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● *Original Contribution*

# ARTERIAL COMPRESSION DURING OVERHEAD THROWING: A RISK FOR ARTERIAL INJURY?

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**Abstract—Case studies reporting aneurysm formation in the axillary artery have been described in overhead throwing athletes, possibly due to repetitive arterial compression by the humeral head that has been transiently observed during sonographic diagnostic arm manoeuvres. Whether compression negatively alters arterial health has not been investigated and was the focus of this study. The throwing arm of elite overhead athletes was screened for inducible axillary artery compression. Compressors (COMP, $n$ = 11, mean age: 20 (SD: 2) year, 7 male, 4 female) were age and sex matched with noncompressing (NONCOMP) athlete controls. Four indices of arterial health (flow mediated dilation [FMD], conduit artery vasodilatory capacity [CADC], glyceryl-trinitrate [GTN]-induced vasodilation and intima-media thickness [IMT]) were assessed with high-resolution ultrasound at the brachial and the axillary, artery. No significant between-group differences were observed at the brachial, or axillary, artery for FMD (brachial: COMP: mean (SD) 6.2 (3.1)%, NONCOMP: 6.1 (3.5)%, $p$ = 0.967, axillary: COMP: 8.0 (5.5)%, NONCOMP: 9.0 (3.6)%, $p$ = 0.602), CADC (brachial: COMP: 10.4 (3.4)%, NONCOMP: 10.4 (5.4)%, $p$ = 0.999, axillary: COMP: 9.6 (4.2)%, NONCOMP: 8.5 (3.2)%, $p$ = 0.492), GTN-induced vasodilation (brachial: COMP: 17.9 (5.1)%, NONCOMP:14.1 (7.2)%, $p$ = 0.173, axillary: COMP: 9.5 (4.3)%, NONCOMP: 7.7 (3.1)%, $p$ = 0.302) or IMT (brachial: $p$ = 0.084, axillary: $p$ = 0.581). These results suggest that transient arterial compression, observed during diagnostic arm manoeuvres in overhead throwing athletes, is not associated with abnormal indices of artery function or structure and that other mechanisms must be responsible for the published cases of aneurysm formation in elite athletes performing overhead throwing actions. (E-mail: c.stapleton@ljmu.ac.uk)** **© 2010 World Federation for Ultrasound in Medicine & Biology.**

*Key Words:* **Vascular compression, Endothelial function, Elite athletes, Overhead throwing, Flow mediated dilation, Intima media thickness.**

## INTRODUCTION AND LITERATURE

Case reports in overhead throwing athletes have documented a continuum of findings from intermittent compression of the axillary artery and its branches, with the arm in an overhead position, to thrombosis, aneurysm formation and peripheral embolisation (Dijkstra and Westra 1978; Kee et al. 1995; Schneider et al. 1999). No epidemiologic data exists for the occurrence of thrombus or aneurysms in overhead throwing athletes, however, Rohrer et al. (1990) reported clinically significant inducible compression (>50% reduction in diameter) in 8% of baseball players. The cause of compression is unclear but excessive translation of the humeral head at the glenohumeral joint and/or a hypertrophied or tight pectoralis minor muscle, combined with repetitive overhead arm motion, have been implicated as the cause of transient compression at the third and second portions of the axillary artery, respectively (Dijkstra and Westra 1978; Stapleton et al. 2008). Repetitive compression is thought to result in damage to the arterial wall, including the endothelial layer with subsequent thrombus and/or aneurysm formation (Schneider et al. 1999; Stapleton et al. 2009). However, the proposed link between repetitive arterial compression with overhead arm motion and arterial health in elite overhead throwing athletes remains speculative. Our group recently studied the arterial health of eight nonathletic individuals who did not participate in activities requiring repetitive overhead activity but who did demonstrated such compression and compared them

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with age and sex matched noncompressor controls. Mean flow-mediated dilation (FMD), an index of conduit artery endothelial function (Kooijman et al. 2008; Mullen et al. 2001), assessed downstream to the site of compression revealed a significantly ($p = 0.006$) lower response in the 'compressor' group (mean (SD): 6.38 (3.28) vs. 10.38 (2.74)%) (Stapleton et al. 2009). These results suggest that the finding of clinically significant inducible axillary artery compression maybe of pathologic significance, potentially inducing endothelial dysfunction, an early sign of compromised vascular health and a potential precursor for the more serious arterial injuries described in published case reports (Dijkstra and Westra 1978; Kee et al. 1995; Schneider et al. 1999).

Therefore, the aim of this investigation is to investigate if performance of repeated, transient arterial compression in an athletic population results in similar changes in arterial function and structure?

## MATERIALS AND METHODS

*Subjects*

The Great Britain (GB) men's and women's waterpolo squads were recruited. Inclusion criteria included participation in the squad's full training programme for at least 2 months prior to testing. However, all subjects had been competing in waterpolo for at least 5 years. All male and female squad members undertook the same training routine with two pool sessions a day, 5 days a week and an additional strength and conditioning session, 2 days of the week. Subjects provided written informed consent prior to completing a health screening questionnaire to exclude those with factors known to influence the indices of vascular health, flow mediated dilation (FMD) and conduit artery vasodilatory capacity (CADC) response, *e.g.* diabetes, asthma, amenorrhea, recent allergic reactions, infections or injuries. Subjects underwent subjective and objective musculoskeletal screening to exclude any past or present musculoskeletal injury that may predispose them to neurovascular compression. The presence or absence of the Arch of Langer (an anomalous musculotendinous slip) was assessed by visual inspection and palpation. Ethical approval was granted from Liverpool John Moores University ethics committee.

All subjects ($n = 37$, mean age (SD): 20 (3) years, sex: 24 males, 13 females) were screened for clinically significant inducible axillary artery compression. *A priori* sample size estimation ($n = 8$ per group) was based on detecting a clinically significant difference in FMD at the brachial artery of 2.5%, with an alpha level of 0.05 and a power of 80% (Woodman et al. 2001). Measurement of FMD at the brachial artery was used for sample size estimation as it has been used extensively in cardiovascular research and has been validated as an indicator of arterial health (Celermajer et al. 1992; Hashimoto et al. 2003; Tsuchiya et al. 2007).

*Data collection – Phase 1: Screening for clinically significant inducible axillary artery compression*

Following 10 min rest in the supine position to stabilise heart rate and blood flow, the subjects' dominant (throwing) arm was passively supported in the baseline position (60° abduction) or the stress position (120° abduction, 30° horizontal extension and 90° external rotation) (Fig. 1). The axillary artery was located and imaged at the axilla and, therefore, it was necessary to incorporate some abduction into the baseline position to (1) accommodate the ultrasound transducer and (2) to optimise image clarity. To aid standardisation of the stress position, the posterior corner of the acromion was positioned over a marker on the treatment table and the arm supported in a custom made arm rest. A high-resolution ultrasound machine (Terason t3000; Teratech, MA, USA) with an 8-10 MHz linear array transducer was used to record arterial diameter and peak systolic flow velocity (PSV) for 1 min in each position. Post-test analysis was performed with wall tracking and edge detection computer software (LabVIEW 7.0; Version 3, National Instruments, Austin, TX). The software (Woodman et al. 2001) produces a mean diameter and a mean PSV from the pre-recorded 60 s of data. A doubling of PSV with the arm in the stress position compared with the baseline position is indicative of a 75% reduction in the cross-sectional area, which is classified as clinically significant arterial compression (Fig. 2a and b; Strandness 2002). Subjects demonstrating this degree of compression formed the compressor (COMP) group whereas the noncompressor (NONCOMP) group comprised of subjects demonstrating less than a 20% rise in PSV with the arm in the stress position

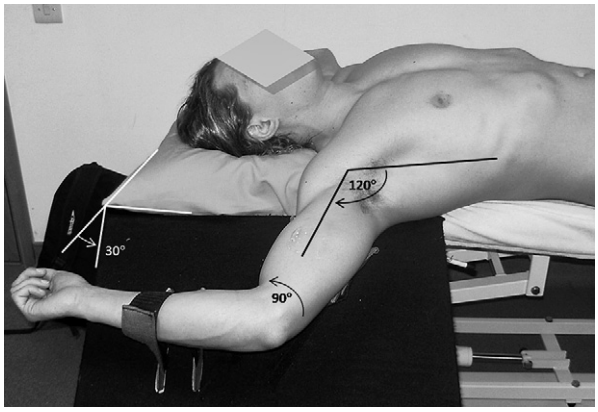


Fig. 1. The subject is pictured in the supine position with the dominant arm passively supported, with a purpose built arm rest, in the stress position (120° abduction, 30° horizontal extension and 90° external rotation).

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