Cox proportional hazard models were used to estimate time from baseline to the first

shoulder-related claim. The use of time-to-event modeling further ensured that all outcomes occurred after the baseline EFHS assessment.

To address missingness in smoking and lipids, which reflects clinical workflow rather than outcome status, we considered missing at random as a working assumption and implemented multiple imputation by chained equations including all model covariates, the outcome, and time at risk. We generated 20 imputed datasets and combined estimates with Rubin's rules. This approach is recommended for prediction studies to reduce bias and loss of efficiency compared with complete case analysis [44-47]

Expanded Framingham Score (EFHS) was assessed in two ways; first a continuous measure to assess for monotonal increase and second by categorically dividing the continuous score into deciles of approximately equal size to assess for non-linear risk-outcome relationships. See Appendix 3 for category details (http://links.lww.com/JOM/C339). Crude and adjusted Logistic Regression of incident workers' compensation claims will calculate Relative Risk (RR) and 95% Confidence Intervals (95% CI). Cox Proportional Hazard Modeling was used to calculate Hazard Ratios (HR) and associated 95% CIs. The proportional hazard model assesses time-to-event. In our case, the truck drivers' first CDME or hire date (whichever is later) is their starting time point (time 0). The event date is the date that they filed a shoulder WC claim. Drivers were censored when their employment was terminated or when the study ended.

Potential confounders we considered were tenure with the company, BMI, Low Back Pain, use of narcotic or habit-forming drugs, and alcohol use. Each was assessed individually. Confounders were retained in the adjusted model if the risk estimate (RR or HR) adjustment

differed by 10% or more from the crude risk estimate. All analyses were conducted using SAS 9.4 (SAS Institute, Cary, NC, USA).

Sensitivity analyses

We performed the following sensitivity checks. First, we repeated models using the body mass index-based Framingham formulation as the exposure to mirror the established adaptation for settings without lipids, and we compared discrimination and calibration to the Expanded Framingham Score. Second, we fit models using an office based nonlaboratory risk tool to evaluate robustness to score specification. Third, we compared complete case estimates with multiply imputed estimates to evaluate the impact of missingness handling. Fourth, we conducted a leave one component out analysis to ensure that the observed associations were not driven solely by any single component such as age or blood pressure. Results were qualitatively consistent across specifications [7, 39, 44-46]

Results

The Expanded Framingham Score was calculated to all drivers. The examination of the dataset, including the CDME, the hire and termination dates, and internal WC claims, had a frequency of 49,771 drivers. Of those observations, 15,402 were excluded because their termination date was within one to seven days after their hire date. The remaining 34,369 observations were used to calculate the crude and adjusted Relative Risk (RR) and Hazard Ratio (HR). Crude and adjusted RR and HR were calculated. Previously identified possible confounders were added to the models and were not found to alter the relationships meaningfully. Imputed

models were not significantly or meaningfully different from models excluding observations with missing data and therefore we are presenting results that did not use imputation.

Most study participants were male (95.4%), had a mean age of approximately 46 years, had a systolic blood pressure (SBP) of 126.3 mmHg (SD 12.6), and had a BMI of 3.17 (SD 7.1). See Table 1 for complete details of demographic information. Of the 34,369 drivers, 238 (0.69%) filed a worker compensation claim involving a shoulder injury. The mean CVD score using the Expanded Framingham score (EFHS) of those who filed a shoulder-related claim is 8.52 (SD 5.90) compared to a score of 7.25 (SD 5.36) for those who did not have a similar claim.

Comparing the crude RR and the RR after adjusting for age, gender, and BMI increases the number of categories with statistical significance and increases the RR for each category, as seen in Table 2. The same observation is made for the crude and adjusted Hazard Ratios (Table 3). Of these three confounders, BMI uniquely demonstrated a statistically significant independent relationship to filing a shoulder-related WC claim. In the EHFS model, BMI had an RR of 1.03 (CI 1.02, 1.05, $p = 0.05$).

A Pearson Correlation Coefficient test revealed that the EFHS and Modified Framingham Score (MFHS) have a correlation value of $r = 0.891$. The EFHS and "No test" FHS (EFHS without SBP measurements) had a correlation value of $r = 0.987$. The mean score of the EFHS was 7.26 (SD 5.36), the mean score of the MFHS was 6.512 (SD 4.32), and the mean score of the "No test" FHS was 6.93 (SD 5.39).

Discussion

Results of the adjusted continuous and categorical values for the Relative Risk (RR) and Hazard Ratio (HR) indicate that drivers who have a higher Expanded Framingham Score (EFHS). representing a higher risk of CVD, at the time of their initial CDME are statistically significantly more likely to file a shoulder-related worker compensation (WC) claim at some point after their medical exam, and they are more likely to file a shoulder-related claim sooner than drivers who have lower CV risk (RR=6.70, 95% CI 2.03, 22.12). By defining the cardiovascular assessment as the temporal baseline and including only incident shoulder claims occurring afterward, the study design allows interpretation of cardiovascular risk as a predictive rather than concurrent factor for shoulder injury. Moreover, because the categorical RR and HR of filing a claim increase as a driver's EFHS increases, there is a dose-response between components of CVD and the likelihood of an incident shoulder injury requiring a WC claim to be made. This suggests that drivers with known risk factors for CVD can reduce their likelihood of shoulder injury by lowering their risk factors for CVD. This is in agreement with the few prior studies investigating relationships between CVD and shoulder injuries, although these data demonstrate a stronger relationship and higher magnitude relationship between CVD and shoulder disorders.

The relationship observed between elevated cardiovascular disease (CVD) risk and subsequent shoulder injury in this cohort of commercial motor vehicle drivers is consistent with and expands upon prior literature linking cardiometabolic health to shoulder musculoskeletal disorders. A case-control study completed by Applegate et al. [3] demonstrated an association between CVD risk factors and rotator cuff tendinopathy. This study similarly used a "modified" Framingham CVD risk score to assess the CV risk in their study sample. The study found a strong

correlation between glenohumeral joint pain and rotor cuff tendinopathy and increased CV risk factors as scored by their modified Framingham CVD risk. In an earlier case-control study done by Wendelboe et al. [6], found that men with a BMI greater than 35 kg/m^3 had an Odds Ratio (OR) of 3.13 (95% CI 1.29, 7.61) of having a shoulder repair surgery. Our retrospective cohort study adds to the existing literature by drawing a temporal relationship between CVD risk factors and shoulder injuries and disorders.

A more recent longitudinal study confirmed that elevated CVD risk predicts future development of musculoskeletal disorders, including shoulder conditions, over nearly a decade of follow up [23]. Other investigations have reported that hypertension and hyperlipidemia are associated with greater size and severity of rotator cuff tears [26] and that diabetes and metabolic syndrome increase the risk of adhesive capsulitis and rotator cuff pathology [27, 29, 31] Imaging-based research has also demonstrated correlations between systemic atherosclerosis and rotator cuff degeneration, supporting a vascular mechanism that links CVD to shoulder tissue vulnerability. [32]

Most prior research, however, was cross sectional or case control in design and therefore unable to establish temporality. The present analysis addresses this limitation by demonstrating a clear temporal relationship between baseline CVD risk and later incident shoulder injury claims within a large, nationally distributed working population. Evidence from industrial and construction cohorts has similarly suggested that vascular and metabolic factors contribute to shoulder disorders [41, 42], but few studies have used longitudinal occupational data of this scale. The current findings therefore extend the literature by providing strong evidence that higher baseline CVD risk precedes and predicts shoulder injury in a working

population, indicating that cardiovascular health may be an important upstream determinant of occupational musculoskeletal injury.

In addition to establishing a temporal relationship by demonstrating that CVD risk factors occurred before incident shoulder injuries, we have created three statistically significant scoring methods that predict the Relative Risk and Hazard Ratio of a shoulder injury based on an individual's CV risk factors. The tight correlation of the Expanded, Modified, and "No Test" Framingham Scores denote that all three scores are useful in predicting CV risk factors and subsequent shoulder injuries and disorders. Of particular interest to clinicians is the "No test" FHS because it does not require the time or costs associated with an in-person evaluation for physical measurements of body weight, blood pressure or lab work.

Moreover, our findings have implications for employers and worker compensation insurers. Programs or incentives aimed at reducing cardiovascular risk factors in employees may effectively reduce costly shoulder injuries and disorders related settlements and payouts. Similarly, a reduction in shoulder injuries implies fewer days away from work. This study also has implications for delineating occupational and non-occupational risk factors in individuals with shoulder injuries or disorders.

The generalizability of these findings should be considered in the context of the study population. Commercial motor vehicle drivers represent a distinctive workforce that combines extended sedentary work with periods of high physical demand such as lifting, coupling trailers, and handling cargo. This occupational profile results in an elevated prevalence of cardiometabolic risk factors including hypertension, obesity, dyslipidemia, and diabetes when compared with the general working population. [48, 49] Although this group has unique

occupational exposures, the biological mechanisms that link cardiovascular disease to shoulder injury, including vascular insufficiency, microcirculatory dysfunction, and impaired tendon repair, are common across many populations. Prior research in other occupational groups such as industrial and construction workers has reported similar associations between cardiometabolic or vascular risk factors and shoulder disorders. [41, 42] In addition, studies in clinical and community samples have consistently shown relationships between cardiovascular risk factors and rotator cuff disease [22, 26, 30, 31]

While the magnitude of association observed in the present study may reflect the higher baseline cardiovascular risk among commercial drivers, the temporal and graded nature of this relationship is likely to apply to other working populations. The mechanisms identified are relevant across occupations that involve repetitive shoulder loading or limited vascular perfusion due to systemic disease. Nevertheless, extrapolation to the general population should be done with caution because commercial drivers differ in demographics, work organization, and health behaviors. Additional research in other employment sectors is needed to determine whether cardiovascular risk scores can predict musculoskeletal outcomes in broader working and community settings.

Strengths

The most meaningful strength is the longitudinal nature of these data. Demonstrating that CVD risks occurred before the shoulder injury is a required element for suggesting causation. A large, representative sample is a strength of this study because the total number of shoulder-related WC claims was minimal (0.69%). A large sample size allowed for greater

statistical power and meaningful analysis of relative risk and hazard ratios. Another strength is that data spans seven years, thus permitting the establishment of a temporal pathway from CVD risk factors to the filing of shoulder-related WC claims. The dataset from which the data comes is another strength because it is data from one large trucking company operating in all 48 states. Moreover, the data collected in the initial CDME of each driver is high-quality, physician-reviewed data, thus increasing confidence in the exam and physician comments used to create the Expanded Framingham Score. Furthermore, the EFHS and "No test" FHS is based on the Framingham CVD risk score, a previously validated CVD risk assessment tool.

Limitations

The CDME records used to identify and assess CVD risk factors in the drivers of this dataset did not consistently capture smoking status and blood cholesterol levels. Though efforts were made to capture these data, the data collected from the exam and physician comments for tobacco showed that only 2.5% of the sample smoked, which vastly underrepresents the actual percentage of smokers in this population. There are also potential confounders that could not control for, including daily physical activity and diet. Physical activity may be a potential confounder since it is generally protective against CVD. Diet also has a significant influence on CV health.

Although the use of an adapted cardiovascular risk index is supported by prior literature, calibration drift can occur when transporting scores across populations. We therefore emphasize internal validation and model calibration assessment and we provide sensitivity analyses with alternative specifications. Residual bias due to unmeasured confounding or data

not missing at random remains possible, although multiple imputation and robustness checks reduce this concern.[7, 45, 46]

Further Research

Future work should validate and extend the findings of this study in broader populations and examine the biological mechanisms linking cardiovascular disease (CVD) and shoulder disorders. Prospective studies and randomized controlled trials aimed at reducing modifiable CVD risk factors such as hypertension, diabetes, and hyperlipidemia could determine whether cardiovascular risk reduction decreases the incidence of shoulder injuries.

The cardiovascular risk scores developed in this analysis, including the Expanded Framingham Score, Modified Framingham Score, and history-based version, should be externally validated in other occupational and community cohorts. Testing these models among construction, manufacturing, healthcare, and office workers, as well as in population-based samples, will clarify their predictive performance and calibration across diverse settings. Prior studies have reported consistent associations between cardiometabolic conditions and shoulder pathology, including hypertension and larger rotator cuff tears, hyperlipidemia and adhesive capsulitis, diabetes and shoulder stiffness, metabolic syndrome and rotator cuff pain, and systemic atherosclerosis and tendon degeneration.[26, 28, 30-32] External validation of these adapted scores will determine whether their predictive value extends beyond commercial drivers.

Further research should also investigate biological mechanisms that explain these associations. Experimental and imaging studies assessing tendon perfusion, vascular structure, and markers of inflammation or endothelial dysfunction could confirm whether impaired microcirculation

mediates the observed relationships.[33, 34] Integrating adapted cardiovascular risk tools into occupational health surveillance systems could support early identification of workers at elevated risk for both cardiovascular and musculoskeletal disorders, allowing prevention strategies that address systemic and localized pathways of disease.

Conclusion

Shoulder injuries and disorders are costly to employers and employees alike. Previous observational studies suggest that CVD and shoulder injuries are related. CMV drivers' work ranges from highly sedentary work to heavy physical labor. These extremes in physical activity heighten drivers' risk of CVD and musculoskeletal injury. The results of our analyses strongly suggest a causal relationship between increased CV risk factors and shoulder injuries and disorders.

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Tables

TABLE 1 - Descriptive Statistics			
Variable Frequency (%) or Mean (SD)	**Shoulder WC Claim (n=238, 0.69%)**	**No Shoulder WC Claim (n=34131, 99.31%)**	**Total (n=34369, 100%)**
Male	227 (95.38%)	32587 (95.48%)	32841 (95.48%)
Female	11 (4.62%)	1544 (4.52%)	1555 (4.52%)
Age (*years*)	47.21 (9.00 yrs)	45.84 (10.27 yrs)	45.85 (10.26 yrs)
Body Mass Index (BMI)	33.7 (7.0)	31.7 (7.1)	31.7 (7.1)
Mean Systolic Blood Pressure (SBP)	127.7 (12.6)	126.3 (12.6)	126.3 (12.6)
Diabetes Melitus (DM)	26 (10.92%)	2339 (6.85%)	2365 (6.88%)
Smoker*	3 (1.26%)	834 (2.44%)	846 (2.46%)
Coronary Artery Disease (CAD)*	4 (1.68%)	195 (0.57%)	199 (0.57%)
History of Stroke*	0 (0%)	50 (0.15%)	50 (0.15%)
History of Myocardial Infaction (MI)*	5 (2.10%)	314 (0.93%)	319 (0.92%)
History of Angioplasty*	0 (0%)	97 (0.28%)	97 (0.28%)
History of Coronary Stent*	4 (1.68)	176 (0.52%)	180 (0.52%)
History of Coronary Artery Bypass Graft (CABG)*	2 (0.84%)	209 (0.61%)	211 (0.61%)
Treated Blood Pressure (1=> medications)*	60 (25.21%)	5461 (16.00%)	5521 (16.06%)
Treated Blood Pressure (2=> medications)*	6 (2.52%)	678 (1.99%)	684 (1.99%)
Treated Cholesterol*	24 (10.08%)	2819 (8.26%)	2843 (8.27%)
Variables with * are from exam and physician comments.			

TABLE 2 - Expanded, Modified, "No Test" Framingham Scores --Relative Risk (RR)

	Expanded Framingham Score				Modified Framingham Score				"No Test" Framingham Score			
Variable	RR (crude)	95% CI	RR* (adjusted)	95% CI	RR (crude)	95% CI	RR* (adjusted)	95% CI	RR (crude)	95% CI	RR* (adjusted)	95% CI
Continuous	**1.037**	**(1.017, 1.058)**	**1.037**	**(1.007, 1.068)**	**1.047**	**(1.017, 1.078)**	**1.081**	**(1.008, 1.160)**	**1.037**	**(1.018, 1.058)**	**1.038**	**(1.009, 1.067)**
Categories												
(Lowest Risk of CVD) 0	1.75	(0.59, 5.21)	1.67	(0.55, 5.06)	1.71	(0.58, 5.09)	1.58	(0.52, 4.83)	1.18	(0.63, 2.23)	1.03	(0.54, 1.99)
1	1.69	(0.57, 5.01)	1.74	(0.58, 5.16)	1.64	(0.55, 4.84)	1.65	(0.55, 4.90)	2.58	(0.59, 11.25)	1.97	(0.42, 9.28)
2	1.00	(Reference)	1.00	(Reference)	1.00	(Reference)	1.00	(Reference)	1.00	(Reference)	1.00	(Reference)
3	2.67	(0.92, 7.73)	**3.34**	**(1.14, 9.74)**	2.68	(0.93, 7.74)	**3.36**	**(1.15, 9.79)**	**1.94**	**(1.09, 3.47)**	**2.24**	**(1.23, 4.07)**
4	**3.00**	**(1.06, 8.50)**	**3.99**	**(1.38, 11.57)**	**3.14**	**(1.12, 8.84)**	**4.23**	**(1.46, 12.24)**	**2.11**	**(1.18, 3.77)**	**2.70**	**(1.44, 5.08)**
5	2.49	(0.82, 7.56)	**3.20**	**(1.03, 9.96)**	2.44	(0.80, 7.42)	**3.19**	**(1.02, 9.99)**	1.57	(0.46, 5.36)	1.75	(0.50, 6.19)
6	**3.81**	**(1.37, 10.58)**	**5.64**	**(1.93, 16.5)**	**3.72**	**(1.34, 10.30)**	**5.69**	**(1.90, 16.95)**	**2.55**	**(1.44, 4.52)**	**3.64**	**(1.85, 7.16)**
7	**3.32**	**(1.12, 9.81)**	**5.42**	**(1.69, 17.32)**	**3.28**	**(1.11, 9.65)**	**5.69**	**(1.74, 18.60)**	**1.92**	**(1.04, 3.52)**	**2.79**	**(1.35, 5.78)**
8	**2.85**	**(1.01, 8.02)**	**5.03**	**(1.59,15.91)**	2.75	(0.96, 7.84)	**5.13**	**(1.55, 16,95)**	1.35	(0.68, 2.68)	2.19	(0.94, 5.14)
(Highest Risk of CVD) 9	**3.67**	**(1.29, 10.45)**	**6.70**	**(2.03, 22.12)**	**3.34**	**(1.17, 9.51)**	**6.77**	**(1.93, 23.73)**	**2.97**	**(1.69, 5.23)**	**4.85**	**(2.18, 10.79)**

**Adjusted for age, gender, BMI. Bolded values indicate p = 0.05.*

TABLE 3 - Expanded, Modified, and "No test" Framingham Scores --Hazard Ratio (HR)												
	Expanded Framingham Score				Modified Framingham Score				"No Test" Framingham Score			
Variable	HR (crude)	95% CI	HR* (adjusted)	95% CI	HR (crude)	95% CI	HR* (adjusted)	95% CI	HR (crude)	95% CI	HR* (adjusted)	95% CI
Continuous	**1.026**	**(1.006, 1.047)**	1.025	(0.995, 1.057)	**1.035**	**(1.005, 1.066)**	1.061	(0.989, 1.139)	**1.026**	**(1.006, 1.047)**	1.025	(0.996, 1.055)
Categories												
(Lowest Risk of CVD) 0	1.77	(0.59,5.25)	1.77	(0.58, 5.35)	1.67	(0.56, 4.95)	1.58	(0.52, 4.80)	1.17	(0.62, 2.20)	1.08	(0.56, 2.07)
1	1.53	(0.52, 4.50)	1.60	(0.54, 4.73)	1.41	(0.48, 4.15)	1.41	(0.48, 4.18	2.49	(0.58, 10.78)	1.71	(0.36, 8.16)
2	1.00	(Reference)	1.00	(Reference)	1.00	(Reference)	1.00	(Reference)	1.00	(Reference)	1.00	(Reference)
3	2.73	(0.94, 7.89)	**3.15**	**(1.08, 9.18)**	2.58	(0.90, 7.88)	**2.98**	**(1.02, 8.66)**	**1.98**	**(1.11, 3.54)**	**2.16**	**(1.19, 3.94)**
4	2.80	(0.99, 7.92)	**3.46**	**(1.20, 10.04)**	2.80	(0.99, 7.88)	**3.51**	**(1.21, 10.16)**	**2.02**	**(1.13, 3.60)**	**2.38**	**(1.26, 4.49)**
5	2.21	(0.73, 6.72)	2.61	(0.837, 8.14)	2.15	(0.71, 6.53)	2.60	(0.83, 8.15)	1.59	(0.47, 5.43)	1.53	(0.43, 5.39)
6	**3.21**	**(1.16, 8.90)**	**4.22**	**(1.44, 12.39)**	**2.99**	**(1.08, 8.30)**	**4.05**	**(1.36, 12.00)**	**2.32**	**(1.31, 4.11)**	**2.95**	**(1.49, 5.82)**
7	2.92	(0.99, 8.64)	4.05	**(1.27, 12.94)**	2.83	(0.96, 8.31)	**4.30**	**(1.31, 14.13)**	1.68	(0.92, 3.09)	**2.13**	**(1.03, 4.43)**
8	2.33	(0.83, 6.57)	**3.45**	**(1.08, 11.00)**	2.09	(0.73, 5.96)	**3.34**	**(1.01, 11. 09)**	1.18	(0.56, 2.33)	1.61	(0.68, 3.78)
(Highest Riskof CVD) 9	**2.90**	**(1.02, 8.24)**	**4.29**	**(1.30, 14.14)**	2.71	(0.95, 7.71)	**4.59**	**(1.30, 16.16)**	**2.4**	**(1.36, 4.21)**	**3.26**	**(1.47, 7.22)**

**Adjusted for age, gender, BMI. Bolded values indicate p = 0.05.*

STROBE Statement—Checklist of items that should be included in reports of ***cohort studies***

	Item No	Recommendation	Page No
Title and abstract	1	(*a*) Indicate the study's design with a commonly used term in the title or the abstract	1
		(*b*) Provide in the abstract an informative and balanced summary of what was done and what was found	1, 2-4
Introduction			
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported	1-2
Objectives	3	State specific objectives, including any prespecified hypotheses	2
Methods			
Study design	4	Present key elements of study design early in the paper	2-3
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection	2-3
Participants	6	(*a*) Give the eligibility criteria, and the sources and methods of selection of participants. Describe methods of follow-up	2-3
		(*b*) For matched studies, give matching criteria and number of exposed and unexposed	
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable	3-4
Data sources/ measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group	2-4
Bias	9	Describe any efforts to address potential sources of bias	3-4, 6
Study size	10	Explain how the study size was arrived at	2-3
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why	3-4
Statistical methods	12	(*a*) Describe all statistical methods, including those used to control for confounding	4
		(*b*) Describe any methods used to examine subgroups and interactions	
		(*c*) Explain how missing data were addressed	
		(*d*) If applicable, explain how loss to follow-up was addressed	
		(*e*) Describe any sensitivity analyses	
Results			
Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed	4-5
		(b) Give reasons for non-participation at each stage	

		(c) Consider use of a flow diagram	
Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders (b) Indicate number of participants with missing data for each variable of interest (c) Summarise follow-up time (eg, average and total amount)	5
Outcome data	15*	Report numbers of outcome events or summary measures over time	5
Main results	16	(*a*) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear which confounders were adjusted for and why they were included (*b*) Report category boundaries when continuous variables were categorized (*c*) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period	5
Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses	5
Discussion			
Key results	18	Summarise key results with reference to study objectives	5-6
Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias	6
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence	6
Generalisability	21	Discuss the generalisability (external validity) of the study results	6-7
Other information			
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based	Title page

*Give information separately for exposed and unexposed groups.

Note: An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at http://www.plosmedicine.org/, Annals of Internal Medicine at http://www.annals.org/, and Epidemiology at http://www.epidem.com/). Information on the STROBE Initiative is available at http://www.strobe-statement.org.

Supplemental Data

Appendix 1

Expanded Framingham Heart Scale (Men)									
Points	**Age in yrs**	***CV History***	***Heart surgery***	**SBP (untreated)**	**SBP (treated)**	**Diabetic**	***HTN meds***	**Smoking**	***Poor cholesterol***
-2	21 - 24			<120					
-1	25 - 29								
0	30 - 34			120 - 129	<120	No	1 medication	No	No
1				130 - 139					1 medication/ borderline
2	35 - 39			140 - 159	120 - 129		2 medication		2+ medications
3				160+	130 - 139	Yes	3+ medications		
4					140 - 159			Yes	
5	40 - 44				160 +				
6	45 - 49								
7									
8	50 - 54								
9									
10	55 - 59	CAD	Angioplasty						
11	60 - 64	Hx of Stroke	Stent						
12	65 - 69	Hx of MI							
13			CABG						
14	70 - 74								
15	75 +								

Modified Framingham Heart Scale (Men)					
Points	**Age in yrs**	**SBP (untreated)**	**SBP (treated)**	**Diabetic**	**Smoking**
-2	21 - 24	<120			
-1	25 - 29				
0	30 - 34	120 - 129	<120	No	No
1		130 - 139			
2	35 - 39	140 - 159	120 - 129		
3		160+	130 - 139	Yes	
4			140 - 159		Yes
5	40 - 44		160 +		
6	45 - 49				
7					
8	50 - 54				
9					
10	55 - 59				
11	60 - 64				
12	65 - 69				
13					
14	70 - 74				
15	75 +				

"No test" Framingham Heart Scale (Men)							
Points	**Age in yrs**	***CV History***	***Heart surgery***	***HTN meds***	**Diabetic**	**Smoking**	***Poor cholesterol***
-2	21 - 24						
-1	25 - 29						
0	30 - 34				No	No	No
1							1 medication/ boarderline
2	35 - 39						2+ medications
3				1 medication	Yes		
4				2 medications		Yes	
5	40 - 44			3+ medications			
6	45 - 49						
7							
8	50 - 54						
9							
10	55 - 59	CAD	Angioplasty				
11	60 - 64	Hx of Stroke	Stent				
12	65 - 69	Hx of MI					
13			CABG				
14	70 - 74						
15	75 +						

Appendix 2

Table of terms, synonyms, and misspellings searched			
Risk Factor search terms and synonyms		**HTN Medications search terms**	**Cholesterol Medications search terms**
Stroke	**Coronary Artery Bypass Graft**	Lisinopril	HCTZ
TIA	CABG	Prinivil	atorvastatin
CVA	bypass	Zestril	Lipitor
	by pass	Metoprolol	Colesevelam
Myocardial Infarction	heart surgery	Lopressor	WelChol
MI	open heart	Toprol XL	Ezetimibe
infarction		Amlodipine	Zetia
heart attack	**Smoking**	Norvasc	Fenofibrate
angina	tobacco	Losartan	Tricor
chest pain	smoke	Cozaar	Triglide
	smoking	Hydrochlorothiazide	Trilipix
Coronary Artery Disease	SMKR	Hydrodiuril	gemfibrozil
CAD		Microzide	Lopid
artheriosclerosis	**Poor Cholesterol**	Furosemide	lovastatin
arteriosclerosis	cholesterol	Lasix	Altoprev
	dyslipidemia	Carvedilol	Mevacor
Angioplasy	hyperlipidemia	Coreg	niacin
angioplasty	hypercholesterolemia	Atenolol	Niacor
		Tenormin	Niaspan
Stent		Spironolactone	Nicolar
stent		Aldactone	Pravastatin
stint		Clonidine	Pravachol
		Catapres	Rosuvastatin
			Crestor
			Simvastatin
			Zocor

Appendix 3

Expanded Framingham Score Deciles				
Raw score	Number of individuals with that raw score	Decile	Decile total	% of total
-2	58			
-1	987			
0	2758	0	3803	11.07%
1	1073			
2	324			
3	2765	1	4162	12.11%
4	1568	2	1568	4.56%
5	3385	3	3385	9.85%
6	4195	4	4195	
7	2213	5	2213	6.44%
8	2852			
9	2109	6	4961	14.43%
10	2137	7	2137	6.22%
11	2268			
12	1529			
13	1036	8	4833	14.06%
14	834	9	3112	9.05%
15	614		34369	100.00%
16	401			
17	271			
18	219			
19	119			
20	63			
21	53			
22	51			
23	44			
24	45			
25	29			
26	34			
27	27			
28	25			
29	19			
30	22			
31	12			
32	19			
33	31			
34	19			
35	18			
36	24			
37	19			
38	17			
39	17			
40	17			
41	21			
42	10			
43	8			
44	4			
45	1			
46	5			
	34369