

# Blood pressure in professional male football players in Norway

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**Objectives:** Scarce data exist on blood pressure (BP) and secondary effects in young athletes. Hence, we aimed to study prevalence of high BP ( $\geq 140/90$  mmHg) and the association to arterial compliance, left ventricle mass and left atrium volume.

**Methods:** A Norwegian preparticipation cardiac screening of male professional football players enrolled 493 white European, 47 matched controls, 49 black and 53 players of other ethnicity. BP was measured as a mean of two measurements. Height and weight were self-reported, and body surface area (BSA) was calculated. The echocardiographic parameters were indexed to BSA. Heart rates (HRs) by electrocardiography and pulse pressure (PP) were considered as surrogates for sympathetic activity. Arterial compliance was calculated as  $\text{stroke volume}_{\text{BSA}}/\text{PP}$ .

**Results:** The players mean age was 25 years (18–38) and mean BP  $122/69 \pm 11/8$  mmHg. There were no significant differences in prevalence of hypertension between all players, 39 (7%), and controls, four (9%), or between white, 32 (7%), and black, five (10%), players. There was a significant positive linear relationship between BP and left ventricle mass<sub>BSA</sub>, left atrium volume<sub>BSA</sub>, stroke volume<sub>BSA</sub>, HR and PP, and negative relationship to arterial compliance<sub>BSA</sub>.

**Conclusion:** Although the prevalence of high BP in professional football players was low, our data indicate a novel association between elevated BP and reduced arterial compliance, increased left ventricle mass and left atrium volume even in young athletes. This emphasizes closer focus on BP measurements and standardized follow-up after preparticipation screening of athletes.

**Keywords:** athletes, blood pressure, echocardiography, football, heart rate, mass screening

**Abbreviations:** BP, blood pressure; BSA, body surface area; HR, heart rate; LAESV, left atrium end-systolic volume; LVIDd, left ventricle internal dimensions; LVMI<sub>BSA</sub>, left ventricle mass indexed to BSA; MAP, mean arterial pressure; PP, pulse pressure; PWTd, posterior wall thickness in/at end diastole; RWT, relative wall thickness; SAC, systemic arterial compliance; SV, stroke volume

## INTRODUCTION

In western countries the prevalence of hypertension is reported to be 14.4 and 21.2% in men aged 20–29 and 30–39 years, respectively [1]. The prevalence of hypertension in physically active people and athletes, however, has been considered to be lower than in the general population [2]. The main background for this knowledge is an investigation from 1979 to 1983 in which 810 sportsmen, aged 14–69 years, had their blood pressure (BP) measured once and were compared with population studies in Germany [3]. Hypertension was defined according to the guidelines of that time in the World Health Organization's report from 1962 [4]. All other BP reports from athletes come from preparticipation screening programs with poor focus on BP recordings [5], wherein hypertension has not been defined according to contemporary guidelines from the European Society of Hypertension [6], which since 1993 have required repeated BP measurements. Finally, the prevalence of hypertension depends on ethnicity and is higher for blacks than for whites in both the USA and the UK [7]. Whether this is also the case for athletes is not known.

Several studies have investigated left ventricular changes and demonstrated increased left ventricular mass in athletes [8]. To our knowledge, however, no studies have investigated the association between BP and left ventricular mass in athletes, which is considered as subclinical organ damage in patients with hypertension [9,10]. In the general population, left atrium size increases with BP [10–12]. Whether this also occurs in athletes with high BP is

Journal of Hypertension 2013, 31:000–000

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Received 5 July 2012 Revised 14 November 2012 Accepted 8 January 2013

J Hypertens 31:000–000 © 2013 Wolters Kluwer Health | Lippincott Williams & Wilkins.

DOI:10.1097/HJH.0b013e3283285eb5fe

unknown. Nor is systemic arterial compliance (SAC), which is reduced in patients with hypertension [13] studied in young athletes with high BP.

High heart rate (HR) and pulse pressure (PP) have shown to be strong predictors for cardiovascular disease, and are both associated with increased sympathetic activity [14,15]. In addition, increased HR also predicts development of essential hypertension [14].

Hence, the main aim of this study was to describe the prevalence of high BP in male professional football players and the associations of BP to HR, PP, left ventricular mass, left atrium size and SAC. Secondly, we wanted to investigate differences in these parameters between hypertensive versus normotensive football players, white European players versus controls, and white versus black players.

## METHODS

### Participants

During the winter 2008, the Norwegian male professional football players underwent mandatory preparticipation cardiac screening [16] when attending a training camp in La Manga, Spain. Players aged from 18 to 40 years were invited to participate in the present study. Male controls of the same age, exercising three or less times per week were selected among the teams' staff or as volunteers from the Oslo region. Athletes and controls responded to a questionnaire regarding their height and weight, if they used snuff every day or the presence of familial hypertension or diabetes in a first-degree relative under the age of 55 years. Also, they were asked whether their ethnicity were white, Afro-American (black) or other. When in doubt, birthplace or parents homeland were used. If the parents came from different continents, the players were categorized as 'mixed' ethnicity. The study was approved by the Regional Ethical Committee.

### Blood pressure and heart rate measurements

The examinations were preferably conducted before exercise. Owing to logistical reasons some of the players exercised before the examination; however, at least 1 h rest was requested. BP was measured by trained personnel after 5 min rest in a sitting position, using a validated automatic BP monitor (Dinamap ProCare DPC300N; GE, Milwaukee, Wisconsin, USA). SBP and DBP were registered as the mean of two consecutive BP measurements and categorized according to European Society of Hypertensions' guidelines [6]. High BP was defined as SBP at least 140 mmHg or DBP at least 90 mmHg. Mean arterial pressure (MAP) was defined as  $DBP + 1/3(SBP - DBP)$ , PP was calculated as the difference between SBP and DBP and SAC as stroke volume (SV) from the two-dimensional echo recordings divided with PP. HR was derived from the electrocardiogram, which was taken in the supine position with ClickECG (Cardiette Cardioline, Milan, Italy).

### Echocardiographic parameters

All echocardiographic recordings were performed with a 2.5-MHz transducer (Vivid 7 and Vivid i; GE Vingmed Ultrasound AS, Horten, Norway) within 30 min after the BP and HR measurements. The digital data were transferred to a computer (Dell Optiplex 755; Dell A/S, Copenhagen,

Denmark) for off-line analysis at the core echolab, Oslo University Hospital, Aker, with the software EchoPAC (BT08) (GE Vingmed Ultrasound AS). The echocardiographic recordings were performed from standard parasternal long-axis and short-axis and apical views. Left ventricular internal dimension (LVID) and posterior wall thickness (PWT) were measured at end diastole as recommended by the American Society of Echocardiography [17,18]. Left ventricular masses were calculated by the formula of Devereux [17]. Left ventricular volumes were measured by modified Simpson's rule from biplane two-chamber and four-chamber views [17,19]. From these data, SV was calculated. Relative wall thickness (RWT) was calculated by  $2 \times (PWT \text{ at end diastole}) / LVID$  [17,20]. Left atrium volume (LAESV) was measured at end systole from the apical two-chamber and four-chamber views by the method of area-length [17]. Data regarding chamber quantification were indexed to body surface area (BSA) according to recommendations [17].

The echocardiographic analyses were all conducted by one of the authors (G.F.G.) who were blinded for the BP results.

### Data analyses

The players were divided into four subgroups according to their office BP [6]. All controls were white Europeans, and they were compared with white European players only to make the comparisons more homogeneous. All white players were compared with all black players, but not to players of other or mixed ethnicity because this category was too heterogeneous. The players were also divided according to their position in the team, as goalkeepers or outfield players. Based on the match-schedule from La Manga, they were also divided according to whether their team had played a match in the evening (1600 or 1900 h) the day before their examinations or not.

LVID, PWT, left ventricular mass, LAESV, SV and SAC were all indexed to BSA, calculated from height and weight. The outcome variables for this study were SBP, DBP, MAP, PP, HR,  $LVID_{BSA}$ ,  $PWT_{BSA}$ , left ventricle mass indexed to BSA ( $LVMI_{BSA}$ ), RWT,  $LAESV_{BSA}$ ,  $SV_{BSA}$  and  $SAC_{BSA}$ . Differences between two subgroups were analysed using *t*-tests for continuous variables, and  $\chi^2$  or Fisher's exact tests for categorical variables (Table 1). Differences in means between all BP categories were calculated using analysis of variance test for trend for continuous variables, and linear-by-linear analysis for categorical variables (Table 2). Differences in means between the first four subgroups were calculated using the Bonferroni procedure to control the risk of a type I error (Table 2). Linear regression analysis was used for correlation between two continuous variables. Data are presented as mean  $\pm$  standard deviation or as numbers (percentages).  $P < 0.05$  was considered statistically significant, and all tests were two-tailed. All statistical analyses were conducted using SPSS (PASW Statistics 18; IBM Corporation 2010, New York, New York, USA).

## RESULTS

Twenty-eight of the 30 Norwegian male professional teams participated in the study, and 595 of 604 eligible players

**TABLE 1. Demographic characteristics of white male European football players and controls**

Characteristics	Controls (n = 47)	Players (n = 493)	P
Age (years)	26.2 (6.5)	25.3 (4.6)	NS
Weight (kg)	77.8 (7.8)	79.3 (6.9)	NS
Height (m)	181.3 (5.8)	183.0 (6.0)	NS
BMI (kg/m <sup>2</sup> )	23.6 (1.7)	23.6 (1.2)	NS
BSA (m <sup>2</sup> )	1.98 (0.12)	2.01 (0.12)	<0.001
Office BP (mmHg)			
SBP	123.8 (10.9)	122.4 (10.8)	NS
DBP	70.8 (6.9)	69.2 (7.8)	NS
MAP	88.5 (7.0)	86.9 (7.6)	NS
PP	53.0 (10.2)	53.2 (10.0)	NS
HR (beats/min)	62.2 (10.6)	55.0 (9.4)	<0.001
Echocardiographic findings			
LVID <sub>BSA</sub> (mm/m <sup>2</sup> )	26.5 (2.1)	27.1 (2.1)	<0.05
PWT <sub>BSA</sub> (mm/m <sup>2</sup> )	4.0 (0.4)	4.5 (0.6)	<0.001
LVM <sub>BSA</sub> (g/m <sup>2</sup> )	75.0 (15.1)	90.4 (16.0)	<0.001
RWT	0.31 (0.04)	0.33 (0.06)	<0.001
LAESV <sub>BSA</sub> (ml/m <sup>2</sup> )	27.8 (10.1)	36.7 (27.8)	<0.001
SV <sub>BSA</sub> (ml/m <sup>2</sup> )	34.7 (6.9)	41.5 (34.7)	<0.001
SAC <sub>BSA</sub> (ml/m <sup>2</sup> )/mmHg	0.68 (0.19)	0.80 (0.20)	<0.001
Snuffing, yes (%)	11 (23.4)	171 (34.7)	<0.001
Familial HT, yes (%)	13 (27.7)	66 (13.4)	<0.05
Familial diabetes, yes (%)	7 (14.9)	37 (7.5)	<0.01

Values are shown as mean (standard deviation) or n(%). P value is related to independent sample t-test for continuous versus categorical variables and to  $\chi^2$ -test for categorical variables. BSA, body surface area; HT, hypertension; LAESV, left atrium end-systolic volume; LVID, left ventricular internal dimension; MAP, mean arterial pressure; NS, not significant; PP, pulse pressure; PWT, posterior wall thickness; RWT, relative wall thickness; SAC, systemic arterial compliance; SV, stroke volume.

(99%) consented to participate (Fig. 1). Forty-seven white European controls were included.

Among the players, 493 (83%) were Europeans, 52 (9%) Africans, 29 (5%) Americans, four Asians, four Australians, and 13 (2%) were of unknown origin. The skin colour was white in 504 (85%), black in 49 (8%), mixed for 13 (2%) and

other for 29 (5%) (Fig. 1). Thirteen (275 players) teams had played a match the evening before examination, whereas 15 teams (319 players) had not.

The players had a mean age of  $25.1 \pm 4.5$  years, mean BMI  $23.7 \pm 1.2$  kg/m<sup>2</sup> and mean BSA  $2.0 \pm 0.1$  m<sup>2</sup>. The mean office BP was  $122 \pm 11/69 \pm 8$  mmHg, and the mean

**TABLE 2. Comparisons of blood pressure categories for male professional football players**

	BP categories (mmHg)				P for trend
	Optimal <120/80 (n = 250)	Normal 120–129/80–84 (n = 197)	High normal 130–139/85–89 (n = 108)	High $\geq$ 140/90 (n = 39)	
Age (years)	24.8 (4.5)	25.0 (4.7)	25.3 (4.3)	26.3 (4.3)	0.072
BMI (kg/m <sup>2</sup> )	23.5 (1.2)	23.7 (1.3)	24.1 (1.2)*,†	23.7 (1.1)	<0.001
BSA (m <sup>2</sup> )	1.98 (0.12)	2.00 (0.11)	2.04 (0.12)*	2.02 (0.11)	<0.001
Office BP (mmHg)					
SBP	112.9 (5.3)	123.6 (3.2)	132.5 (4.3)	145.1 (9.4)	NC
DBP	65.8 (6.4)	70.1 (7.0)	72.8 (7.4)	78.1 (9.0)	NC
MAP	81.5 (5.2)	87.9 (4.7)*	92.6 (4.8)*,‡	100.4 (7.2)*,‡,§	<0.001
PP	47.1 (6.5)	53.5 (8.2)*	59.7 (9.4)*,‡	67.1 (11.8)*,‡,§	<0.001
HR (beats/min)	54.4 (8.9)	55.3 (10.0)	55.8 (8.2)	59.0 (9.7) <sup>  </sup>	0.008
Echocardiographic findings					
LVID <sub>BSA</sub> (mm/m <sup>2</sup> )	27.1 (2.1)	27.2 (2.2)	26.9 (2.0)	26.7 (2.5)	0.335
PWT <sub>BSA</sub> (mm/m <sup>2</sup> )	4.5 (0.6)	4.5 (0.6)	4.5 (0.6)	4.7 (0.7)	0.209
LVM <sub>BSA</sub> (g/m <sup>2</sup> )	88.5 (14.9)	91.0 (17.7)	93.1 (15.5)	95.7 (19.5)	0.001
RWT	0.34 (0.05)	0.33 (0.06)	0.34 (0.05)	0.36 (0.07)	0.109
LAESV <sub>BSA</sub> (ml/m <sup>2</sup> )	35.3 (9.7)	38.5 (10.3) <sup>  </sup>	36.7 (9.8)	38.0 (11.1)	0.046
SV <sub>BSA</sub> (ml/m <sup>2</sup> )	40.1 (8.0)	41.3 (7.8)	42.3 (8.3) <sup>  </sup>	42.3 (10.3)	0.015
SAC <sub>BSA</sub> (ml/m <sup>2</sup> )/mmHg	0.87 (0.20)	0.79 (0.18)*	0.72 (0.18)*,†	0.65 (0.22)*,†	<0.001
Snuffing, yes (%)	73 (30)	57 (29)	34 (32)	13 (33)	0.738
Familial HT, yes (%)	33 (13)	25 (13)	18 (17)	6 (15)	0.526

Values are shown as mean (standard deviation) or n(%). Differences in means between all BP categories were calculated using analysis of variance test for trend for continuous variables, and linear-by-linear analysis for categorical variables. Differences in means between the four subgroups were calculated using the Bonferroni procedure. NC indicates not computed, because office SBP and DBP values were used to categorize the individuals. BP, blood pressure; BSA, body surface area; HT, hypertension; LAESV, left atrium end-systolic volume; LVID, left ventricular internal dimension; MAP, mean arterial pressure; NS, not significant; PP, pulse pressure; PWT, posterior wall thickness; RWT, relative wall thickness; SAC, systemic arterial compliance; SV, stroke volume.

\*P < 0.001 versus optimal BP.

†P < 0.05 versus normal BP.

‡P < 0.001 versus normal BP.

§P < 0.001 versus high normal BP.

||P < 0.05 versus optimal BP.

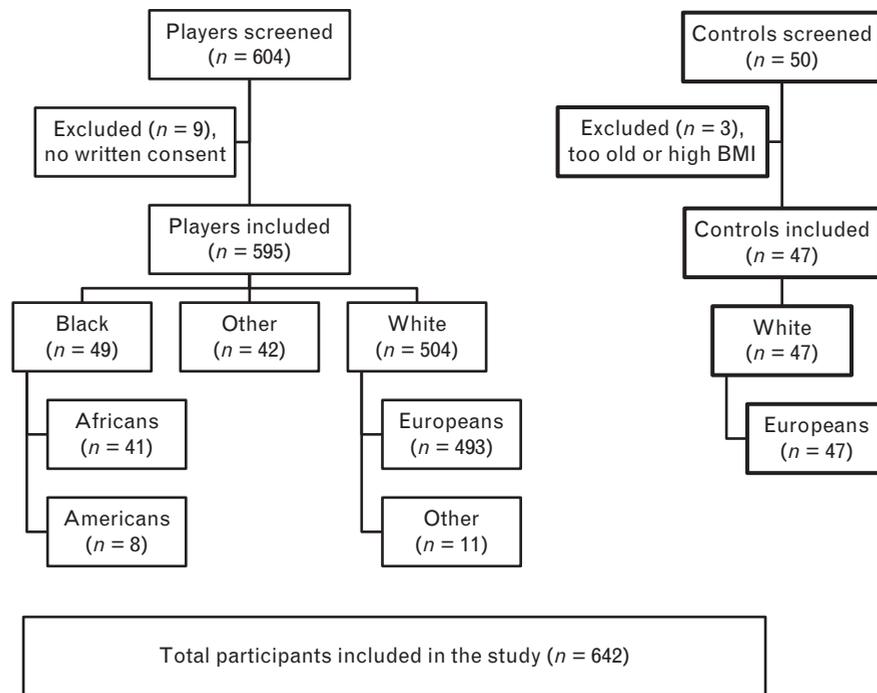


FIGURE 1 Study flow chart.

reduction between the first and second BP recordings was  $0.8 \pm 6.5$  mmHg in SBP ( $P=0.002$ ) and  $0.2 \pm 5.7$  mmHg in DBP ( $P=0.341$ ). For all differences, see Table 1, Supplemental Digital Content, <http://links.lww.com/HJH/A240>. MAP and PP were on average  $87 \pm 8$  and  $53 \pm 10$  mmHg, respectively. The HR was  $55 \pm 9$  beats/min. Altogether 250 (42%) of the players had optimal BP, 197 (33%) had normal BP, 108 (18%) had high normal BP and 39 (7%) had high BP. Isolated systolic increased BP was recorded for 169 (86%) of the players with normal BP, 97 (90%) with high normal BP and 33 (85%) with high BP. There was no significant difference in prevalence of high BP between players, 39 (7%), and controls, four (9%), between black, five (10%), and white, 32 (7%), or between players who had played a match the day before, 13 (5%) versus no match, 26 (8%). For other demographic characteristics and the echocardiographic findings of white European players and controls (see Table 1). There was no difference in BP between goalkeepers,  $122/70 \pm 10/7$  mmHg, and outfield players,  $122/69 \pm 11/7$  mmHg [ $P=0.85$  (SBP) and  $P=0.45$  (DBP)], or between players who had played a match the day before,  $122/69 \pm 11/7$  mmHg, compared to those who did not play,  $122/70 \pm 11/8$  mmHg [ $P=0.82$  (SBP) and  $P=0.08$  (DBP)].

Table 2 shows a significant positive linear relationship between increasing BP category and increasing  $LVM_{BSA}$ ,  $LAESV_{BSA}$ ,  $SV_{BSA}$ , PP and HR, and negative to  $SAC_{BSA}$  ( $P<0.05$ ) (Fig. 2). Subgroup analysis showed significant relationship between increasing BP category and the same parameters in white players (see Table 2, Supplemental Digital Content, <http://links.lww.com/HJH/A240>), but only for HR and PP ( $P<0.05$ ) in blacks. Analysing the same variables with linear regression analyses for continuous variables with SBP shows the same significant positive

relationship for BMI, BSA, MAP, PP, HR,  $LVM_{BSA}$ ,  $LAESV_{BSA}$ ,  $SV_{BSA}$  and  $SAC_{BSA}$  ( $P<0.05$ ) (see Table 3, Supplemental Digital Content, <http://links.lww.com/HJH/A240>).

Daily use of snuff was reported in 177 (30%) players, but no significant association between use of snuff and high BP was found (Table 2).

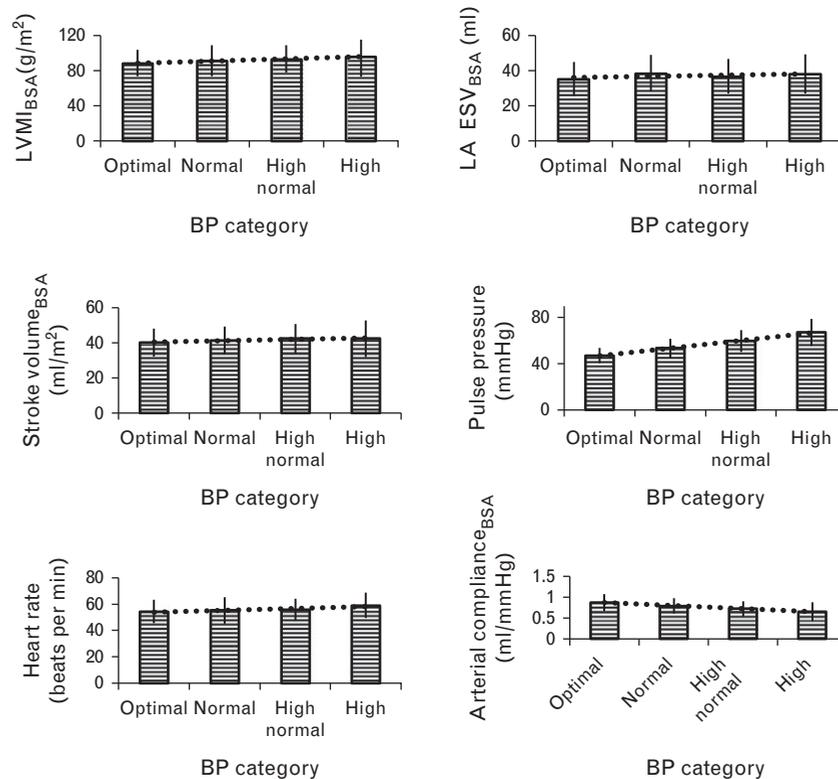
## DISCUSSION

The present study, which is the first comprehensive BP evaluation of a large homogeneous group of male professional football players in Europe, showed a low prevalence of high BP. Furthermore, in such a cohort of young athletes we showed a significant positive linear relationship between BP and  $LVM_{BSA}$  and negative to  $SAC_{BSA}$ , which both are considered as hypertensive subclinical organ damages [9,21]. Increasing BP was also associated with increasing left atrium size, and these findings have previously not been demonstrated in athletes.

### Left ventricle mass and left atrium volume

It is well known that hypertension contribute to left ventricular concentric remodelling in patients, whereas physical activity contributes to eccentric hypertrophy as in athletes' heart [22]. In the same group of football players, Gjerdalen *et al.* [23] have recently shown an increase of left ventricular mass driven by an increase in both left ventricular cavity size and wall thickness, though well within normal limits.

Several other studies have confirmed the relation between left atrium enlargement and increase of BP in older, nonathletic cohorts [11,24]. In the present study  $LAESV_{BSA}$  increased by 11% from those with optimal to



**FIGURE 2** The diagrams illustrate the significant linear relationships between mean ( $\pm$ standard deviation) for BP categories versus left ventricle mass (LVMl<sub>BSA</sub>) ( $P < 0.01$ ), left atrium end-systolic volume (LAESV<sub>BSA</sub>) ( $P < 0.05$ ), stroke volume ( $P < 0.05$ ), pulse pressure ( $P < 0.001$ ), heart rate ( $P < 0.01$ ), and systemic arterial compliance<sub>BSA</sub> ( $P < 0.001$ ) for professional male football players. BSA, body surface area.

high BP. This adds further evidence to the notion that hypertension-related effects have already taken place in these young men. There has also been increasing evidence for the link between left atrium size and atrial fibrillation in endurance athletes [25,26]. Although increased pressure overload is the cause for increased left atrium volume in those with hypertension [24], volume load is responsible for left atrium enlargement in athletes [27]. As atrial fibrillation also is associated to hypertension and reduced HR, one might speculate whether athletes with elevated BP have an increased risk of developing future atrial fibrillation as a result of the combination of pressure and volume overload [25].

### Heart rate, pulse pressure and systemic arterial compliance

Athletes usually have lower HR than physically inactive people, as demonstrated in our study,  $55 \pm 9$  versus  $62 \pm 11$  beats/min ( $P < 0.001$ ), respectively. The association between elevated BP and increased PP and HR indicates increased sympathetic activity [14,15]. An adrenergic overdrive is documented in patients with hypertension, and the sympathetic activation is directly related to the severity of the hypertensive state [28]. Whether HR itself is a risk factor for development of hypertension or just a marker for increased sympathetic activation is still a matter of debate [29]. The rise in PP (or SBP) with age in healthy individuals relates to fall in SAC, whereas elevation of PP in the young has been suggested to be related to increase in SV

[30]. The present study demonstrated a significant decrease in SAC from the optimal to high BP group of 25% (Table 2). This reduction in SAC, however, was mainly driven by the increase in PP, and has previously not been shown in such a young cohort of well trained individuals. It is, however, an established knowledge in older hypertensive populations [13]. SAC is an important factor for the left ventricle–arterial coupling, and a reduced compliance reflects increased pulsatile left ventricular afterload, which is probably one mechanism for the increased left ventricular mass and left atrium volume observed in the present study [15,24].

### Isolated increased SBP

In this study the majority of athletes with high BP had increased SBP, whereas DBP was normal. In the 1999–2004 US National Health and Nutrition Examination Survey, the prevalence of isolated systolic hypertension in males aged 25–34 years was only 2.4% [31], which is half the prevalence of isolated high SBP in the present study (5.5%). This discrepancy may also be because of spurious systolic hypertension, which could be more common in athletes with greater SV and up to 30–40 mmHg higher SBP in the brachial artery compared to the central aorta [32]. We could, however, also show a weak, but significant association between SV and SBP.

### Implications of casual high blood pressure

The clinical implication of increased BP in young athletes is important as even slight elevations in SBP during early

adulthood warrants particular concern because of increased risk for further cardiovascular disease in later life [33,34]. One single high BP measure in young age may also predict hypertension in older age. This is reported in several studies, among them 'The Bergen Blood Pressure Study' with follow-up for 27 years [35]. In a nationwide cohort study from Sweden, 1.2 million young men were followed for median 24 years after military conscription examinations, and a higher risk of cardiovascular mortality was found in those with higher BP [36].

Although comparable studies are difficult to find, healthy Norwegian football players showed less high BP than non-athletes at similar age, which coincides with most results from athletes in other countries [2]. We consider that the high inclusion rate in our study makes the results representative for male professional football players in Norway and may probably be valid for athletes in similar sporting disciplines and at similar age.

### Blood pressure and ethnicity

There was no significant difference in the prevalence of high BP between black and white players, which might be due to the low prevalence of high BP and a small number of black players, hence too low power of this study. However, a meta-analysis from the UK and the Netherlands did not show any consistency regarding BP differences in black and white people, which the authors explained by the variations in how BP was measured and the classification of black groups in some studies [37]. These findings are consistent with a review in the *Lancet* showing huge differences depending on countries of origin [1]. Our definition of ethnicity according to white and black skin colour and dependent on birthplace or parents' homeland does not include environmental factors such as country of upbringing, which also might influence BP status [38,39].

### Limitations

Our controls might represent selection bias as half of them were chosen among the teams' staff. Their mean BP was lower than in physically active people in the Netherlands, exercising more than 2 h/week with comparable mean age [32]. However, we do not consider this to have any important impact on the main results of this study.

ECG, BP and echocardiography were all recorded consecutively, but not simultaneously or in the same position. BP was recorded in a sitting position and SV in a supine position, which leads to an increase in SV. However, we do not think that this has made any substantial impact on our results, that is the SAC findings as the recordings for the SV measurements were performed after 10–15 min, when steady state is expected to have occurred. In addition to this all participants did the same procedure.

The mean difference between the two BP measurements, even less than 1 mmHg, was significant for SBP. A third recording was not scheduled, but should have been performed to get more precise measurements.

To accomplish screening without the influence of exercise during a time-limited training camp is difficult, but time between exercise and BP measurements should have been recorded as postexercise hypotension might

have contributed to false low BPs in those players who had their examinations performed in too close relation to training [40]. On the contrary, approximately half of the players had played a match the day before and should therefore reflect a group of players with little influence on BP. Moreover, there were no BP differences between this group and those who trained the same day as the examinations. Nor was there any significant difference in BP between players and controls who did not train before the examination. We therefore consider the anticipated post-exercise hypotension to be a limitation, but not a major problem in our study.

Mean office BP from only one single occasion and not repeated or supplemented by ambulatory BP measurements or radial arterial tonometry may have led to an overestimation of elevated BP because of white-coat hypertension or spurious systolic hypertension. On the contrary, our BP measurements can have been underestimated also because of masked hypertension.

In conclusion, the current study has demonstrated a low prevalence of high BP in a selected group of young male professional football players. The association between elevated BP and reduced arterial compliance, increased left ventricular mass and left atrium size, emphasize the need for closer focus on BP measurements and standardized follow-up even in these young athletes. More studies, however, including repeated BP measurements and 24-h ambulatory BP monitoring are needed to increase our knowledge of prevalence of hypertension in athletes in different sporting disciplines and ethnic groups.

### ACKNOWLEDGEMENTS

The authors would like to thank all players and controls that participated in the study, the clubs' medical teams, The Football Association of Norway, The Professional Norwegian Football League, cardiologists and nurses for collecting data and Kristoffer Steine for helping organizing the examination and the database and Professor Ingar M.K. Holme for statistical support.

The Oslo Sports Trauma Research Center has been established through generous grants from the Eastern Norway Regional Health Authority, the International Olympic Committee, the Royal Norwegian Ministry of Culture, Norsk Tipping AS and the Norwegian Olympic Committee & Confederation of Sport.

The research reported in this article was supported by Oslo Sports Trauma Research Center and Akershus University Hospital, Lørenskog, Norway.

### Conflicts of interest

There are no conflicts of interest.

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## **Reviewer's Summary Evaluation**

### **Reviewer 2**

This is a very interesting article assessing the prevalence of high blood pressure and the association to left ventricle mass in young athletes. There is a major methodological query. Blood pressure measurement for some players was made after (1 h) and not before exercise.

It is well known that post exercise blood pressure measurements can be different from resting blood pressure. Usually blood pressure levels after exercise decrease and the magnitude of the decrease is related to the intensity of exercise. Moreover the intensity of exercise in various player positions can easily affect also the percentage prevalence of hypertensive professional football players.