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Direktor: Prof. Dr. med. Bernd Wolfarth

Habilitationsschrift

„Kardiale und inflammatorische Anpassungsvorgänge: Präventive Diagnostik in der sportmedizinischen Betreuung junger Hochleistungssportler“

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von

Dr. med. Jan Christoph Wüstenfeld

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Dekan: Prof. Dr. med. J. Spranger

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Abkürzungsverzeichnis

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|---------------------|-------------------------------------------------------------------------------|
| ACSM | American College of Sports Medicine |
| AHA | American Heart Association |
| ARVC | arrhythmogene rechtsventrikuläre Kardiomyopathie |
| BSG | Blutsenkungsgeschwindigkeit |
| CMD | cranio-mandibuläre Dysfunktion |
| CRP | C-reaktives Protein |
| DBS | Deutscher Behinderten Sportbund |
| DGSP | Deutsche Gesellschaft für Sportmedizin und Prävention |
| DOSB | Deutscher Olympischer Sportbund |
| EKG | Elektrokardiogramm |
| ESC | European Society of Cardiology, European Society of Cardiology |
| ESH | European Society of Hypertension |
| h | Stunde/Stunden |
| HCM | hypertrophe Kardiomyopathie |
| HZV | Herzzeitvolumen |
| IL | Interleukin |
| IOC | Internationales Olympisches Komitee |
| ITA | Italien |
| LSB | Landessportbund |
| LZ-Blutdruckmessung | Langzeit-Blutdruckmessung |
| LZ-EKG | Langzeit-Elektrokardiogramm |
| MET | metabolisches Äquivalent |
| mmHg | millimeter Quecksilbersäule |
| PSI | Periodontal Sign of Inflammation, Periodontal Screening Index |
| QTc Zeit | frequenzkorrigiertes Zeitintervall von Beginn Q-Zacke bis Ende T-Welle im EKG |
| SCD | sudden cardiac death, plötzlicher Herztod |
| TNF-alpha | Tumor Nekrose Faktor alpha |
| TPE | time peak to end (T-Welle) |

Anmerkung:

Zur besseren Lesbarkeit wird in dieser Schrift das generische Maskulinum verwendet.
Die in dieser Arbeit verwendeten Personenbezeichnungen beziehen sich – sofern nicht anders kenntlich gemacht – auf alle Geschlechter.

1. Einleitung und Grundlagen

1.1. Einleitung und historischer Überblick über sportmedizinische Vorsorgeuntersuchungen

Die Ausübung von Hochleistungssport in seinen vielfältigen Ausprägungen setzt unabhängig von der ausgeübten Sportart die Gesundheit und Belastbarkeit des Athleten zur gefahrlosen Ausübung des Sports voraus. In diesem Kontext spielt die sportmedizinische Vorsorge- oder auch Tauglichkeitsuntersuchung eine wichtige Voraussetzung zur Sicherstellung der individuellen Gesundheit und Belastbarkeit in der ausgeübten Sportart. Dabei liegt der Fokus der Vorsorgeuntersuchungen im Hochleistungssport, anders als in der ärztlichen Sporttauglichkeitsuntersuchung im Rahmen der Primär- und Sekundärprophylaxe der Allgemeinbevölkerung, mehr in der Begutachtung der Tauglichkeit hinsichtlich der deutlich extremeren Beanspruchungen des Organismus durch die ausgeübte Sportart bei in der Regel gesunden, überwiegend Jugendlichen und jungen Erwachsenen. In einer Beobachtungsstudie über einen Zeitraum von 20 Jahren konnte Corrado 2006 aufzeigen, dass das Risiko für einen plötzlichen Herztod beim Sport durch sportärztliche Vorsorgeuntersuchungen gesenkt werden konnte [1]. Der deutsche olympische Sportbund (DOSB) und der deutsche Behinderten Sportbund (DBS) empfiehlt, fordert und finanziert aus o.g. Gründen bei seinen Athleten der diversen Bundeskader olympischer und nicht-olympischer sowie paralympischer Sportarten jährliche sportmedizinische Vorsorgeuntersuchungen [2]. Ebenso fördern und empfehlen die verschiedenen Landessportbünde (LSB) vergleichbare sportmedizinische Sporeingangs- und Tauglichkeitsuntersuchungen bei jungen Leistungssportlern. Die Empfehlungen zum Ablauf und Inhalt dieser Vorsorgeuntersuchungen für den Leistungssport werden durch die Expertenkommission des DOSB bereits seit 1970 fortlaufend weiterentwickelt und angepasst. Eine Empfehlung für den allgemeinen Sport findet sich in der S1 Leitlinie „Vorsorgeuntersuchung im Sport“, die von Mitgliedern der deutschen Gesellschaft für Sportmedizin und Prävention (DGSP) erarbeitet wurde [3]. Des Weiteren organisiert der DOSB für Sportmediziner, die in der Betreuung von Spitzenathleten eingebunden sind, seit 1984 eine jährliche Fortbildung für den spitzensportspezifischen Wissenstransfer.

1.1.2. Ziele der sportmedizinischen Vorsorgeuntersuchung

Vorrangiges Ziel der sportmedizinischen Vorsorgeuntersuchung ist die Feststellung der körperlichen Gesundheit bzw. Früherkennung von asymptomatischen Erkrankungen zur Sicherstellung der allgemeinen Gesundheit und sportartspezifischen Belastungsverträglichkeit. Ebenso soll frühzeitig ein drohender sportbedingter Schaden erkannt und verhindert werden. Letztlich ist insbesondere die Verhinderung des plötzlichen Herztodes als ultimative Form der sportbedingten Schädigung ein

wesentlicher Grund für die Durchführung der sportmedizinischen Vorsorgeuntersuchung. Um der Komplexität der an diese Untersuchungsform gestellten Aufgabe gerecht werden zu können, unterteilt sich die sportmedizinische Vorsorgeuntersuchung in der Regel in eine allgemein-internistische Untersuchung mit erweiterter kardiologischer Begutachtung (Ruhe-EKG, Echokardiografie, ggf. LZ-EKG, ggf. LZ-Blutdruckmessung), eine orthopädische Begutachtung zur Untersuchung des Stütz- und Bewegungsapparates sowie eine (möglichst sportartspezifische) Belastungsuntersuchung. Neben der Feststellung der leistungsphysiologischen Parameter dienen die internistische Diagnostik und die Belastungsuntersuchung vorrangig der Erkennung möglicher lebensbedrohlicher Herz- und Gefäßerkrankungen. Des Weiteren dient insbesondere die im Rahmen der Untersuchung durchgeführte Anamnese, klinische Untersuchung und Labordiagnostik von Blut der Erkennung von entzündlichen Erkrankungen bzw. Infektionen, da diese im Rahmen körperlicher Belastungen im Hochleistungssport mit einem erhöhten Risiko kardial entzündlicher Veränderungen wie Peri- oder Myokarditis einhergehen. Somit dient die sportmedizinische Vorsorgeuntersuchung nicht nur der Früherkennung von bestehenden Herz- oder Gefäßerkrankungen, die lang- oder mittelfristig eine Gefährdung in der Sportausübung darstellen, sondern auch der Erkennung von akuten Gefährdungen des Sportlers, z. B. durch bestehende entzündliche Körperprozesse.

1.2. Physiologische Anpassungsmechanismen im Hochleistungssport

Um eine umfassende, sportmedizinisch und fachlich fundierte Begutachtung im Rahmen einer sportmedizinischen Vorsorgeuntersuchung durchführen zu können, bedarf es seitens des begutachtenden Arztes eines vertieften Wissens um die physiologischen Anpassungsreaktionen des menschlichen Körpers auf anhaltende körperliche Belastungen in Form von entweder Ausdauer- oder Krafttraining. Nur mit diesem Wissen ist es dem Sportmediziner möglich, physiologische, normale körperliche Anpassungsvorgänge von bestehenden oder sich parallel zur laufenden Belastung entwickelnden pathologischen Veränderungen differenzieren zu können. Dabei ist selbstverständlich immer die Dauer und Form der körperlichen Belastung in Bezug zum Geschlecht, Alter, Genetik und Trainingshistorie zu betrachten [4]. Körperliche Belastungsformen werden typischerweise in Ausdauer- und Kraftbelastungen unterteilt. Ausdauertraining wird klassischerweise gegen eine relativ geringe Belastung über einen langen Zeitraum durchgeführt, während Krafttraining gegen eine relativ hohe Belastung für eine kurze Dauer durchgeführt wird. Reine Ausdauer- und reines Krafttrainingseinheiten sind jedoch selten. Die meisten Sportarten kombinieren Ausdauer und Kraft. Darüber hinaus haben neuere Studien zeigen können, dass kurzes Training mit hoher Intensität zu Ausdaueranpassungen führen kann und Übungen mit geringer Belastung, die bis zur Erschöpfung

geführt werden, zu Kraftanpassungen führen können, was das Verständnis darüber in Frage stellt, welche Art von Training zu welcher phänotypischen Verschiebung der Muskeln führt [5].

Allgemein lässt sich konstatieren, dass klassisches Ausdauertraining zu einem verbesserten Herzzeitvolumen, zu einer erhöhten maximalen Sauerstoffaufnahme und zur Erhöhung der muskulären Mitochondriendichte führt [6-10]. Klassisches Krafttraining dagegen führt zu einer Zunahme des Muskelquerschnitts, Verbesserung der neuronalen muskulären Ansteuerung und damit einer verbesserten motorischen Leistung und verbesserten maximalen Kraft [11-14].

1.3. Das Sportherz im Hochleistungssport

Die Entwicklung des Sportherzens im Hochleistungssport, welches letztlich eine Adaption an regelmäßiges Training darstellt, ist vor allem abhängig von der Intensität und Dauer der ausgeübten sportlichen Belastung. Insbesondere Ausdauersportbelastungen führen dabei zu einer harmonischen Vergrößerung der Herzhöhlen und Zunahme der Myokardmasse. Die Adaptionen des kardiovaskulären Systems auf sportliche Belastungen sind dabei schon lange bekannt und wurden bereits frühzeitig untersucht [13, 15-17]. Die Erkenntnis über die Entwicklung einer Sportherzanpassung besteht sogar bereits seit über 120 Jahren. Henschen beschrieb bereits 1899 bei Skilangläufern eine Herzvergrößerung, die er auf die körperliche Aktivität der Sportausübung zurückführte [18].

Physiologisch betrachtet, führt die Vergrößerung des Ventrikels zu einem erhöhten Schlagvolumen und somit, bei gleichbleibender maximaler Herzfrequenz unter Belastung, zu einem erhöhten Herzminutenvolumen, welches konsekutiv mit einem erhöhten Sauerstofftransport und -angebot an die arbeitende Muskulatur einhergeht. Die Entwicklung des Sportherzens mit Myokardhypertrophie und damit einhergehender Ventrikeldilatation ist, im Unterschied zu einer Myokardhypertrophie ohne Ventrikeldilatation wie sie beispielsweise bei einer hypertrophen Kardiomyopathie (HCM) zu beobachten ist, als eine physiologische Anpassungsreaktion auf körperliche Belastung zu verstehen. Dabei bleibt das Verhältnis von Myokarddicke zum Durchmesser des (v.a. linken) Ventrikels im Wesentlichen konstant [19] (entspricht einem konstanten „Hypertrophieindex“). Die physiologische Entwicklung einer Sportherzvergrößerung tritt jedoch nicht bei allen Sportlern einheitlich auf, sondern hängt stark von individuellen (v.a. genetischen) Voraussetzungen ab. Dabei finden sich in der Kohorte der hochausdauertrainierten Athleten in circa 15% der Fälle eine Sportherzvergrößerung im oberen, noch als physiologisch angesehenen Grenzbereich der Vergrößerung. Dabei wird ein linksventrikulärer Durchmesser von bis zu 60mm Innendurchmesser bzw. eine Verdickung der linksventrikulären Wandstärken auf 13-15mm als physiologisch oberer Grenzbereich angesehen [20].

1.3.1. Der Einfluss der Trainingsbelastung auf die Entwicklung des Sportherzens

Um eine relevante Sportherzvergrößerung zu erreichen, ist ein mehrjähriges Ausdauertraining mit einem wöchentlichen Trainingsumfang von mindestens 5-10h/Woche notwendig [21], wobei sehr große, individuelle Unterschiede zu verzeichnen sind, die letztlich als Ausdruck individueller Leistungsvoraussetzung verstanden werden können. Dabei wurden in der Literatur die ausgeprägtesten Sportherzen bei Ruderern, Radfahrern und Skilangläufern beschrieben, [20, 22], während eine Sportherzentwicklung bei Kraftsportlern in der Regel nicht oder nur eingeschränkt zu erwarten ist. Wenngleich diesbezüglich in der Literatur nach wie vor keine vollständige Einigkeit besteht, geht man davon aus, dass Kraftsportler - wenn überhaupt - eher eine konzentrische kardiale Hypertrophie ohne konsekutive Vergrößerung der Ventrikeldurchmesser erfahren. Dabei ist eine Vergrößerung des Herzens, relativ in Bezug auf die Körpermasse, in der Regel nicht zu beobachten [23-26].

1.3.2. Einfluss der Blutdruckbelastung auf die Herzvergrößerung

Die Ursache der physiologischen Sportherzadaptationsreaktion mit exzentrischer Hypertrophie, die vornehmlich im Ausdauersport zu beobachten ist, wird auf eine repetitive Volumenbelastung des rechten und linken Teils des Herzens im Rahmen der kardiovaskulären Belastung bei körperlichen Ausdauersportbelastungen zurückgeführt. Im Gegensatz zur Volumenbelastung des Herzens bei Ausdauersportarten, die durch eine Erhöhung des Herzzeitvolumens des zirkulierenden Blutflusses bedingt wird, kommt es während der Ausübung von Kraftsportarten zu einer Erhöhung des peripheren (Kreislauf-) Widerstandes, arteriellen Blutdruckerhöhung und Zunahme der linksventrikulären Nachlast. Dieses führt zu einer vornehmlich konzentrischen Hypertrophie des Herzens, welche vergleichbar mit den Mechanismen einer pathologischen Herzhypertrophie bei chronischer Drucküberlastung des Herzens (z. B. bei arterieller Hypertonie) zu finden ist [27]. Die Entwicklung einer physiologischen oder pathologischen Hypertrophie ist dabei vorrangig abhängig von der Art der Herzbelastung und nicht deren Dauer [28-31]. So kann bereits eine intermittierende Drucküberlastung des Herzens zu einer pathologischen Hypertrophie führen, während intermittierende sportliche Belastungen eine physiologische Hypertrophie bewirken [32]. Dabei unterscheiden sich die Abläufe der Anpassungsvorgänge pathologischer und physiologischer Hypertrophie fundamental. Im Rahmen der physiologischen Hypertrophie kommt es zu einer erhöhten Energieproduktion und -effizienz, Angiogenese proportional zum Wachstum der Ventrikelwand, mitochondriale Qualitätskontrolle sowie Kardiomyozytenproliferation und -regeneration. Diese Wege antagonisieren aktiv pathologische Reaktionen [27]. Im Gegensatz dazu führen die Signalmechanismen im Rahmen der pathologischen Hypertrophie zu pathologisch kardialer Umgestaltung und Dysfunktion. Dabei kommt es zu Zelltod, Fibrose, mitochondrialer Dysfunktion, metabolischer Reprogrammierung, Reaktivierung der fetalen

Genexpression, gestörter Protein- und mitochondrialer Kontrolle, veränderter Sarkomerstruktur und unzureichender Angiogenese [27]. Bluthochdruck wirkt dabei fördernd auf die pathologische Hypertrophie durch neuroendokrine Hormone und die auftretenden mechanischen Kräfte.

1.3.3. EKG-Veränderungen im Hochleistungssport

Eine wichtige Rationale zur Durchführung von Sporttauglichkeitsuntersuchungen ist die Verhinderung von plötzlichen Todesfällen im Sport. Ein medizinisch bedingter, plötzlicher Tod während der Sportausübung ist am häufigsten auf kardiale Ursachen zurückzuführen [33]. Daher liegt es nahe, im Rahmen der ärztlichen Sporttauglichkeitsuntersuchung den Fokus auf die Früherkennung kardialer Veränderungen zu legen, die mit einem erhöhten Sterberisiko unter körperlicher Belastung einhergehen. In den vergangenen Jahrzehnten wurden umfangreiche wissenschaftliche Untersuchungen zur Ätiologie des plötzlichen Herztodes im Sport veröffentlicht [33-37]. Die erste größere Untersuchung zur Prävalenz des plötzlichen Herztodes im Sport geht auf Corrado et. al. zurück, die in einer prospektiven Untersuchung plötzliche Todesfälle bei Sportlern und Nichtsportlern in Italien (Region Veneto) über einen langen Zeitraum von 1979 bis 1996 untersuchten und die Wertigkeit einer EKG Untersuchung zur Risikoabschätzung eines plötzlichen Herztodes im Sport dokumentierten [38]. Somit sind die Ursachen des plötzlichen Herztodes im Sport seit längerem bereits beschrieben und werden bei jüngeren Sportlern (< 35 Jahre) im Wesentlichen auf eine hypertrophe Kardiomyopathie (HCM), arrhythmogene rechtsventrikuläre Kardiomyopathie (ARVC), Koronaranomalien oder Störungen im Reizleitungssystem (Long-QT Syndrom, Brugada Syndrom) zurückgeführt. Neuere Untersuchungen benennen zudem nicht ischämische, ventrikuläre Narben als eine der vorher wenig beschriebenen Ursachen für einen plötzlichen Herztod bei Sportlern [39], während die HCM, die lange Zeit als eine der Hauptursachen für den SCD angesehen wurde, nach neueren Publikationen als eine wahrscheinlich weniger „gefährliche“ Grunderkrankung im Kontext der Sportausübung angesehen wird [40]. Viele dieser Erkrankungen können bei Normalpersonen im Ruhe-EKG als Abweichungen vom Normalbefund detektiert werden. Die sportbedingten Veränderungen des Ruhe-EKGs, die insbesondere bei Hochleistungsathleten und ausdauersporttreibenden Sportlern als Normvariante beobachtet werden, können jedoch eine Herausforderung in der Beurteilung des Ruhe-EKGs für den behandelnden Arzt darstellen, wenn es um die Abgrenzung von pathologischen gegenüber physiologischen, sportbedingten EKG-Veränderungen geht. Die physiologischen Adaptationsmechanismen des Herzens als Reaktion auf langjähriges (Ausdauer-) Training, die vorrangig zu einer harmonischen Vergrößerung der Herzhöhlen bei gleichzeitiger Zunahme der ventrikulären Muskelmasse führt, der deutlich erhöhte parasympathikotone Tonus und Anpassungen der hormonellen Regulation, bedingen dabei die als physiologisch angesehenen EKG Veränderungen.

Um die Abgrenzung von sportbedingten EKG-Veränderungen von pathologischen EKG-Veränderungen, denen potentiell eine lebensbedrohliche, kardiale Erkrankung zugrunde liegt, zu ermöglichen, wurden in den vergangenen Jahrzehnten verschiedene Empfehlungen zur Interpretation von Sportler-EKGs veröffentlicht. Die erste Konsensus Empfehlung einer großen Dachgesellschaft geht auf die Europäische Herzgesellschaft (European Society of Cardiology, ESC) aus dem Jahr 2005 zurück [41]. Diese Empfehlung wurde durch die ESC 2010 zunächst aktualisiert [42], bevor mit den sogenannten „Seattle Kriterien“ im Rahmen eines internationalen Expertenkonsens [43] eine international gültige Empfehlung zur Beurteilung von Sportler-EKGs definiert wurde. Allen Empfehlungen ist gemein, dass eindeutig sportbedingte EKG-Veränderungen von nicht-sportbedingten Veränderungen, die potentiell auf eine zugrundeliegende kardiale Pathologie hinweisen, unterschieden werden. Daneben werden Veränderungen des EKGs beschrieben, die einen „Graubereich“ der Diagnostik beschreiben und sowohl sportbedingt als auch pathologisch begründet sein könnten und einer weiterführenden kardiologischen Abklärung (Echokardiografie, Belastungs-EKG, Langzeit-EKG etc.) bedürfen. Als Herausforderung in der Definition von Empfehlungen zur Interpretation von Sportler-EKGs hat sich über alle Jahre die Notwendigkeit einer hohen Sensitivität der Beurteilung zur Detektion von zugrundeliegenden Pathologien im Kontrast der Vermeidung zu vieler „falsch positiver“ Befunde bei sportbedingt normalen „gesunden“ Veränderungen erwiesen. Dies hat im weiteren Verlauf zu einer Vielzahl von weiteren Empfehlungen zur Anpassung der „Seattle-Kriterien“ geführt, die unter anderem in einer internationalen Expertenkonferenz 2017 definiert wurden [44].

Unabhängig von der Vielzahl der publizierten Empfehlungen und erfolgten Anpassungen verbleibt für den beurteilenden Arzt die Herausforderung in der Differenzierung von EKG Veränderungen bei Sportlern, die im „Graubereich“ der Diagnostik liegen. Dabei soll zum einen die Sicherheit des Athleten in der Ausübung seines Sports gewährleistet und auf der anderen Seite eine unnötige „Überdiagnostik“ bei im Grunde gesunden Sportlern vermieden werden. So konnte in einer Studie bei sporttreibenden Kindern gezeigt werden, dass durch die zuletzt 2017 aktualisierten internationalen Empfehlungen im Vergleich zu den „Seattle-Kriterien“ und ESC-Empfehlungen die Spezifität (International = 98% ESC = 64% Seattle = 95%) deutlich erhöht werden konnte. Dies ging jedoch mit einer geringeren Sensitivität (ESC und Seattle 86% vs. International 57%) einher [45]. Aus diesem Grund sind auch weiterhin wissenschaftliche Untersuchungen notwendig, die insbesondere in spezialisierten Sportlerkollektiven (Kinder und übergewichtige Kinder, extreme Ausdauersportler etc.) die Sensitivität und Spezifität der EKG-Interpretation weiter erhöhen.

1.4. Blutdruckregulation bei körperlicher Belastung

Körperliche Belastung führt vor allem zu einem erhöhten Sauerstoffbedarf der arbeitenden Skelettmuskulatur. Dieser erhöhte Bedarf wird physiologisch durch einen erhöhten Blutfluss durch die Muskulatur gedeckt. Dazu muss zunächst das Herzzeitvolumen (HZV), welches ein Produkt aus Herzfrequenz und Schlagvolumen darstellt, erhöht werden. Diese Erhöhung des HZV bedingt einen erhöhten Blutdurchfluss durch das arterielle Gefäßsystem. Gemäß dem Hagen-Poiseuilleschem Gesetz, wonach der Druck im einem starren Rohr proportional zu Fluss und Widerstand ist, müsste die Erhöhung des HZV bereits zu einer Erhöhung des arteriellen Blutdrucks führen. Allerdings kann die „Dehnbarkeit“ (=Compliance) der arteriellen Gefäße den Druck der arteriellen Pulsdruckwelle aufnehmen und wirkt daher umgekehrt proportional zum arteriellen Widerstand. Die physiologische Reaktion auf das erhöhte HZV unter körperlicher Belastung führt somit zu einer verbesserten lokalen Durchblutung der arbeitenden Skelettmuskulatur durch aktive Vasodilatation der Skelettmuskelarterien mit erhöhtem Sauerstoffbedarf und Druckaufnahme des erhöhten HZV durch Dehnung der nachgiebigen Arterien [46, 47]. Da die Compliance der Gefäße jedoch mit steigender Druckvolumenbelastung abnimmt, führt eine zunehmende Steigerung des HZV zu einem Anstieg des arteriellen (Blut-)drucks, welcher bei gesunden Personen moderat ausgeprägt ist. Eine verminderte vaskuläre Compliance, wie sie bei Patienten mit arteriellem Bluthochdruck zu finden ist, führt somit zu einem stärkeren Anstieg des arteriellen Drucks unter körperlicher Belastung. Als ein „normaler“ (systolischer) Blutdruckanstieg im arteriellen Gefäßsystem wird ein Anstieg von 10 ± 2 mmHg pro metabolischem Äquivalent (MET) angesehen, wobei bei Ausbelastung ein Plateau erreicht wird [48]. Der diastolische Blutdruck nimmt dabei in der Regel leicht ab oder bleibt stabil. Ein Anstieg des diastolischen Blutdrucks unter Belastung ist somit als nicht physiologisch zu betrachten [49]. Welche Maximalwerte des systolischen Blutdrucks unter körperlicher Belastung als obere „Normalwerte“ zu betrachten sind, ist in der Literatur nicht eindeutig bestimmt. In der Regel werden systolische Spitzenwerte von 210 mmHg bei Männern und 190 mmHg bei Frauen angenommen. Der diastolische maximale Blutdruck sollte 110 mmHg (Männer und Frauen) nicht überschreiten.

1.4.1. Belastungsblutdruckverhalten und arterielle Hypertonie

Ab welchen Werten ein Anstieg des systolischen Blutdrucks unter steigender, körperlicher Belastung als hypertensive Belastungsreaktion gewertet werden sollte, ist letztlich noch immer nicht vollständig geklärt [50-52]. Davon unabhängig ist jedoch bekannt, dass eine Erhöhung des systolischen Blutdrucks bei leichter und mittlerer Belastung bereits frühzeitig ein erhöhtes Risiko für kardiovaskuläre Erkrankungen voraussagt [53]. Zur Früherkennung einer bisher nicht bekannten Hypertonie erweist sich zudem die Beurteilung des Belastungsblutdruckverhaltens unabhängiger von externen Störvariablen als die Ruheblutdruckmessung, wobei bereits geringe körperliche Belastungen zur

Beurteilung ausreichend zu sein scheinen [54]. Aus diesem Grund empfehlen die entsprechenden Leitlinien der ESC (European Society of Cardiology) und ESH (European Society of Hypertension) eine weiterführende Blutdruckabklärung im Falle einer nachgewiesenen, ungewöhnlich hohen Blutdruckreaktion bei niedriger oder mittlerer, körperlicher Belastung [55]. Personen mit einer hypertensiven Blutdruckregulation unter Belastung können bereits Veränderungen wie z. B. linksventrikuläre Hypertrophie, Albuminurie oder diastolische Dysfunktion aufweisen, wie man sie sonst bei Patienten mit manifester Hypertonie beobachtet [52]. Da bereits beschrieben wurde, dass eine belastungsinduzierte arterielle Hypertonie häufig der Entwicklung einer manifesten Hypertonie vorausgeht [56], zeigt sich die klinische Relevanz der Notwendigkeit weiterführender Diagnostik und gegebenenfalls frühzeitiger, therapeutischer Intervention der arteriellen Hypertonie zur Verhinderung langfristiger Endorganschädigungen.

1.5. Der plötzliche Herztod im Sport bei (jungen) Hochleistungssportlern

Die Häufigkeit von plötzlichen, kardial bedingten, Todesfällen beim Sport wird gemeinhin mit einer Inzidenz von circa 0,5-2 : 100.000 pro Jahr angegeben [57]. In einem Meta-Review von Lear et al. wird die Inzidenz bei jungen Sportlern übergreifend mit 0,98:100.000/Jahr beschrieben [58]. Allerdings finden sich je nach untersuchter Kohorte und Sportart zum Teil erheblich davon abweichende Angaben zur Inzidenz des plötzlichen Herztodes (sudden cardiac death = SCD) bei Sportlern. So wurde gezeigt, dass die Inzidenz bei US-College Sportlern mit 1,7:100.000/Jahr deutlich höher lag und in einigen ausgewählten Sportarten (Basketball) sogar Werte von 1:9000/Jahr erreichte [34]. Ebenso zeigte sich die Inzidenz bei jungen Fußballern mit 6,8:100.000/Jahr gegenüber dem Durchschnitt über alle Sportarten deutlich erhöht [59]. Über die Ursache für den plötzlichen Herztod bei Sportlern ab einem Alter von 35 Jahren besteht unter den verschiedenen Autoren im wesentlichen Einigung. Hier wird als mit Abstand häufigste Ursache eine koronare Herzerkrankung angegeben, die unter körperlicher Belastung zu einer Myokardischämie und konsekutiv als Trigger für das Auftreten von potentiell letalen Kammerarrhythmien führt. Im Unterschied zu den Sportlern, die älter als 35 Jahre sind, variieren die angegebenen Ursachen für den plötzlichen Herztod bei jüngeren Sportlern (< 35 Jahre) je nach Autor, untersuchter Sportart und vor allem der untersuchten Region. So werden als Ursache des SCD in Spanien, Italien und Frankreich vorrangig Kardiomyopathien angegeben, während in den Vereinigten Staaten ungeklärte Todesfälle ohne Auffälligkeiten in der Autopsie vorherrschen und in Deutschland, der Schweiz und Norwegen vorzeitig auftretende koronare Herzerkrankungen führend als Ursache des SCD angesehen werden [34, 37, 59-64].

Darüber hinaus liegt dem SCD beim Sport, je nach Alter und untersuchter Kohorte, in 8-20 % der Fälle eine Myokarditis zugrunde [61, 65, 66]. In einer aktuellen Untersuchung zum SCD bei jungen Sportlern

(<35 Jahre) von Böhm et al. zeigte sich die Myokarditis mit einem Anteil von 11 % als dritthäufigste Ursache des SCD [67].

Neben der ausgeübten Sportart und der Herkunft des Sportlers hat insbesondere auch das Geschlecht und die Ethnizität einen maßgeblichen Einfluss auf das Risiko für einen SCD im Sport. Insbesondere in Bezug auf den Einfluss des Geschlechts variieren dabei jedoch die Angaben bezüglich des erhöhten Risikos von männlichen Sportlern gegenüber weiblichen Sportlern von 2.3-fach bis 23-fach [58, 61, 62, 68]. Des Weiteren scheinen Kardiomyopathien und Koronaranomalien, die zum SCD beim Sport führen, bei afro-amerikanischen Athleten circa doppelt so häufig aufzutreten wie bei kaukasischen Athleten [35, 69].

1.6. Inflammation und körperliche Aktivität

1.6.1. Myokarditis und Sport

Als Myokarditis bezeichnet man einen entzündlichen Prozess des Myokards bei dem ein histologischer Nachweis von Herzmuskelzelldegeneration und Nekrose zu finden ist. Dabei kommt es zur Infiltration des Myokards mit Entzündungszellen, die eine inflammatorische Kaskade in Gang setzen. Klinisch kann dieser entzündliche Prozess sowohl Einfluss auf das Reizleitungssystem des Myokards als auch auf dessen Funktion und Integrität haben und somit zu (potentiell tödlichen) Herzrhythmusstörungen oder Herzinsuffizienz führen. Als Auslöser einer entzündlichen Myokarderkrankung im Sport sind in aller Regel virale Infektionen mit vorrangig „kardiotropen“ Viren zu betrachten. Dabei sind vor allem Erkältungs-, Grippe- (Influenza) Viren, das Epstein-Barr-Virus oder das Parvovirus B19 sowie Adeno- und Enteroviren als Erreger zu nennen [70]. Aber auch Bakterieninfektionen können unter Umständen zu einer Myokarditis im Sport führen [71]. Weitere Formen der Myokarditis (nicht infektiöse Formen), die vor allem auf toxisch/chemische Noxen wie beispielsweise Alkohol, Medikamente, Drogen und radioaktive Strahlen oder Autoimmunerkrankungen zurückzuführen sind, spielen in der Genese der Myokarditis im Sport in der Regel keine Rolle. Wie bereits beschrieben, stellt die Myokarditis im Sport ein erhebliches Risiko für das Auftreten eines plötzlichen Herztodes unter Belastung dar [61, 65-67].

1.6.2. Die Interaktion von oraler Inflammation und systemischer Inflammation

Orale inflammatorische Erkrankungen wie Gingivitis und Parodontitis sind häufig nicht nur auf die Mundhöhle begrenzt, sondern können darüber hinaus Einfluss auf die systemisch entzündliche Aktivität des Individuums haben. Somit verursachen Parodontalerkrankungen, vornehmlich das Vorliegen einer (schweren Form) der Parodontitis, systemische Inflamationsprozesse, die wiederum u.a. einen Einfluss auf kardiovaskuläre Erkrankungen haben und deren Progression begünstigen

können [72]. Andersherum kann eine suffiziente Therapie inflammatorischer Zustände des Zahnhalteapparates zur einer Verminderung der systemischen Inflammationsparameter wie CRP oder IL-6 führen und kardiovaskuläre Risikofaktoren wie erhöhten diastolischen Blutdruck oder den Plasmaglukosespiegel senken [73, 74]. Des Weiteren konnte gezeigt werden, dass eine aktive Parodontitis bei ansonsten gesunden Menschen mit einer signifikanten Leukozytose (vorrangig durch Erhöhung der Neutrophilenzahl), erhöhter Blutsenkungsgeschwindigkeit und erniedrigter Thrombozytenzahl als Ausdruck einer erhöhten systemischen Inflammation assoziiert; im weiteren kann die Therapie der chronischen Parodontitis zu einer Senkung der Blutleukozytenzahl führen [75]. Als Ursachen der erhöhten systemischen Inflammation wird, neben der parodontalen Entzündungsreaktion, zudem eine von der Mundhöhle ausgehende erhöhte Bakteriämie angesehen, die konsekutiv zu einer Akut-Phase-Reaktion im Blut sowie zu metabolischen und inflammatorischen Veränderungen im Körper führen kann. Diese sind neben den bereits oben genannten Einflüssen auf kardiovaskuläre Erkrankungen und Diabetes mellitus bzw. erhöhte Insulinresistenz ebenso assoziiert mit einem vermehrten Auftreten von neurodegenerativen Veränderungen wie z. B. Alzheimer Demenz, gastrointestinale und kolorektale Krebserkrankungen, Autoimmunerkrankungen, Atemwegserkrankungen und Schwangerschaftspathologien [76, 77].

Am Beispiel des Diabetes mellitus lässt sich zeigen, dass es einen bidirektionalen Zusammenhang zwischen erhöhter systemischer Inflammation und Parodontitis gibt. So begünstigt zum einen das Vorhandensein eines Diabetes mellitus die Progression einer Parodontitis, zum anderen hat eine bestehende Parodontitis einen negativen Einfluss auf den Krankheitsverlauf bei Diabetikern [78]. Vergleichbar mit Erkrankungen, die eine erhöhte systemische Inflammation bewirken, könnte es einen Zusammenhang zwischen vermehrter körperlicher Aktivität und parodontalen Erkrankungen geben. Es ist bekannt, dass körperliche Aktivität einen hemmenden Einfluss auf proinflammatorische Prozesse im Körper hat [79]. Zudem weisen Personen mit moderater körperlicher Aktivität (3-5 Trainingseinheiten pro Woche) weniger orale Entzündungen auf [80]. Jedoch muss beachtet werden, dass intensive und hochintensive körperliche Belastungen eher proinflammatorisch wirksam sind (Erhöhung von proinflammatorischen Zytokinen wie CRP, IL-6, IL-8, IL-10, TNF-alpha) [81, 82] und es zu einer Abnahme von Immunglobulin A im Speichel kommt [83]. Des Weiteren ist der Zusammenhang von erhöhten TNF-alpha und IL-6 Spiegel im Blut und dem erhöhten Auftreten von Parodontitis bekannt [84, 85]. Dementsprechend lässt sich postulieren, dass systemische proinflammatorische Effekte, wie sie auch unter intensiver körperlicher Aktivität auftreten, einen Einfluss auf entzündliche Prozesse in der Mundhöhle haben könnten, während es bisher diesbezüglich noch keinen überzeugenden wissenschaftlich fundierten Nachweis gibt [86].

Wenngleich es hinsichtlich des pathognomonischen Zusammenhangs zwischen systemischer und oraler Inflammation noch Bedarf an weiterführenden wissenschaftlichen Befunden besteht, konnte

auf die oben genannte Hypothese stützend gezeigt werden, dass die Prävalenz von Gingivitis und Parodontitis bei Spitzensportlern signifikant höher als bei Amateur- und Nichtsportlern ist [87-89].

1.6.3. Einfluss von oraler Inflammation auf körperliche Leistungsfähigkeit

Nach wie vor gibt es nur wenig gesichertes Wissen hinsichtlich der Auswirkung von oraler Inflammation auf die körperliche Leistungsfähigkeit von Spitzensportlern. In einer Befragung unter Teilnehmern der olympischen Spiele gaben 7-18 % der Befragten an, dass sie glaubten ihre Mundhygiene habe einen Einfluss auf ihr körperliche Leistungsfähigkeit in den letzten 3 bzw. 12 Monaten gehabt [90, 91]. In einer weiteren Studie glaubten 40% der befragten Fußballer einen negativen Einfluss auf den Sport durch mangelnde Mundgesundheit zu verspüren [92]. Dennoch bleibt dieser Zusammenhang bisher unvollständig geklärt, da die meisten der durchgeführten Studien zur Auswirkung eingeschränkter Mundgesundheit auf die körperliche Leistungsfähigkeit entscheidende, methodische Schwächen (kleine Stichproben, selektioniertes Probandenklientel, fehlende Längsschnittdaten etc.) hatten. Darüber hinaus sind schwere inflammatorische Erkrankungen der Mundhöhle unter Spitzensportlern selten, sodass die Effektstärke des Einflusses gegebenenfalls gering sein könnte. So konnte in einer Studie an Elite-Ruderern, die den Zusammenhang zwischen Kariesprävalenz und aerober Leistungsfähigkeit untersuchte, kein Zusammenhang gefunden werden, wobei die Unterschiede in der Mundgesundheit der Untersuchungsgruppen gering waren [93]. Unabhängig von Untersuchungen bei Leistungssportlern gibt es jedoch bereits Hinweise, dass eine Parodontitis mit einer verminderten körperlichen Fitness [94] und verminderter maximaler Sauerstoffaufnahme einhergeht [95, 96]. Obwohl diese Studien methodisch hohen Anforderungen gerecht wurden, ist die Übertragbarkeit auf Spitzensportler aufgrund der unterschiedlichen Studienpopulationen (meist ältere Probanden, sitzende Lebensweise etc) eingeschränkt. Vergleichbare Untersuchungen zur Auswirkungen der Parodontitis auf die körperliche Leistungsfähigkeit bei Leistungssportlern liegen bisher nicht vor.

2. Wissenschaftliche Veröffentlichungen

2.1. Physiological Effects of Training in Elite German Winter Sport Athletes.

Wie bereits in der Einleitung beschrieben führt ein langjähriges hochleistungssportliches Training vor allem im Ausdauersportbereich zu umfangreichen körperlichen Adaptationen, welche als physiologische Anpassungserscheinungen ohne pathognomonischen Hintergrund angesehen werden. Insbesondere die kardiovaskulären Veränderungen, die durch Ausdauertraining im Hochleistungssport erfolgen, sind weiterhin Gegenstand intensiver wissenschaftlicher Begutachtung. Die Herausforderung wissenschaftlicher Untersuchungen zur kardiovaskulären Adaptation bei Hochleistungssportlern im Grenzbereich der menschlichen Leistungsfähigkeit liegt naturgemäß in der geringen Anzahl höchstleistungsfähiger Athleten auf internationalem Spitzenniveau. Aufgrund der geringen Anzahl der Topathleten und deren begrenzter Verfügbarkeit zur wissenschaftlichen Begutachtung, wird in der internationalen Literatur häufig auf Leistungssportler etwas niedrigerer Leistungsklassen zurückgegriffen. Diese zeigen zwar in der Regel vergleichbare kardiovaskuläre Anpassungsreaktionen auf ihr leistungssportliches Training, jedoch ist die wissenschaftliche Begutachtung der Athleten, welche sich auf internationaler Höchstleistungsfähigkeit in ihrer ausgeübten Sportart befinden notwendig, um die genannten Effekte auch im Grenzbereich der menschlichen Leistungsfähigkeit zu untersuchen. Dabei ist insbesondere der Vergleich von Ausdauersportlern, die zwar ausdauertrainingsbedingt ähnliche physiologische Adaptationen zeigen, aber aufgrund unterschiedlicher Anforderungen in ihrer jeweiligen Wettkampfleistungsstruktur verschiedenartigen Belastungen ausgesetzt sind, besonders interessant, um herausfinden zu können, inwieweit geringgradig unterschiedliche Trainingsbelastungsformen im langfristigen Leistungsaufbau zu unterschiedlichen kardiovaskulären Anpassungen führen. Ein weiterer wichtiger Aspekt der oben genannten Studie liegt darin begründet, dass das Internationale Olympische Komitee (IOC) in seiner Sitzung 2021 beschlossen hat, Skibergsteigen im Rahmen der olympischen Winterspiele 2026 (Mailand und Cortina d'Ampezzo, ITA) als 16. Sportart aufzunehmen [97]. Da erfahrungsgemäß in der sportmedizinischen Literatur der Fokus auf olympische Sportarten gerichtet war und ist, findet sich bisher wenig wissenschaftliche Forschung im sportmedizinischen Bereich mit dem Fokus auf Skibergsteigen. Insofern lag es nahe Athleten einer Sportart mit langer olympischer Tradition (Skilanglauf und Biathlon) und somit auch umfangreicher, sportmedizinische Begutachtungshistorie mit Athleten einer „neuen“ olympischen Sportart zu vergleichen.



Article

Physiological Effects of Training in Elite German Winter Sport Athletes: Sport Specific Remodeling Determined Using Echocardiographic Data and CPET Performance Parameters

Paul Zimmermann^{1,2,3}, Isabelle Schöffl^{2,4}, Volker Schöffl^{2,5,6,7,8} , Lukas Zimmermann², Max L. Eckstein³, Othmar Moser^{3,9} and Jan Wüstenfeld^{10,11,*}

- ¹ Department of Cardiology, Klinikum Bamberg, 96049 Bamberg, Germany; paul.zimmermann@arcormail.de
- ² Interdisciplinary Center of Sportsmedicine Bamberg, Klinikum Bamberg, 96049 Bamberg, Germany; isabelle.schoeffl@me.com (I.S.); volker.schoeffl@me.com (V.S.); luki.z@web.de (L.Z.)
- ³ Division of Exercise Physiology and Metabolism, Department of Sport Science, University of Bayreuth, 95447 Bayreuth, Germany; max.eckstein@uni-bayreuth.de (M.L.E.); othmar.moser@uni-bayreuth.de (O.M.)
- ⁴ Department of Pediatric Cardiology, University Hospital Erlangen-Nuremberg, 91054 Erlangen, Germany
- ⁵ Department of Traumatology and Orthopaedics, Klinikum Bamberg, 96049 Bamberg, Germany
- ⁶ Department of Trauma Surgery, Friedrich Alexander Universität Erlangen-Nürnberg, 91054 Erlangen, Germany
- ⁷ Section of Wilderness Medicine, Department of Emergency Medicine, University of Colorado School of Medicine, Aurora, CO 80045, USA
- ⁸ School of Applied and Medical Sciences, Leeds Beckett University, Leeds LS1 3HE, UK
- ⁹ Interdisciplinary Metabolic Medicine Research Group, Division of Endocrinology and Diabetology, Medical University of Graz, 8036 Graz, Austria
- ¹⁰ Department Sports Medicine, Charité–Universitätsmedizin Berlin, Corporate Member of Freie Universität Berlin and Humboldt Universität zu Berlin, 14195 Berlin, Germany
- ¹¹ Insitute for Applied Training Science, 04109 Leipzig, Germany
- * Correspondence: jan.wuestenfeld@charite.de



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Abstract: Nine ski mountaineering (Ski-Mo), ten Nordic-cross country (NCC), and twelve world elite biathlon (Bia) athletes were evaluated for cardiopulmonary exercise test (CPET) performance and pronounced echocardiographic physiological cardiac remodeling as a primary aim of our descriptive preliminary report. In this context, a multicenter retrospective analysis of two-dimensional echocardiographic data including speckle tracking of the left ventricle (LV-GLS) and CPET performance analysis was performed in 31 elite world winter sports athletes, which were obtained during the annual sports medicine examination between 2020 and 2021. The matched data of the elite winter sports athletes (14 women, 17 male athletes, age: 18–32 years) were compared for different CPET and echocardiographic parameters, anthropometric data, and sport-specific training schedules. Significant differences could be revealed for left atrial (LA) remodeling by LA volume index (LAVI, $p = 0.0052$), LV-GLS ($p = 0.0003$), and LV mass index (LV Mass index, $p = 0.0078$) between the participating disciplines. All participating athletes showed excellent performance data in the CPET analyses, whereby significant differences were revealed for highest maximum respiratory minute volume (VE_{maximum}) and the maximum oxygen pulse level across the participating athletes. This study on sport specific physiological demands in elite winter sport athletes provides new evidence that significant differences in CPET and cardiac remodeling of the left heart can be identified based on the individual athlete's training schedule, frequency, and physique.

Keywords: physiological demands; winter sport athlete; ski-mountaineering; biathletes; Nordic-cross country; echocardiography; cardiopulmonary exercise testing

1. Introduction

The three described participating winter sport disciplines, ski-mountaineering (Ski-Mo), Nordic-cross country (NCC), and biathlon (Bia), are known to be very challenging

for the individual athlete because they are performed in altitude environmental conditions and involve the whole body and uphill locomotion [1–6].

The individual sport performance of these athletes, especially the physiological demands and functional and structural cardiac remodeling have been reported in previous studies focusing on energy cost, training methods, training frequency, and pronounced structural and functional remodeling of the left heart by transthoracic echocardiographic assessment as well as cardiopulmonary exercise testing (CPET) performance data analyses [4–12].

Nevertheless, the outdoor sport community in general as well as the leisure winter sport community has been rapidly growing in the last decades and enjoys growing popularity. However, despite its popularity, sport-specific research about these professional athletes, particularly Ski-Mo, is up to now scarce [7,11,12]. As a consequence of the increasing professionalization of Ski-Mo from an unorganized leisure sport to a professional sport discipline, it will be represented as a winter sport discipline for the first time in the Milan-Cortina Olympic winter games in 2026 as stated by the International Olympic Committee with two disciplines (speed and individual) [7,13].

In the last decades, the term “athlete’s heart” has been defined by its individual structural, physiological, and functional sport-specific as well as electrophysiological remodeling based on cardiac imaging, CPET performance analysis, and clinical cardiologic evaluation [12,14–20]. In this context, the sport-specific athlete’s background and training impact have to be taken into consideration for varying cardio-physiological adaption [12,14,16,20,21].

In our descriptive preliminary report on the physiological demands based on CPET performance data analyses as well as echocardiographic data assessment in German elite winter sport professionals, we elucidate for the first time an individual athlete’s sport-specific physiological adaption to the performed winter sport discipline. We are able to present significant physiological performance differences between the three different participating cohorts of athletes and to interpret the results against the individual athlete’s physique, training schedule, and training frequency. The matched data of the three participating cohorts represent the ideal comparison group for sport-specific cardiac remodeling, as their training impact and frequency as well as their sport physiological demands are comparable [12,22]. Our presented study on performance data analyses in elite winter sport athletes might contribute to individualizing athletes’ training schedules and their competition performance in the future and might pave the road to future scientific effort to strengthen the scientific basis of evidence.

2. Materials and Methods

2.1. Study Design

This was a multicenter retrospective data analysis of sport specific cardiac remodeling assessment by two-dimensional echocardiographic analysis focusing on the left heart including speckle tracking analysis and CPET performance data in 31 elite winter sport athletes, which were obtained in 2020 and 2021 during the annual medical sports medicine check-up [12,22]. The matched data of the elite winter sports athletes (14 women, 17 male athletes, age: 18–32 years) were compared for different echocardiographic and CPET performance parameters, athlete’s physique data, and sport-specific training aspects.

2.2. Participating Athletes

Thirty-one young elite winter sport professionals, all active members of the German National Team, participating in world championships as well as World Cups, were examined during the season of 2020/2021. During the severe worldwide COVID-19 pandemic situation, no participating athlete was infected or had to be excluded due to post-COVID-19 infection syndromes.

All participants (14 women, 17 male athletes, age: 18–32 years) were assessed by an individual sports medicine check-up in their supervising sports medicine performance

center—the Interdisciplinary Center of Sportsmedicine, Klinikum Bamberg or the Institute for Applied Exercise Science, Leipzig. All participating athletes showed comparable anthropometric data, whereby male athletes weighed 65–81 kg, had a body mass index (BMI) of 18–25 kg/m², and had a height of 175–186 cm. The participating female athletes weighed 47–69 kg, showed a BMI of 18–23 kg/m², and had a height of 154–176 cm, as reported before by Zimmermann et al. [12,22]. All participants were professional winter sport athletes with a total amount of 20–25 training hours per week during the season—based on the individual high volume training schedule—and performed up to 10 trainings hours in the season’s recreational time, including functional strength training (ST) units, continuous endurance training (ET) such as running and cycling, and individual training units to improve muscle disbalances, as pictured in Table 1. No participating athlete had to be excluded from our study due to adverse cardiac events or arrhythmias in any individual athlete’s history. Due to the anthropometric variability with Ski-Mo athletes representing the youngest and physically smallest athletes, and variable fewer lifetime training hours and training schedules, across the three participating winter sport athletes an age-matched analysis is not possible.

Table 1. Baseline training schedule in winter sport professionals.

| Athlete | Average Years of Training | Pre-Season | | | In-Season | | |
|---------|---------------------------|----------------------------|----------|-------------------------------|-------------------------------|----------|-------------------------------|
| | | 10 Training Hours per Week | | | 20–25 Training Hours per Week | | |
| | | Endurance | Strength | Movement Specific/Flexibility | Endurance | Strength | Movement Specific/Flexibility |
| Ski-Mo | 5 ± 3 | 90% | 5% | 5% | 90% | 7% | 3% |
| NCC | 15 ± 5.3 | 84% | 10% | 6% | 89% | 8% | 3% |
| Bia | 14 ± 4.5 | 76% | 11% | 13% | 87% | 9% | 4% |

Abbreviations: Ski-Mo, Ski-mountaineering; NCC, Nordic Cross-Country; Bia, Biathletes.

2.3. Echocardiographic Assessment and CPET Performance Analysis

During the annual sports medicine evaluation in the accompanying performance center, we performed twelve-lead electrocardiograms (ECGs), two-dimensional echocardiographic examination including left ventricle-global longitudinal strain (LV-GLS), and CPET performance analysis in all participating elite winter sport athletes.

The twelve-lead ECG was performed in a lying position with 50 mm/s (CardioSoft V6.73, GE Medical Systems, Munich, Germany and Custo med cardio, Custo med GmbH, Ottobrunn, Germany) to define resting heart rate in beats per minute (bpm) after 5 min. Anthropometric data were additionally evaluated for all athletes, such as body mass index (BMI in kg/m²), body surface area (BSA in m²), and resting blood pressure level (in mmHg) after resting for five minutes in a supine position.

As part of the sports medicine checkup, the echocardiographic functional and morphological assessment was performed using a commercially available echocardiographic system Phillips EPIQ 7 device with an X5-1 Matrix-array transducer (Phillips Healthcare, Eindhoven, The Netherlands), following a standard protocol as described before [12,23]. The acquired images were analyzed and stored digitally; for measurements, sequences of at least three heart beats were stored and analyzed [12]. Our participants were evaluated by two-dimensional echocardiographic analyses performed according to the general recommendations [12,23–25]. The systolic LV ejection fraction (LV-EF) was evaluated using biplane Simpson rule, based on the apical two- as well as the apical four-chamber view and using Teichholz method ejection fraction calculation in the parasternal long axis. For both atria and both ventricles, two-dimensional linear dimensions assessment was performed according to the recommendations [23–25]. A morphological and functional analysis of the right heart, including an estimation of the right ventricular (RV) systolic function using the TAPSE (tricuspid annular plane systolic excursion) was performed in the apical four-chamber view.

Furthermore, based on the two-dimensional echocardiographic measurements of the left heart, specific calculations for each participating athlete were performed for the following indexes by an individual specific validated method: the left ventricular mass index (LV Mass index) by a validated method [26], the relative wall thickness (RWT) of the left ventricle (LV) based on the formula $(2 \times \text{posterior wall thickness}) / \text{left ventricle enddiastolic diameter (LVedd)}$ [26], and the left atrial volume index (LAVI) by a validated method [25].

For functional assessment of the LV, the LV diastolic function was assessed by the pulse-wave Doppler in the apical four-chamber view referring to the peak early filling (E wave) and late diastolic filling (A wave) velocities. To quantify the peak early velocity E' , a tissue Doppler imaging of the lateral mitral annulus in the apical four chamber view was performed [12,23,25].

To reveal the individual athlete's LV-GLS pattern by two-dimensional strain in the apical views, we performed LV speckle tracking analysis and focused on the LV and did not evaluate the RV and LA strain pattern. Therefore, the Philips QLAB cardiac analysis application "AutoStrain" was used (Phillips Healthcare, Eindhoven, The Netherlands). Each athlete was evaluated for the prevalence of left and right heart valve regurgitation during each individual athlete's standard echocardiographic assessment.

The CPET performance analyses were conducted in accordance with the recommendations of the American Heart Association (AHA) [27], and as predetermined by the national winter sport discipline association, either on a bicycle or on a treadmill. Therefore, the CPET step-wise protocol started with a workload of 80 Watts increasing the workload per 40 Watts every 3 min or alternatively starting with 100 Watts increasing per 30 Watts every 3 min until volitional exhaustion. Alternatively, the treadmill tests started with a workload of 10 km/h for male and 8 km/h for female athletes for 3 min and then increasing the speed by 1.0 km/h or 1.5 km/h every 3 min, as described in our previous reporting on CPET [22]. All performance data from the CPET, twelve-lead ECG, and blood pressure were recorded continuously. To define individual athletes' peak performance criteria for CPET analysis, attention was paid to several performance parameters. Primarily, each participant was observed to define individual peri- and post-exercise lactate level with a capillary blood analysis from the earlobe at each workload step, individual anaerobic threshold (4 mmol/L), and their recovery time (maximum 15 mmol/L) [22]. Secondly, several additional peak performance parameters were recognized: a respiratory exchange rate (RER) of 1.15 at peak performance, reaching 85% of the individual maximum predicted heart rate ($220 \text{ bpm} - \text{age in years}$), leveling off of the $\text{VO}_2_{\text{maximum}}$, and individual assumed exercise time of CPET duration. Each athlete was evaluated for the specific athlete's exertion level by the Borg RPE scale (Values ≥ 17). In summary, to define an individual athlete's maximal CPET effort, a minimum of three of the above-mentioned criteria were taken into consideration [22]. Next to the recorded CPET performance data, the oxygen pulse at VT2 (Oxygen pulse VT2) as well as the peak oxygen pulse (Oxygen pulse_{maximum}) were assessed by dividing the derived VO_2 by the heart rate at VT2 or the maximum heart rate [28].

To summarize the obtained data from the echocardiographic and CPET evaluation, additional information for each participating athlete about the training schedules and frequency was collected from the responsible national winter sport discipline association. In particular, detailed information about the individual athletes' training component and frequency was provided to understand the distinctions between our three participating cohorts of world elite winter sports athletes.

2.4. Statistical Analysis

Our data were analyzed with Graph Pad Prism 8.2.1(279) (Graph Pad Software; San Diego, CA, USA). Firstly, all acquired data were assessed for normal distribution by analyzing the data by means of Kolmogorov–Smirnov normality testing. Afterwards, using non-parametric Kruskal–Wallis, we tested our numerical data group comparisons

for nine Ski-Mo athletes (5 male, 4 women), ten NCC athletes (6 male, 4 women), and twelve elite Bia athletes (6 male, 6 women). Afterwards, a gender-specific analysis for the interesting echocardiographic and CPET parameters was utilized equally by using non-parametric Kruskal–Wallis testing. $p \leq 0.05$ was accepted as statistically significant. Since the manuscript is focusing on a clinical scientific outcome in a small limited number of world elite winter sport professionals, the sample size is not calculated in our preliminary reporting.

2.5. Ethical Consideration

The study protocol (17_21 B) was approved by the local ethics committee of the University of Nurnberg-Erlangen. In general, the study was conducted in conformity with the declaration of Helsinki and Good Clinical Practice [29]. Prior to any trial-related activities and measurements, our participating athletes gave their written informed consent and were informed about the study protocol and the following measurements.

3. Results

3.1. Baseline Characteristics and Anthropometric Data

In our descriptive preliminary report, a total of 31 young professional winter sports athletes, including Ski-Mo, NCC, and Bia athletes, were examined. The matched data of the three different participating cohorts were compared for different anthropometric data, for morphological and functional sport-specific cardiac remodeling by echocardiographic assessment and CPET performance parameters. For the echocardiographic assessment, we examined one more male Ski-Mo athlete, who felt too unwell to perform CPET and was therefore solely analyzed for sport specific echocardiographic remodeling, as presented below. The recorded athletes' heart rate at baseline (bpm), individual heart rate response at VT2 as well as at maximum effort during CPET, the resting blood pressure level (mmHg), height, weight, BMI, and BSA are represented in Table 2, adapted to Zimmermann et al. [12,22].

Table 2. Baseline winter sport professional characteristics.

| | Ski-Mo <i>n</i> = 9 | | NCC <i>n</i> = 10 | | Biathletes <i>n</i> = 12 | |
|---------------------------------------------|----------------------|------------------------|----------------------|------------------------|--------------------------|------------------------|
| | Male <i>n</i> = 5 | Female <i>n</i> = 4 | Male <i>n</i> = 6 | Female <i>n</i> = 4 | Male <i>n</i> = 6 | Female <i>n</i> = 6 |
| Age (y) | 21.4 ± 1.8 | 20.8 ± 2.4 | 26.3 ± 4.1 | 25.5 ± 0.5 | 27.3 ± 3.6 | 29.0 ± 3.2 |
| Height (cm) | 178.0 ± 3.9 | 163.5 ± 8.8 | 181.3 ± 4.7 | 171.2 ± 5.8 | 180.9 ± 5.1 | 172.8 ± 3.7 |
| Weight (kg) | 66.5 ± 0.8 | 53.2 ± 6.5 | 72.0 ± 3.0 | 63.4 ± 5.9 | 77.1 ± 3.7 | 62.5 ± 4.1 |
| Resting blood pressure | 118 ± 5.4 | 100 ± 8.2 | 125 ± 8.3 | 105 ± 7.2 | 117 ± 7.6 | 108 ± 6.2 |
| systolic/diastolic (mmHg) | 78 ± 4.0 | 72 ± 1.5 | 78 ± 2.9 | 71 ± 3.8 | 77 ± 2.2 | 70 ± 3.3 |
| Resting heart rate (bpm) | 41 ± 4.6 | 44 ± 4.5 | 42 ± 3.6 | 46 ± 5.1 | 41 ± 4.2 | 45 ± 5.1 |
| Heart rate VT2 (bpm) | 133 ± 22 | 132.3 ± 1.9 | 136.3 ± 11.6 | 128.3 ± 9.1 | 148.5 ± 20.9 | 134.5 ± 8.1 |
| Maximum heart rate (bpm) | 185.6 ± 6.3 | 171.8 ± 2.5 | 183 ± 14.3 | 173.8 ± 4.0 | 179.5 ± 10.3 | 181.0 ± 12.9 |
| BMI (body mass index in kg/m ²) | 19.9 ± 1.4 | 19.8 ± 0.4 | 22.0 ± 1.1 | 22.0 ± 1.1 | 23.6 ± 0.9 | 20.9 ± 1.0 |
| BSA (body surface area in m ²) | 1.70 ± 0.06 | 1.61 ± 0.12 | 1.88 ± 0.04 | 1.81 ± 0.07 | 1.92 ± 0.04 | 1.77 ± 0.05 |

Data are presented as mean with standard deviation Abbreviations: y, years; cm, centimeter; kg, kilogram; bpm, beats per minute; m², square meter

3.2. Morphological and Functional Cardiac Remodeling

Evaluating the morphological and functional cardiac remodeling of our participating elite winter sport athletes, we could reveal significant differences between the three different winter sport disciplines (results shown in Table 3).

Table 3. Echocardiographic Measurements in elite winter sport athletes, adapted from Zimmermann et al., 2021 [12].

| | Ski-Mo (I) <i>n</i> = 10 | | NCC (II) <i>n</i> = 10 | | Biathletes (III) <i>n</i> = 12 | | <i>p</i> -Value |
|-----------------------------|--------------------------|---------------|------------------------|---------------|--------------------------------|--------------|-----------------------------|
| | Male Female | | Male Female | | Male Female | | |
| | <i>n</i> = 6 | <i>n</i> = 4 | <i>n</i> = 6 | <i>n</i> = 4 | <i>n</i> = 6 | <i>n</i> = 6 | |
| LV edd (mm) | 50.83 ± 4.22 | 45.25 ± 5.96 | 55.50 ± 3.83 | 50.75 ± 3.50 | 55.50 ± 5.24 | 49.50 ± 1.52 | ns |
| | 48.6 ± 5.48 | | 53.6 ± 4.27 | | 52.5 ± 4.83 | | |
| LV Mass Index (g/m) | 97.2 ± 25.2 | 76.3 ± 26.7 | 130.7 ± 16.5 | 106 ± 16.4 | 133.5 ± 20.6 | 102.3 ± 14.8 | 0.0078 * |
| | −88.8 ± 26.6 * | | 120.8 ± 20.1 * | | 117.9 ± 23.6 * | | |
| Relative wall Thickness RWT | 0.38 ± 0.03 | 0.34 ± 0.06 | 0.40 ± 0.04 | 0.41 ± 0.04 | 0.40 ± 0.04 | 0.42 ± 0.04 | Ski-Mo vs. NCC 0.0230 * |
| | 0.37 ± 0.05 | | 0.41 ± 0.03 | | 0.41 ± 0.04 | | Ski-Mo vs. Bia 0.0230 * |
| IVSd (mm) | 8.67 ± 1.97 | 8.25 ± 2.50 | 11.00 ± 0.63 | 10.50 ± 0.58 | 10.83 ± 0.98 | 9.67 ± 1.37 | Ski-Mo vs. NC C 0.0266 * |
| | 8.5 ± 2.07 | | 10.4 ± 1.17 | | 10.3 ± 1.29 | | Ski-Mo vs. Bia 0.0337 * |
| LVPWs (mm) | 3.97 ± 11.03 | 7.75 ± 1.50 | 11.17 ± 0.41 | 10.50 ± 0.58 | 12.33 ± 2.07 | 10.17 ± 1.17 | Ski-Mo vs. NC C 0.0161 * |
| | 8.9 ± 1.52 | | 10.9 ± 0.57 | | 11.3 ± 1.96 | | Ski-Mo vs. Bia 0.0030 * |
| E/A | 2.18 ± 0.58 | 1.98 ± 0.17 | 2.48 ± 0.26 | 2.40 ± 0.77 | 1.97 ± 0.52 | 1.75 ± 0.40 | NCC vs. Bia 0.0166 * |
| | 2.1 ± 0.45 | | 2.5 ± 0.49 | | 1.9 ± 0.46 | | |
| E/E' | 6.75 ± 1.71 | 7 ± 1.79 | 6.80 ± 0.86 | 6.13 ± 1.22 | 7 ± 0.86 | 6.37 ± 1.04 | ns |
| | 6.9 ± 1.66 | | 6.4 ± 1.09 | | 6.7 ± 0.97 | | |
| LAVI (mL/m ²) | 51.83 ± 12.1 | 46.25 ± 11.1 | 150 ± 84.58 | 89.3 ± 45.7 | 117.5 ± 37.7 | 72.8 ± 19.6 | 0.0052 * |
| | 49.6 ± 11.4 | | 125.7 ± 75.2 | | 95.2 ± 36.9 | | |
| RA (cm ²) | 19.17 ± 3.87 | 16.75 ± 2.87 | 24.83 ± 3.73 | 18.28 ± 4.72 | 20.78 ± 3.64 | 15.50 ± 2.40 | ns |
| | 18.2 ± 3.55 | | 22.2 ± 5.16 | | 18.1 ± 4.03 | | |
| GLS | −18.26 ± 2.21 | −18.83 ± 2.93 | 21.21 ± 1.99 | −23.25 ± 3.23 | 22.62 ± 1.26 | 22.34 ± 1.42 | 0.0003 * |
| | −18.5 ± 2.38 | | −22.0 ± 2.61 | | −22.5 ± 1.29 | | |

Data are presented as mean with standard deviation. *p* value *, statistically significant (*p* < 0.05). Abbreviations: LV edd, left ventricle enddiastolic size; LV, left ventricular; IVSd, interventricular septal wall thickness at diastole; LVPWd, left ventricular posterior wall thickness at diastole; E/A and E/E', parameters for diastolic function of the left ventricle; LAVI, left atrial volume index; RA, right atrium; GLS, global longitudinal strain; ns, non-significant.

In the two-dimensional echocardiographic examination, all participating elite winter sport athletes showed a normal to little reduced systolic LV ejection fraction (LV-EF) estimated by the biplane Simpson and Teichholz rule. For all three different winter sport professionals, we calculated the LV Mass Index as an indexed parameter and obtained significantly higher values for this parameter for NCC and Bia athletes compared to Ski-Mo athletes (results shown in Table 3). Additionally, significant higher relative wall thickness (RWT) could be shown in NCC and Bia athletes in comparison to Ski-Mo professionals (results shown in Table 3).

Analyzing the morphological structure of the left atrial remodeling, significant differences could be revealed across our three participating athletes. The LAVI (mL/m²), as an indexed parameter, was significantly enlarged in NCC and Bia athletes compared to Ski-Mo professionals (Figure 1). This was also true for the sex-related sub-analyses. In detail, male and female Ski-Mo athletes showed the smallest values (results shown in Table 3).

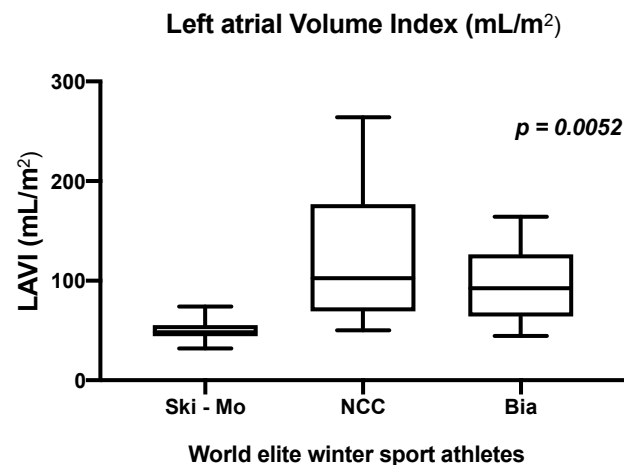


Figure 1. Analysis of the left atrial volume index (LAVI)—significant different results defined by the athletic sporting discipline in world elite winter sport professionals ($p = 0.0052$), modified from Zimmermann et al. 2021 [12].

Focusing the left ventricular remodeling, especially the eccentric remodeling of the LV, such as interventricular septal wall diameter (IVSd), the left ventricular posterior wall diameter (LVPWd), and the RWT, we could elucidate significant differences across our participating athletes. The NCC and Bia athletes in general as well as in the gender-specific analyses showed significantly thicker LV wall diameter than Ski-Mo athletes. No significant structural anatomic differences between our athletes could be proven for LVedd, for the end-diastolic volume (LV EDVed), the right heart dimensions as right atrial end-systolic diameter (RA endsyst), and right ventricular end-diastolic size (RV edd) as well as the TAPSE of RV.

Focusing on the functional cardiac remodeling, i.e., diastolic function and speckle tracking analysis in our elite winter sport athletes, we were able to prove significant differences for the E/A ratio as criteria for LV diastolic function, but no significant differences in the gender sub-analysis (results shown in Table 3). The speckle tracking analysis with the main emphasis on the LV-GLS in our athletes revealed significant differences with regards to the background of the sport specific discipline in our small cohort. The LV-GLS in male athletes with Ski-Mo athletes had the lowest values ($p = 0.0003$, Figure 2 and results shown in Table 3).

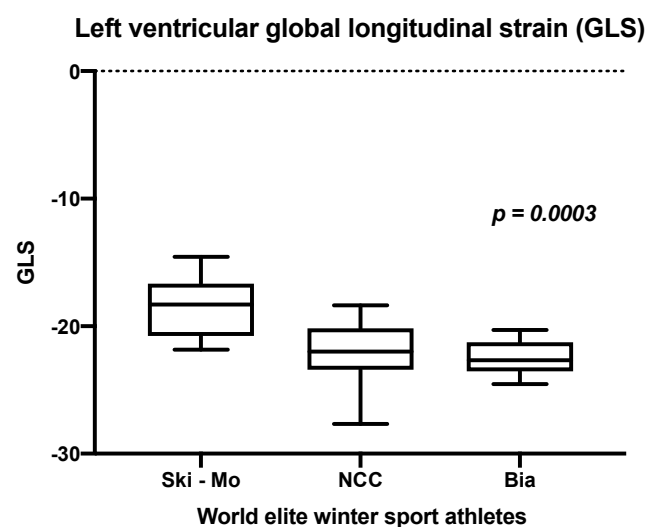


Figure 2. Analysis of the left ventricular global longitudinal strain (GLS) in world elite winter sport professionals ($p = 0.0052$), modified from Zimmermann et al., 2021 [12].

3.3. Sport-Specific Physiological Performance by Laboratory CPET Analyses

Next to the presented anthropometric data and echocardiographic assessment, our professional winter sport athletes were compared for CPET performance parameters, whereby all participating athletes showed excellent performance data as presented in Table 4 (adapted from Zimmermann et al., 2022 [22]). Updated Olympic-medal-level performance benchmark data were used as reference [30].

Table 4. Cardiopulmonary Exercise Testing (CPET) performance parameters in elite Winter Sport professionals, adapted from Zimmermann et al., 2022 [22].

| | Ski-Mo (I) | | NCC (II) | | Biathletes (III) | | p-Value Male | p-Value Female | Overall p-Value |
|------------------------------------|-----------------------|----------------|-----------------------|----------------|-----------------------|----------------|-----------------------------------|-----------------------------------|-----------------------------------|
| | Male | Female | Male | Female | Male | Female | | | |
| VE _{maximum} (L) | 134.9 ± 24.6 | 109.2 ± 20.6 | 166.2 ± 28.4 | 118.2 ± 23.8 | 175.8 ± 11.7 | 125.4 ± 9.1 | Ski-Mo vs. Bia 0.0087 * | ns | Ski-Mo vs. Bia 0.0409 * |
| | 123.5 ± 25.4 | | 147.0 ± 3 5.4 | | 150.6 ± 28.1 | | | | |
| VO _{2 maximum} (mL) | 3964.8 ± 1367.8 | 3021.3 ± 515.1 | 4620.8 ± 603.8 | 3315.3 ± 576.0 | 4935.2 ± 525.1 | 3555.7 ± 274.7 | Ski-Mo vs. Bia 0.0087 * | Ski-Mo vs. Bia 0.0381 * | ns |
| | 3545.4 ± 643.7 | | 4098.6 ± 876.2 | | 4245.4 ± 823.8 | | | | |
| VO _{2/kg maximum} (mL/kg) | 65.0 ± 7.9 | 57.4 ± 4.5 | 64.5 ± 7.1 | 52.7 ± 4.9 | 64.6 ± 4.4 | 57.4 ± 2.3 | ns | ns | ns |
| | 61.6 ± 7.5 | | 59.7 ± 8.6 | | 61.0 ± 5.0 | | | | |
| Oxygen pulse maximum (mL/min) | 20.8 ± 30 | 15.6 ± 31 | 26.9 ± 4.2 | 19.4 ± 31 | 27.8 ± 3.2 | 22.8 ± 5.4 | Ski-Mo vs. NCC 0.0303 * | Ski-Mo vs. Bia 0.0190 * | Ski-Mo vs. NCC 0.0231 * |
| | 18.5 ± 4.0 | | 23.9 ± 5.3 | | 25.3 ± 4.9 | | | | |

Data are presented as mean with standard deviation. *p* value *, statistically significant (*p* < 0.05). VO/kg, ventilatory oxygen uptake per kilogram; L liter; mL, milli-liter; min, minute; ns, not significant. Abbreviations: CPET, cardiopulmonary exercise testing; Ski-Mo, Ski-mountaineering; NCC, Nordic Cross-Country; VE, respiratory minute volume; VO, ventilatory oxygen uptake.

Analyzing the sport specific aerobic capacity in our participating athletes, the highest maximum respiratory minute volume (VE_{maximum}) was elucidated for Bia athletes, who showed significantly higher values in comparison to Ski-Mo athletes (results presented in Table 4).

No significant differences could be revealed for the maximum ventilatory oxygen uptake (VO_{2 maximum}) nor for the indexed ventilatory oxygen uptake (VO₂) at the maximum performance level (VO_{2/kg maximum}) across the three participating winter sport professionals.

Analyzing the Oxygen pulse_{maximum} as presented in Table 4, NCC and Bia athletes showed significantly higher performance values than our participating Ski-Mo athletes (Figure 3).

Focusing on the gender-specific CPET performance data analyses, the female Bia athletes were able to assume the highest peak oxygen pulse (Oxygen pulse_{maximum}) performance parameters across the three winter sport groups (*p* = 0.0190).

Additionally, in our male winter sport professionals, the highest maximum respiratory minute volume (VE_{maximum}, *p* = 0.0087), the highest maximum ventilatory oxygen uptake (VO_{2 maximum}, *p* = 0.0087, Figure 4), and the best peak oxygen pulse (Oxygen pulse_{maximum}, *p* = 0.0260) were highlighted for the male Bia athletes in comparison to the other two participating sport disciplines (results shown in Table 4).

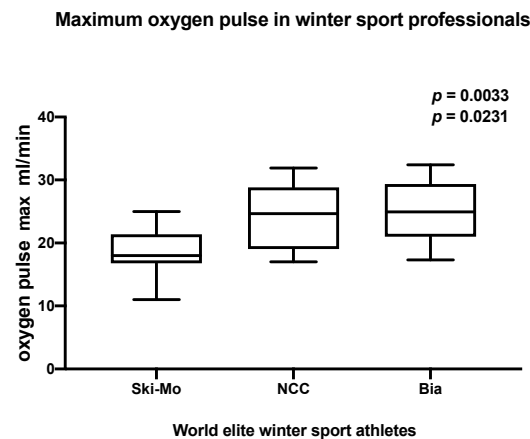


Figure 3. Analysis of maximum oxygen pulse in world elite winter sport professionals ($p = 0.0033$, $p = 0.0231$), modified from Zimmermann et al., 2022 [22].

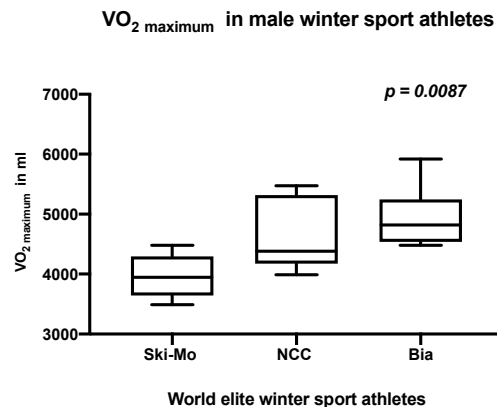


Figure 4. Analysis of maximum ventilatory oxygen uptake (VO_{2 maximum}) in male world elite winter sport professionals ($p = 0.0087$).

4. Discussion

In the present descriptive preliminary report, morphological and functional cardiac remodeling as well as sport specific CPET performance parameters in German world elite winter sport athletes were investigated and compared with each other for the first time. The investigated winter sports in this study are known for their high energy demands, involve the whole body, and are often performed at altitude [3–5,9,10]. Environmental conditions as well as exceptionally high aerobic turnover, an excellent anaerobic power, and different race speed qualities have to be taken into consideration as variable parameters influencing individual athletes' performance [8,15,22,31–34].

In this context, repeated intensity fluctuations and physiological adaptations have been reported before in cross-country skiing [33]. These results emphasize the impact of the high endurance demand in our analyzed winter sports on sport-specific echocardiographic remodeling as well as physiological response assumed by CPET parameters.

The impact of two-dimensional echocardiography on morphological and functional cardiac remodeling of the athlete's heart has been studied and described before. Morphological features, especially left heart remodeling with enlarged heart structures in male athletes in general are the main findings [35].

Classifying our observed descriptive findings in this context, several influencing factors have to be taken into consideration. The training schedule and frequency show inter-group differences. These differences, especially the individual training strategies, focusing on ET or ST components, might slightly and rather likely in long-term perspective contribute to individualized variable sports specific adaptations. Analyzing the baseline morphological and functional echocardiographic remodeling across our three participating

elite winter sport disciplines, we could reveal significant differences in the NCC and Bia sports in comparison to Ski-Mo athletes. The Ski-Mo athletes, representing the youngest and physically smallest athletes, with less lifetime training hours and the highest amount of endurance training (ET) during training schedule [4,12], revealed significantly lower values for LV mass index for Ski-Mo compared to NCC and Bia athletes. In athletes focusing on ST, higher LV wall thickness can be observed [12,36]. In our study, we elucidated LA remodeling especially in NCC and Bia athletes by the analyzed indexed parameter—LAVI. Significantly higher values were observed for the NCC and Bia athletes compared to Ski-Mo athletes with less life time training hours. Nevertheless, in elite endurance athletes, LA remodeling has been reported as a typical characteristic and might contribute to a transient balanced cardiomyopathy with the further risk of developing an atrial cardiomyopathy [37]. In the end, these descriptive echocardiographic findings do not translate to an increased $\text{VO}_2/\text{kg}_{\text{maximum}}$ as the reference parameter for peak performance in our NCC and Bia athletes. Nevertheless, we could elucidate significantly higher oxygen pulse $_{\text{maximum}}$ levels in Bia and NCC athletes, which might be suggestive for enhanced peak performance. This enhanced peak performance—displayed by higher oxygen pulse $_{\text{maximum}}$ levels—might be assumed by varying morphological and functional cardiac remodeling across our three groups. Regarding this parameter, it has to be stated clearly that several parameters and circumstances might influence this parameter next to the analyzed baseline echocardiographic parameters at rest. Interindividual exercise-dependent blood pressure increase, variable hemoglobin levels, mild right-left atrial shunt, mild intrapulmonary shunting with oxygenation mismatch, or variable dynamic stroke volume determined by alternating volume preload conditions, transient balanced sport specific atrial remodeling, or variable autonomous vagal regulation in athletes are previously known influencing factors [38,39]. These mentioned influencing factors have to be taken additionally into consideration judging the athlete's peak performance. Our athletes' performance assessment based on resting echocardiographic assessment and CPET performance analysis has to be interpreted with caution and displays only partly relevant influencing parameters for athletes' race performance. Transient balanced sport-specific atrial remodeling might contribute to an improved exercise capacity and cardiac output during exercise in athletes revealed by positive correlations between $\text{VO}_2_{\text{maximum}}$ and LA passive emptying fraction [40] as well as mild association between peak cardiac performance output and resting left heart cardiac parameters [41].

Analyzing the controversially discussed topic of functional LV remodeling by E/A and E/E' ratio observation, our data in world elite winter sport professionals revealed comparable results in elite athletes with previous research [16,35]. Various factors influencing diastolic function have been reported before, such as low resting heart rate, increased vagal tone, and improved hemodynamic filling of the LV in athletes [16]. Highlighting the impact of LV-GLS analysis in winter sport professionals, we revealed slightly reduced values in Ski-Mo compared to NCC and Bia athletes. By using LV-GLS observation, the distinction between inherited or acquired cardiomyopathies and pronounced physiological cardiac remodeling can be improved. Whereas the normal LV-GLS strain range is estimated to be between -18% and, -25% , strain analysis in general can detect functional abnormalities and early changes in cardiac mechanics long before structural damages can be revealed [12,42–44].

Our findings have to be interpreted carefully and are limited due to several influencing circumstances. On the one hand, the impact of various athletes' anthropometric data, different training schedule, and frequency, and on the other hand the data acquisition in the preseason preparation time as well as an interobserver variability might have contributed to the observed differences [12]. Nevertheless, our descriptive results might emphasize the impact of pronounced specific training-induced cardiac remodeling in athletes. While the clinical atrial cardiomyopathy is difficult to objectify, an increased risk for future degeneration to a pathological entity within the lifetime of a sports career might be assumed [12].

We evaluated the sport-specific physiological performance of professional winter sport athletes with laboratory CPET analyses as predictors of performance. Thus, we were able to elucidate different “adaption patterns” related to the impact of sport discipline and training schedule. First of all, Ski-Mo athletes are known for their enhanced aerobic capacity due to repeated intensity fluctuations, high intensity sprints, uphill locomotion, and high aerobic energy turnover [4,7,13,22,45]. Our data indicate similar findings with a comparable VO_2 maximum but a lower VE maximum and a lower cardiac output, determined by a lower Oxygen pulse maximum. Our findings are supported by previous data, which revealed a positive correlation between increasing age and maximum oxygen uptake and anaerobic threshold in German Nordic combined athletes [46]. An improved development of VO_2 maximum in relation to athlete’s age and training conditions, i.e., training volume and lifetime training hours, was also noted in NCC athletes [47].

Summarizing the obtained results from our descriptive reporting study, we were able to detect valuable parameters for significant sport specific cardiopulmonary adaption in participating winter sport athletes. Although it might be difficult to derive reliable conclusions in this small sample size of world elite winter sport professionals, the reported CPET performance data and sport specific cardiac remodeling in the physically stronger NCC and Bia athletes might contribute to an enhanced peak performance of these two established winter sport disciplines. On the one hand, the participating athletes did not differ significantly with regard to the indexed ventilatory oxygen uptake at the maximum performance level (VO_2/kg maximum), but additionally analyzing the oxygen pulse maximum, NCC and Bia athletes showed significantly higher peak performance values than our participating Ski-Mo athletes. Carefully interpreting these obtained descriptive findings, we might assume the described physiological adaption as well as sport specific echocardiographic remodeling, displayed by higher LV Mass index, larger left atrial remodeling as measured by LAVI, and higher values for the LV-GLS in NCC and Bia athletes. These obtained data and their drawn logical conclusion remain, in the end, speculative, limited by the small sample size of world elite winter sport professionals. Regarding the sport-specific left heart remodeling in interaction with the above-described individual athlete’s physiological adaption, an enhanced cardiac and physiological performance in NCC and Bia athletes—especially at maximum effort—might be assumed. These obtained findings will have to be confirmed in larger study populations and long-term follow-up observation in cooperation with the supervising national team staff and might contribute to individual sport-specific training planning in these elite winter sport professionals in the future.

Our study has several limitations as mentioned above in the discussion. Firstly, the number of participating elite winter sport athletes is relatively small as we enrolled only high-level athletes from the German national teams competing at World Cups and world-class events. Secondly, the echocardiographic assessment and CPET performance data of the participating athletes were assessed in a multicenter study design, implying a certain interobserver variability with respect to data acquirement. Thirdly, our performance measurements were acquired in the preseason preparation time in summer in a multicenter design with respect to a deviation in individual training schedules resulting in inter-group heart volume and training intensity variability. Furthermore, the anthropometric variability due to Ski-Mo athletes representing the youngest and physically smallest athlete category with fewer lifetime training hours might contribute to an interindividual variability evaluating sport specific cardiac remodeling and physiological adaption in these athletes. Additionally, the mixture of young and experienced NCC and Bia athletes entails an intra-cohort variability and contributes to a certain standard deviation in our cardiac and CPET measurements. Last, we focused on speckle tracking of LV-GLS and not on the circumferential LV strain analysis. We performed no specific strain analysis in the RV and LA, which has to be stated as an important limitation of this study. Taking these circumstances into consideration, we agree that our paper should be likely regarded as an interesting descriptive preliminary reporting for sport-specific remodeling in elite German winter sport athletes. Future research might focus on larger athlete sample sizes to

specify the presented descriptive findings with statistical adjustment for anthropometric baseline characteristic, athlete's age, lifetime training hours adjustment, or detailed training schedule adjustment.

5. Conclusions

This descriptive reporting provides new evidence that in different German world elite winter sport professionals, significant differences in morphological and functional remodeling of the left heart as well as for CPET parameters can be demonstrated, against the background of athlete's anthropometric data, athlete's physique, and training components and frequency.

Our results have to be handled with care due to the mentioned limitations and might serve as a preliminary report. Therefore, our results analysis—in general as well as in the gender-specific subgroup analyses—can identify physiological differences in morphological and functional sport specific cardiac remodeling. This was revealed in the speckle tracking analysis, focusing the LV-GLS, LV mass index parameters, and LA remodeling as measured by LAVI. On the other hand, sport-specific individual differences in the CPET performance can be elucidated for our three participating cohorts, especially due to the maximum performance parameters, such as VE_{maximum} and Oxygen pulse_{maximum}.

These obtained differences between the three participating groups might define a pronounced athlete's individual structural and functional sport specific cardio-physiological adaption. Nevertheless, when interpreting an athlete's heart by echocardiographic assessment and individual CPET performance data analysis, the impact of the athlete's physique, training schedule, and frequency have to be taken into consideration. From this aspect, our descriptive reporting might pave the road to future studies with greater number of participating athletes and long-term follow-up to verify the impact on sport-specific athletes' heart adaption and to further strengthen the scientific evidence base.

Author Contributions: Data curation, P.Z., L.Z. and J.W.; formal analysis, P.Z. and L.Z.; investigation, P.Z., J.W. and V.S.; methodology, P.Z. and I.S.; project administration, P.Z. and J.W.; supervision, P.Z., I.S. and J.W.; writing—original draft, P.Z. and J.W.; writing—review and editing, V.S., L.Z., M.L.E., O.M. and I.S. All authors have read and agreed to the published version of the manuscript.

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Institutional Review Board Statement: The study was conducted according to the guidelines of the Declaration of Helsinki, and approved by the Ethics Committee of the University of Nurnberg-Erlangen (study protocol 17_21 B).

Informed Consent Statement: Informed consent was obtained from all subjects involved in the study.

Data Availability Statement: Individual anonymized data supporting the analyses of this study contained in this manuscript will be made available upon reasonable written request from researchers whose proposed use of data for a specific purpose has been approved.

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Conflicts of Interest: The authors declare no conflict of interest.

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2.2. Blood pressure response to dynamic exercise testing in adolescent elite athletes, what is normal?

Nachdem bereits im ersten Abschnitt der Veröffentlichungen (2.1.) der wissenschaftliche Fokus auf Athleten einer Sportart gelegt wurde, in der bisher wenig bis kaum Daten zu kardiovaskulären Anpassungserscheinungen in Bezug auf die Ausübung ihrer hochleistungssportlichen Tätigkeit (Skibergsteigen) veröffentlicht wurden, beschäftigt sich die zweite vorgestellte Arbeit mit einem weiteren Klientel von Athletinnen und Athleten, die wissenschaftlich häufig nur unzureichend hinsichtlich ihrer kardiovaskulären Sportanpassungsreaktionen untersucht werden (Kinder und Jugendliche). Obwohl die Begutachtung und Interpretation von Ruhe- und Belastungsblutdruckverhalten in der Inneren Medizin und Sportmedizin von jeher einen integralen Bestandteil jeder medizinischen Untersuchung darstellt und dies auch im Klientel der Kinder- und Jugendsportmedizin regelhaft erfolgt, ist es umso erstaunlicher festzustellen, dass es bisher nur sehr wenige Daten zum Blutdruckverhalten von Kindern und Jugendlichen bei Belastungsuntersuchungen gibt. Um jedoch pathologische Zustände im Blutdruckverhalten frühzeitig zu erkennen und gegebenenfalls primär – oder sekundärprophylaktisch ärztlich handeln zu können, ist die Kenntnis des Normalzustandes eines Parameters (bzw. dessen Normalverteilung innerhalb eines definierten Kollektivs) unabdingbare Voraussetzung. Zwar existierten bisher vereinzelte Daten und Angaben zu Normalwerten des Blutdruckverhalten von Kindern und Jugendlichen bei Belastungsuntersuchungen, jedoch lagen bisher noch immer unzureichende Daten zum Belastungsblutdruckverhalten junger Leistungssporttreibender vor. Die oben genannte Arbeit bietet Sportmedizinerinnen und Sportmedizinern somit umfassende Anhaltspunkte zur Begutachtung des Blutdruckverhaltens von leistungssporttreibenden Kindern und Jugendlichen im Rahmen von Belastungsuntersuchungen auf dem Ergometer.



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EDITED BY

Isabelle Schöffl,
Friedrich-Alexander-Universität
Erlangen-Nürnberg, Germany

REVIEWED BY

Nicole Müller,
University Hospital Bonn, Germany
Stephan Gerling,
University Medical Center Regensburg,
Germany

*CORRESPONDENCE

Jan C. Wuestenfeld
wuestenfeld@iat.uni-leipzig.de

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Blood pressure response to dynamic exercise testing in adolescent elite athletes, what is normal?

J.C. Wuestenfeld^{1,2*}, F. Baersch³, P. Ruedrich², C. Paech⁴ and B. Wolfarth^{1,2}

¹Department Sports Medicine, Charité – Universitätsmedizin Berlin, Corporate Member of Freie Universität Berlin and Humboldt, Universität zu Berlin, Berlin, Germany, ²Institute for Applied Training Science (IAT), Department of Sports Medicine, Leipzig, Germany, ³Faculty of Sports Science, Institut of Sports Medicine, University Leipzig, Leipzig, Germany, ⁴Department for Pediatric Cardiology, University of Leipzig –Heart Center Leipzig, Leipzig, Germany

Background: In general, only few studies are dedicated to blood pressure behavior under physical stress in children and adolescents. Even less is published about the blood pressure behavior of young high-performance athletes on the ergometer. For this reason, we evaluated the blood pressure behavior under stress compared to non-athletes in a large collective ($n = 739$) of young high-performance athletes (age 10–20 years, mean 15.8 years, male 442, female 297) of different sports. A complete echocardiographic examination was available in all athletes.

Result: Regardless of gender, the young competitive athletes achieved significantly higher maximum blood pressure values than investigated populations from previous studies. Based on the data obtained, blood pressure percentiles are now defined explicitly for junior athletes across sports as well as age- and gender-dependent, which did not exist in this form of normal values for the special clientele of young competitive athletes. The echocardiographic examinations demonstrated stress-induced cardiac adaptation adaptations in the majority of athletes, which thus correlate with the comparatively higher stress blood pressures compared to non-athletes.

Conclusion: For the first time, blood pressure percentiles for exercise tests on the ergometer for age groups and gender in high performance athletes are defined based on a comparatively large collective of young competitive athletes. Upper limits were determined, in particular for systolic blood pressure under stress, and categorized according to gender and age. Performance diagnosticians and physicians are now enabled to make a more accurate assessment of the corresponding blood pressure regulation of young athletes under exercise conditions.

KEYWORDS

exercise testing, blood pressure, adolescent athletes, upper limits of blood pressure, sport

Introduction

Arterial hypertension is one of the major treatable cardiovascular risk factors (1). It is associated with elevated mortality and the incidence of heart insufficiency, myocardial infarcts, and apoplex (2). Elevated blood pressure in high-performance athletes is one of the most significant risk factors for cardiovascular diseases (3). Exaggerated blood pressure response to exercise testing is commonly regarded as a predictor of developing overt hypertension (4, 5). However, findings in adults are inconsistent (6), and no commonly accepted upper limits indicative of increased risks have been defined so far (7). There are only a few recommendations for tolerable upper blood pressure limits in exercise testing (8, 9). In the Guidelines of the European Society of Cardiology (ESC), it states that an exceeding systolic blood pressure (SB) of 210 mmHg in men and 190 mmHg in women has been termed “exercise hypertension” (7). In the American Heart Association (AHA) guideline for exercise testing (10) a limit of 214 mmHg (based on the 90th percentile calculated from >27,000 treadmill tests (11) is reported beyond which the risk of developing hypertension appears particularly increased (7). Compared to adults, the prevalence of elevated blood pressure in children and adolescents is clearly lower. However, there is a correlation between elevated blood pressure in children and relation to obesity. In contrast to the recommended upper blood pressure limits in adults, the definition of arterial hypertension in children and adults is based on body height and age-dependent limits. These blood pressure limits are determined in healthy children and adolescents. However, such blood pressure limits do not exist for young high-performance athletes, who are exposed to frequent exercise-induced blood pressure increase. There is only limited data available in which the effects of exercise-induced blood pressure elevation have been investigated in children and adolescents. Furthermore, very little is known about the exercise-induced blood pressure response in children. The study by Wanne et al. (12) investigated the blood pressure response under maximal dynamic movement in 497 healthy 9 to 18-year-old on a treadmill in young non-athletes. They described higher systolic values in postpuberty youths than in prepuberty. Szmigielska et al. (13) examined 711 (age 10–18 y) young athletes (training load $7.62 \text{ h} \pm 4.2 \text{ h}$ per week). In the maximal testing on the bike ergometer, the SBP was significantly higher in boys than in girls ($183.2 \pm 27.9 \text{ mmHg}$ vs. $170.9 \pm 21.4 \text{ mmHg}$, $p = 0.03$). Description of normative response to physical exercise in healthy children and adolescents in terms of percentiles was just recently given by Sasaki et al. and Clark and al. In the study by Clarke and colleagues normative percentiles of blood pressure response on a treadmill for healthy children and adolescents were described (14) in contrast to resting blood pressure and individual height which was not considered in the study by

Sasaki et al. (15) Although exercise testing in young elite athletes is frequently performed during preparticipation screenings, very little is known about the “normal” magnitude and distribution of exercise-induced blood pressure in this cohort. Therefore, this study aimed to evaluate the magnitude and determinants of blood pressure response to dynamic exercise testing in young elite athletes.

Materials and methods

Study population

In the department of sports medicine at the Institute for applied Training Science (IAT) in Leipzig, mainly high-performance athletes, from different sports, are seen mostly for preparticipation screenings. A retrospective analysis of our database from 2010 to 2019 for exercise testing on a bicycle ergometer identified 4,899 athletes. After excluding athletes with an age of >20 years, 2,217 datasets remained. Since the exercise-induced blood pressure regulation was intended to be analyzed in contrast with echocardiographic findings, only exercise tests of athletes were included who had a complete echocardiographic examination within the same year. After data adjustments for doublings, 739 datasets of young athletes remained. Since most of the echocardiographic studies were performed on the day of exercise testing or within a few weeks relevant breaks from sport after injury or illness between exercise testing and echocardiography can be ruled out in general. All athletes were highly trained elite athletes of their sports and members of the national team squad according to their age. None of the tested pupils suffered from any kind of cardiovascular disease nor took any kind of medication that might have an effect on the intrinsic blood pressure reaction

Anthropometry

Height and weight were measured using a scale with integrated straightedge (Seca scale, Model 701 with telescope measuring stick Model 220, Seca GmbH & Co.Kg. Hamburg, Germany), and body mass index (BMI) was calculated by dividing weight in kilograms through the square of height in meters. Body fat was determined by skinfold thickness using the ten-fold model introduced by Parizkova (16). Fat percentage was then used to partition total body mass into fat mass and fat-free mass. Resting and systolic blood pressure (rSBP) and diastolic blood pressure (rDBP) was measured with a standard sphygmomanometer (Erka, Bad Tölz, Germany) adjusted to the individual's arm circumference on both arms in a sitting position after at least 5 min of resting; the average was calculated for the present study. Exercise systolic blood pressure (eSBP) and diastolic blood pressure

(eDBP) was measured with with the similar sphygmomanometer (Erka, Bad Tölz, Germany).

Exercise testing

Tests were performed on cycle ergometers (Lode sport Excalibur, Lode, Groningen, the Netherlands). The standard protocol for all females started with an initial workload of 50 Watt (W), increment 30 W; stage duration was 3 min. Only petite and lightweight girls started with an initial workload of 25 W. The initial workload for all male athletes was 100 W (increment 30 W, stage duration 3 min). Boys of little height or weight (<50 kg) could also be tested with the girl's protocol. The supervising physician decided the test protocol. All tests were supervised by experienced staff and were performed until subjective exhaustion. All participants were motivated to reach their maximal limits of work capacity by oral motivation. Since all participants were experienced athletes, all participants were familiar with voluntary maximal exhaustion. Heart rates and BP were measured at the end of each stage, including maximal SBP and DBP (mSBP; mDBP). Heart rates were obtained automatically from 12-channel ECG (cardio 300, Custo med GmbH, Ottobrunn, Germany) but were verified and corrected (if necessary) manually afterward. BP was measured manually using standard sphygmomanometers adjusted to the individual's arm circumference.

Echocardiography

Experienced echocardiographers performed two-dimensional echocardiography according to guidelines of the German Society of Cardiology valid for the study period (Compare: Hagedorff et al.; Manual zur Indikation und Durchführung der Echokardiographie). From 2010 to 2014, Philips IE 33 system (Philips Healthcare, Hamburg, Germany) and after 2014 a Philips Epiq, both with a 3.5 MHz transducer. Left atrial diameters (anteroposterior) were assessed from either B-mode or M-mode parasternal long-axis views. LV end-diastolic diameter and septal wall thickness were measured in the parasternal long-axis at the level of the LV minor axis, approximately at the mitral valve leaflet tips. The absolute and relative mass of the heart was calculated using the formula established by (17) Pulsed Doppler profiles at the distal margins of the mitral valve leaflets, deceleration time of the E wave and, tissue-Doppler-derived mitral annular velocities (average of septal and lateral E') were assessed as indices of diastolic function. Movement in the M-mode (TAPSE and MAPSE) further assessed the function of the right and the left ventricle. For the relevant echocardiographic of this study z-scores were calculated. During the time of the study interval overall 3 physicians were involved in

echocardiographic data gathering. One of the examiners was a specialist for sports medicine with more than 20 years experience in echocardiography, one was cardiologist and sports cardiologist with the highest certified level of training in transthoracic echocardiography and one was resident physician in training for internal medicine and sports medicine who was trained and observed in echocardiography by the cardiologist. Complete echocardiographic data were available for all athletes of the presented study.

Sporting disciplines and training intensity

The classification of sports by Schnabel and Thieß (18) subdivides disciplines in technical-acrobatic sports, double-fight, endurance, sprint power/ strength and game sports. Based on this, all athletes were classified in one of these clusters to compare sport related differences in exercise related BP reaction. Furthermore, all athletes were clustered for their individual training intensity (by training hours per week) 4 clusters of training intensity were defined: 1–5, 6–10, 11–15 and 16–20 h/week. Given that not only the training intensity but also the age has an impact on the regulation of the BP during exercise, the component of age was considered separately. Athletes aged 10–12 years, 13–15 years, 16–18 years, and 18–20 years were clustered for age-dependent examinations.

Statistical analysis

The data obtained from the stress tests and echocardiographic examinations were processed in Microsoft Office Excel 2016 for Windows from Microsoft Corporation (Redmond, USA) and statistically calculated using IBM SPSS Statistics 25.

Except for age, all data fulfilled criteria for normal statistical distribution as verified by previous authors (19) thus consistent descriptive presentation as meanSD was chosen for all variables including age. Mean comparisons were calculated with a single factorial variance analysis. Due to the literature (20), the robust Welch test was also tested for corresponding significance. In significant differences, a posthoc test and Bonferroni correction were calculated. The significance level was $\alpha = 0.05$. According to Cohen (1988), the effect sizes were interpreted as follows: 0.01 small effect, 0.06 medium effect, and 0.14 large effect. Correlation effects are described by the correlation coefficient $|r|$. According to Cohen Guidelines (1988) data was interpreted as: $|r| = 0.10$ a weak $|r| = 0.30$ a moderate $|r| = 0.50$ describe a strong correlation. Associations of variables were analyzed using Pearson's correlation coefficients. Normative ranges were defined as the central 95% of all observations, and lower and upper limits were estimated assuming normality as means ± 2 -fold SD. Additionally, 95% confidence

intervals for limits of normative ranges are presented in squared brackets [e.g., (1234)].

Results

Study population

A total of $n = 739$ athletes (male = 442, female = 297) with a median age of 15.8 years (± 2.3 years) were analyzed. There was no significant difference in age and duration of training years between male and female athletes. The demographic and clinical characteristics of this population are summarized in **Table 1**. Male athletes were significantly heavier and taller than their female counterparts, had less body fat, a higher fat-free mass (FFM), and a higher resting blood pressure rSBP and rDBP (Compare **Table 1**)

Echocardiographic findings at baseline

An overview of the echocardiographic data is shown in **Table 2** below. Z-scores of the relevant echocardiographic results are given in **Table 3**. The absolute cardiac size in male athletes was significantly higher (815.4 ± 188.2 g) than in female athletes (637.7 ± 115.4 g). In relation to the body weight, the male heart volume was also higher 11.85 ± 1.54 g/kg body weight than that of the female 10.79 ± 1.56 g/kg body weight. There were no significant differences in both the medial and lateral E wave and the right ventricular function in the form of TAPSE (tricuspid annular plane systolic excursion). The diameter values of the left ventricular posterior wall during the diastole is within the normal range for both female and male post-growth athletes. The male subjects have significantly higher wall thicknesses of $0.99 \pm$

0.15 cm than the female subjects with 0.88 ± 0.13 cm ($p < 0.001$). The diameter of the left atrium 3.42 ± 0.43 (♂), bzw. 3.2 ± 0.41 cm (♀) was within the standard range. The values of male athletes were significantly higher than those of female athletes [0.221; 95% CI (0.158, 0.283)]. The differences between the sexes in the left ventricular diameter's values during the diastole (LVEDD) and the interventricular septum thickness were significantly different, too (both with $p.001$). All subjects were in the standard range with their respective values. All relevant echocardiographic data are presented in relation to athletes age and sex in **Figures 4–11**.

Exercise-induced blood pressure response

Table 4 summarizes the findings related to exercise testing. SBP increased significantly ($p < 0.001$ for both sexes), whereas DBP remained almost unchanged in female and male athletes. Male athletes absolved more exercise stages, started with a

TABLE 1 Baseline anthropometric and clinical characteristics of study population.

| | All | Male | Female | p-value |
|--------------------------|-------------|-------------|-------------|-------------|
| Number of participants | 739 | 442 (59.8%) | 297 (40.2%) | |
| Age (years) | 15.8 ± 2.3 | 15.9 ± 2.8 | 15.6 ± 2.4 | $p = 0.149$ |
| Years of training | 6.7 ± 2.8 | 6.7 ± 2.8 | 6.7 ± 2.6 | $p = 0.834$ |
| Hrs. of training/week | 14 ± 6.2 | 13.8 ± 6.3 | 14.4 ± 6 | $p = 0.194$ |
| BMI (kg/m ²) | 21.2 ± 3.2 | 21.3 ± 3.2 | 21.1 ± 3.1 | $p = 0.370$ |
| Height (cm) | 175 ± 11.3 | 179 ± 11.1 | 168 ± 7.6 | $p < 0.001$ |
| Weight (kg) | 65.5 ± 14.8 | 69.3 ± 15.5 | 59.9 ± 11.5 | $p < 0.001$ |
| Fat mass (kg) | 10.1 ± 4.6 | 9.2 ± 4.1 | 11.3 ± 4.9 | $p < 0.001$ |
| Body fat % | 15.3 ± 5.0 | 13.2 ± 4.0 | 18.4 ± 4.7 | $p < 0.001$ |
| Lean mass (kg) | 55.5 ± 12.5 | 60.1 ± 13 | 48.7 ± 7.9 | $p < 0.001$ |
| Resting SBP (mmHg) | 121 ± 12 | 124 ± 13 | 117 ± 10 | $p < 0.001$ |
| Resting DBP (mmHg) | 78 ± 8 | 80 ± 8 | 76 ± 8 | $p < 0.001$ |

TABLE 2 Echocardiographic characteristics of study population.

| variable | all | ♂ | ♀ | p-value |
|----------------------------------------|---------------|---------------|---------------|-------------|
| e'-wave lateral (cm/s) | 5.16 ± 1.08 | 5.12 ± 1.07 | 5.22 ± 1.11 | $p = 0.210$ |
| e'-wave medial (cm/s) | 7.46 ± 1.50 | 7.40 ± 1.48 | 7.55 ± 1.53 | $p = 0.191$ |
| Heart volume absolute (g) | 743.9 ± 184.7 | 815.4 ± 188.2 | 637.7 ± 115.4 | $p < 0.001$ |
| Relative heart volume (g/kg) | 11.42 ± 1.64 | 11.85 ± 1.54 | 10.79 ± 1.56 | $p < 0.001$ |
| Left ventr. posterior wall (cm) | 0.95 ± 0.15 | 0.99 ± 0.15 | 0.88 ± 0.13 | $p < 0.001$ |
| TAPSE (cm) | 2.43 ± 0.38 | 2.44 ± 0.38 | 2.40 ± 0.38 | $p = 0.228$ |
| Endsystolic left atrial diameter (cm) | 3.33 ± 0.44 | 3.42 ± 0.43 | 3.20 ± 0.41 | $p < 0.001$ |
| Left ventr. enddiastolic diameter (cm) | 4.90 ± 0.47 | 5.05 ± 0.46 | 4.68 ± 0.39 | $p < 0.001$ |
| Enddiastolic septal wall (cm) | 0.90 ± 0.13 | 0.94 ± 0.13 | 0.84 ± 0.11 | $p < 0.001$ |

TABLE 3 Z-scores of echocardiographic results.

| Age group | Sex | Mean value | | | |
|-----------|-----|------------|--------|---------|---------|
| | | IVSd-Z | IVSs-Z | LVIDd-Z | LVIDs-Z |
| 10–12 | M | 0.849 | 1.109 | 0.077 | 0.035 |
| 10–12 | W | 0.689 | 0.757 | 0.102 | 0.113 |
| 13–15 | M | 0.757 | 1.261 | 0.345 | 0.266 |
| 13–15 | W | 0.469 | 0.840 | 0.130 | 0.079 |
| 16–17 | M | 0.766 | 1.454 | 0.070 | 0.169 |
| 16–17 | W | 0.481 | 0.895 | -0.173 | -0.094 |
| 18–20 | M | 1.166 | 1.842 | -0.111 | 0.222 |
| 18–20 | W | 0.938 | 0.840 | -0.270 | 0.139 |

higher workload, attained higher peak workloads, and showed a more pronounced SBP response than female athletes

Influence of age on the maximum of exercise-induced blood pressure

In the cohort of 10–12 year old junior athletes, the SBP at maximal effort was 159 mmHg (± 18 mmHg). In the progression of the age of the athletes, the SBP increased continuously. 13–15 year old athletes had an average SBP of 182 ± 20 mmHg, 16–17 year old athletes 192 ± 20 mmHg, and 18–20 year old athletes 196 ± 22 mmHg. There was a significant difference between male and female athletes in which male athletes gained higher values. Within the different age cohorts, the SBP was also significantly different between sexes. (see **Figure 1**)

Influence of training frequency

The influence of the training frequency on the maxSBP is shown in **Figure 2**. The statistical calculation showed that there were significant differences between the frequency of the training and the blood pressure development of the subjects ($F = 11.823$, $p < 0.001$). The post-hoc comparison of the categories of these significant differences between the lowest training frequency of 1–5 h/week and 6–10 h/week (-11.161 ;

95% CI $[-16.43; -5.89]$ and between a training effort of 1–5 h and 11–15 h per week (-14.754 ; 95% CI $[-23.07; -6.44]$). The maximum systolic blood pressure of children and adolescents is initially linear. From a training range of 6–10 h, the values flatten and the blood pressure increases only minimally in both sexes. The maximum values are reached with a training range of 16–20 h.

Influence of sporting type

To investigate the influence of the sporting type, the athletes were divided into sporting type groups according to Schnabel (1993) in technical-acrobatic sports, double-fight, endurance, sprint power/strength and game sports. The maximum systolic blood pressure values differed significantly in terms of the respective sports ($F(4;732) = 4.850$, $p = 0.001$). The highest mSBP values were observed in the sprint/ strength athletes with an average value of $190 (\pm 23)$ mmHg. The lowest values were recorded by athletes from technical-acrobatic sports with an average mSBP value of $178 (\pm 21)$ mmHg. Significant differences were found in the maximum systolic blood pressure values between the technical-acrobatic and endurance sports (-9.657 ; 95%-CI $[-17.12; 1.04]$) as well as the technical-acrobatic and rapid-power or strength athletes (-12.653 , 95%-CI $[-21.147; -3.83]$). In both cases, the technical-acrobatic athletes had lower mSBD values. Compare **Table 5**.

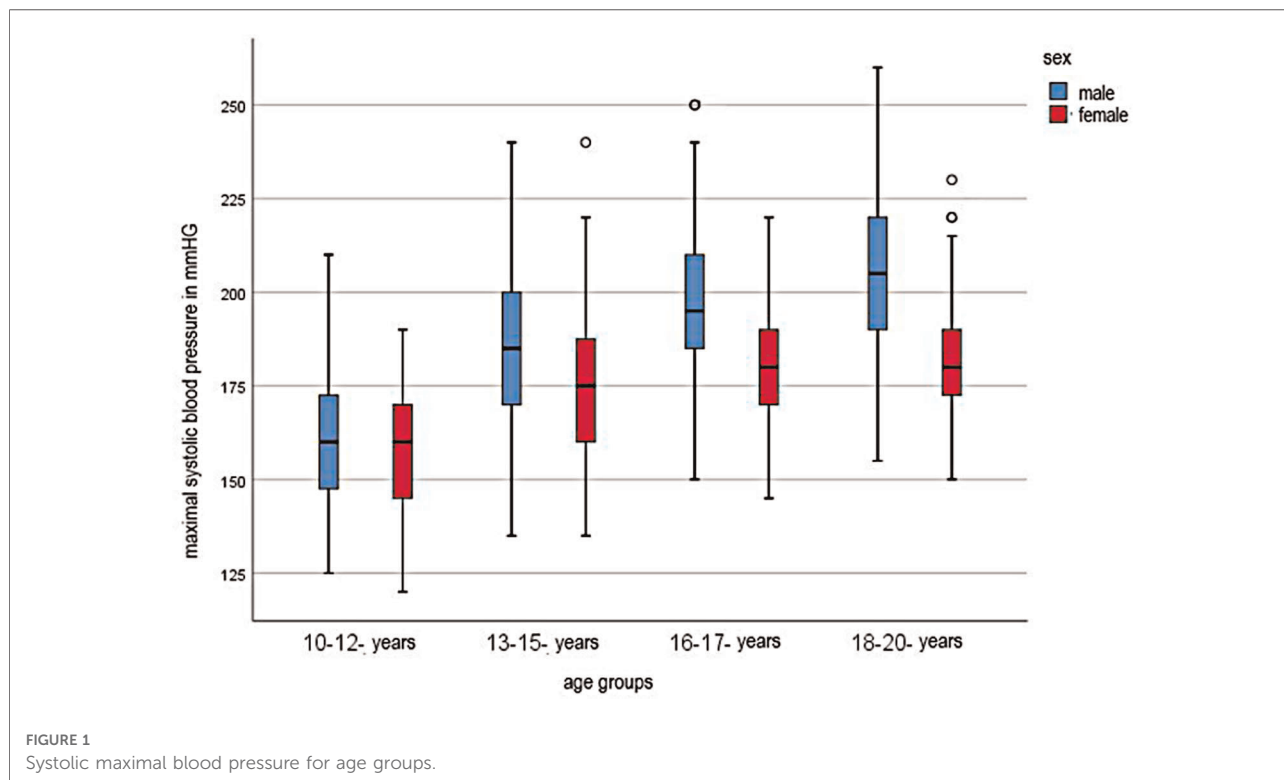


FIGURE 1
Systolic maximal blood pressure for age groups.

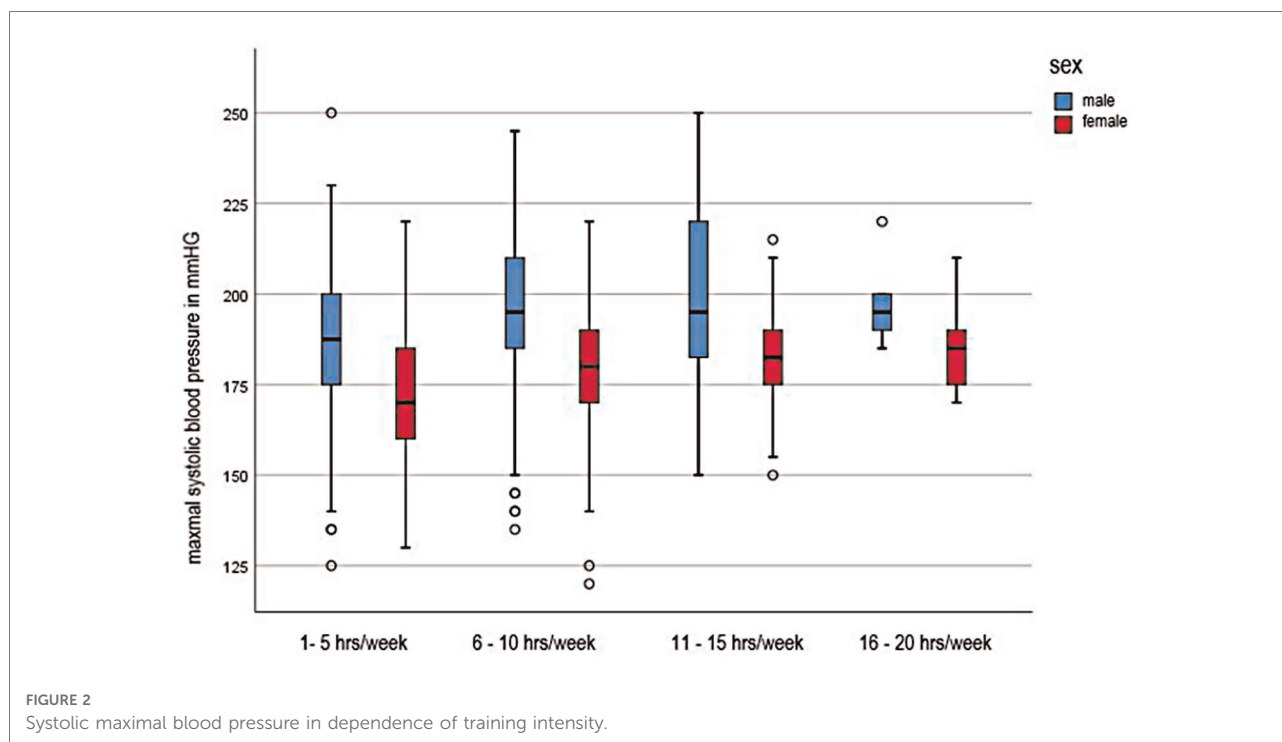


TABLE 4 Findings in exercise testing.

| | All | ♂ | ♀ | p-value |
|---------------------------------|-------------|-----------|-----------|------------------|
| Initial workload (watt) | 75.5 ± 10.6 | 92 ± 19 | 52 ± 9 | <i>p</i> < 0.001 |
| Max workload (watt) | 231 ± 68 | 258 ± 67 | 190 ± 45 | <i>p</i> < 0.001 |
| Max workload relative (watt/kg) | 3.5 ± 0.8 | 3.8 ± 0.7 | 3.2 ± 0.7 | <i>p</i> < 0.001 |
| Max lactat (mmol/L) | 8.7 ± 2.4 | 8.8 ± 2.4 | 8.3 ± 2.5 | <i>p</i> = 0.015 |
| Max SBP (mmHg) | 187 ± 23 | 193 ± 23 | 177 ± 19 | <i>p</i> < 0.001 |
| Max DBP (mmHg) | 80 ± 9 | 81 ± 8 | 79 ± 9 | <i>p</i> < 0.001 |
| Max. heart rate (x/min) | 189 ± 11 | 189 ± 11 | 189 ± 11 | <i>p</i> = 0.622 |

Association of the mSBP with cardiac dimensions

The Pearson correlation coefficient shows a moderate positive correlation between the relative heart volume (g/kg BM) and the mSBD ($r=0.171$). The thicker the interventricular septum during the junior athletes' diastole, the higher the systolic rest values were ($r=0.323$). The effect can be interpreted as moderate. If one combines above two echo parameter with the maximum systolic blood pressure value, the effect ($r=0.490$) strengthens and indicates that the higher the echocardiographic diameters, the higher the mSBD values during the load.

Finally, it was investigated whether those young athletes who had significantly higher systolic blood pressure levels during the stress test also showed corresponding abnormalities in cardiac ultrasound. (see **Figure 3**) Significant differences of

the mean SBP were found in the statistical calculation of the left ventricular diameter during the diastole, $F(3; 738) = 27.552$, $p.001$ effect strength high ($\eta^2 = 0.101$) (Cohen, 1988). It is noticeable that with increasing deviation from the mean, the diameters in the left ventricle also increase during the diastole. Those young athletes who had a maximum systolic blood pressure value >229 mmHg also showed the highest end-diastolic diameter. The differences were highly significant ($F(3.738) = 28.134$, $p.001$) with also high effect strength ($\eta^2 = 0.103$). Both between the single and the double SD (-0.0983 ; 95%-CI $[-0.136; -0.061]$) and between the simple and the more than double SD, i.e., a systolic blood pressure development of more than 229 mmHg (-0.1376 ; 95%-CI $[-0.193; -0.082]$), the calculations showed highly significant results. Equally significant were the calculations of the left ventricular posterior wall thickness ($p.001$). Similar to the above results, significant differences between the single and the double SD (-0.1099 ; 95% CI $[-0.152; -0.068]$) and between the simple SD and the classification >229 mmHg (-0.1374 , 95% CI $[-0.2; -0.075]$) were found. Concerning right ventricular function in the form of TAPSE, the groups do not differ significantly ($p=0.089$). The left atrium diameter showed significant differences between the mSBD classifications, $F(3.727) = 12.308$, $p.001$. The post-hoc testing significant differences between the group of athletes with single SD and double SD (-0.236 ; 95%-CI $[-0.365; -0.108]$) and between single SD and the classification group "blood pressure >229 mg" (-0.288 ; 95%-CI $[-0.478; -0.096]$).

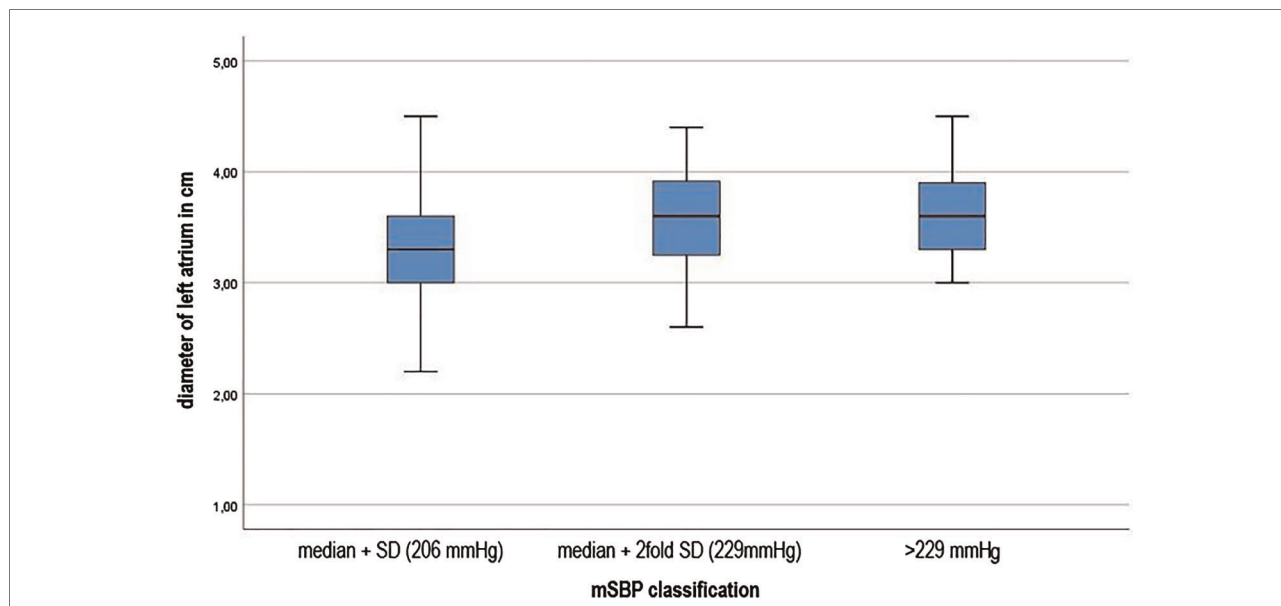


FIGURE 3 Left atrial dimension in dependence of increasing deviation from the mean (SBP).

Blood pressure percentiles

Of the 739 studied young athletes, percentiles of blood pressure levels were calculated. These were initially calculated jointly for both sexes and all age groups. Within the 95th percentile are blood pressure values diastolic of 95 mmHg and systolic of 230 mmHg. Across all age groups and separated by gender, 95% of young male athletes aged 10–20 years have a blood pressure below 230/95 mmHg. For female athletes, the 95th percentile is 206/90 mmHg.

In addition to distinguishing blood pressure limits by sex, age-dependent distinctions are of particular interest. Thus, all subjects were additionally assigned to a corresponding age group. For each age group, blood pressure limits have now been determined by calculating the percentiles, which can quickly provide information about possible exercise-induced hypertonia (see **Tables 6–8**).

Discussion

This work aimed to investigate the blood pressure behavior of young athletes on the bicycle ergometer and to establish blood pressure limits that apply to young competitive athletes. In contrast to many exercise testing in international studies ergometry was conducted on the bicycle ergometer. Since the reason for exercise testing was focused on preparticipation screening in sports the quality of the exercise ECG to detect any cardiac pathology is essential. In terms of torso

TABLE 5 Influence of sporting type.

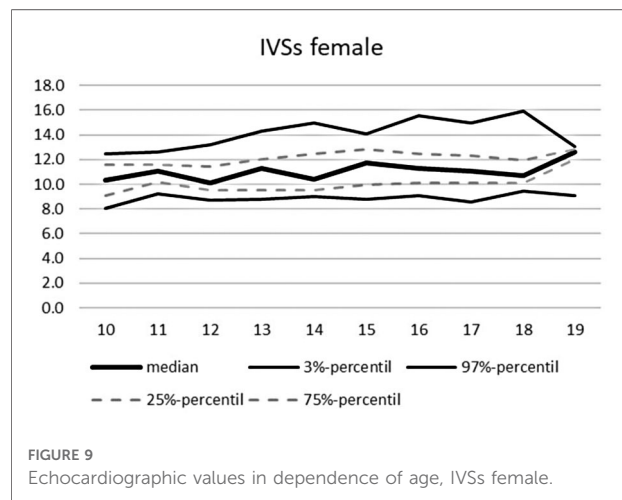
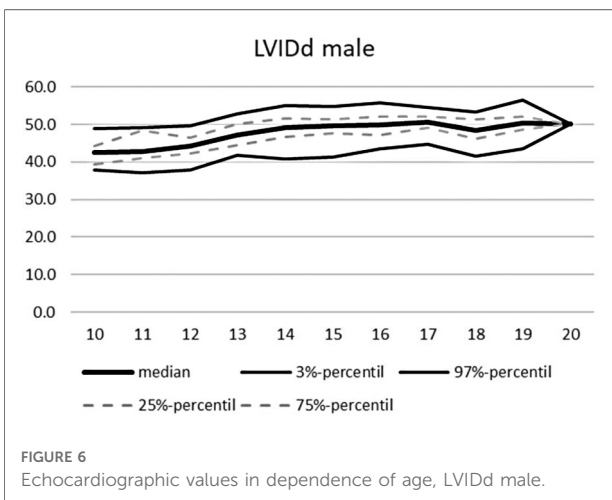
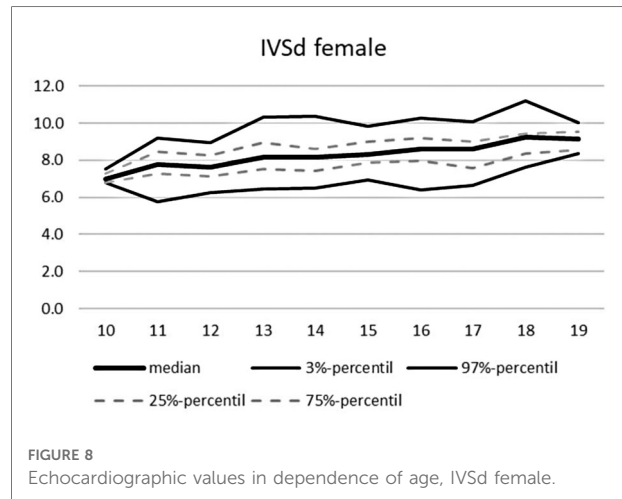
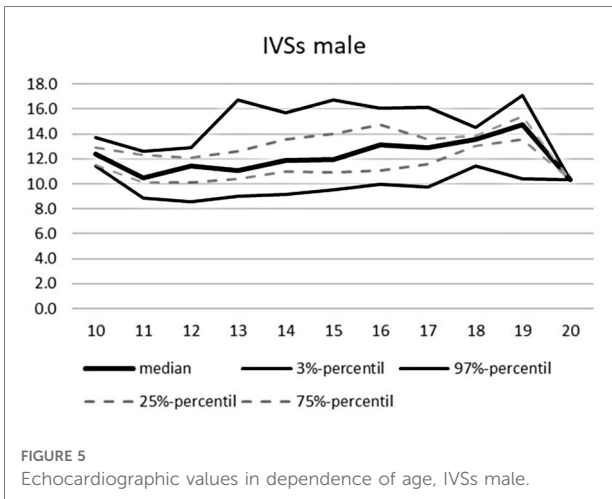
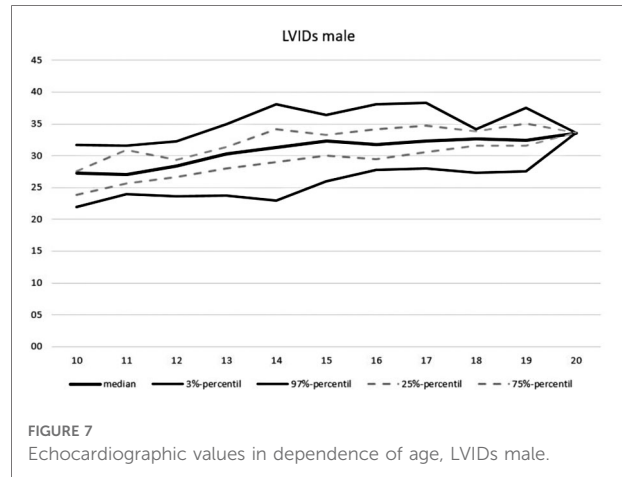
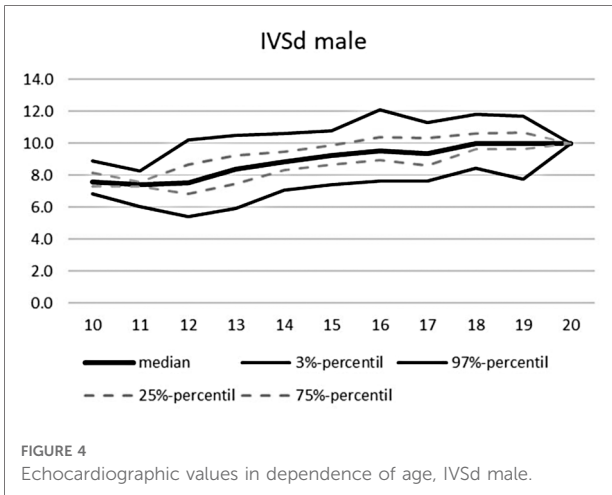
| Type of sport | n | mSBD (MW ± SD) |
|----------------------------|-----|----------------|
| Technical-acrobatic sports | 97 | 178 ± 21 |
| Double-fight | 100 | 178 ± 21 |
| Endurance | 316 | 187 ± 23 |
| Sprint power/ strength | 113 | 190 ± 23 |
| Game sports | 112 | 189 ± 23 |

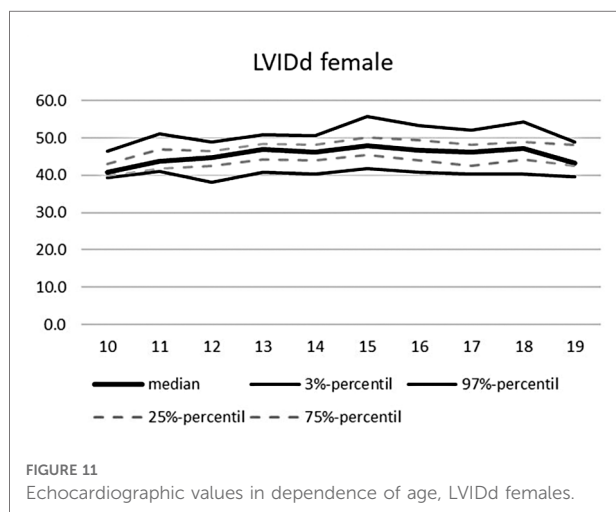
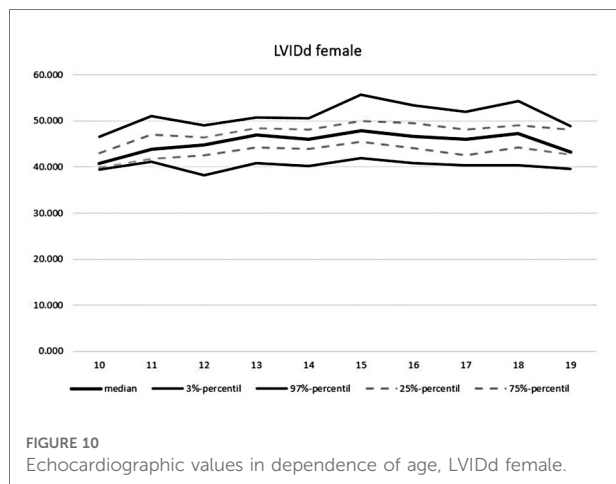
TABLE 6 Age dependent blood pressure percentiles of maximal systolic blood pressure in mmHg for male and female junior athletes.

| Percentiles | Age | | | |
|----------------|-----------------------|------------------------|------------------------|-----------------------|
| | 10–12-years n = 91 | 13–15-years n = 342 | 16–17-years n = 230 | 18–20-years n = 90 |
| 75. percentile | 173 mmHg | 195 mmHg | 200 mmHg | 215 mmHg |
| 90. percentile | 180 mmHg | 210 mmHg | 220 mmHg | 227 mmHg |
| 95. percentile | 190 mmHg | 217 mmHg | 235 mmHg | 230 mmHg |
| 99. percentile | — | 236 mmHg | 248 mmHg | 247 mmHg |

TABLE 7 Age dependent blood pressure percentiles of male junior athletes.

| Percentiles | Age | | | |
|----------------|----------------|----------------|----------------|----------------|
| | 10–12-year old | 13–15-year old | 16–17-year old | 18–20-year old |
| 75. percentile | 175 mmHg | 200 mmHg | 210 mmHg | 220 mmHg |
| 90. percentile | 181 mmHg | 210 mmHg | 230 mmHg | 230 mmHg |
| 95. percentile | 200 mmHg | 220 mmHg | 240 mmHg | 240 mmHg |
| 99. percentile | — | 237 mmHg | 250 mmHg | 258 mmHg |





movement related ECG artefacts the ECG quality is much higher than during an exercise ECG testing on a treadmill.

According to the guidelines of the European Society of Hypertension (ESH), the resting blood pressure values of the athletes are within the normal range (21). In this study, male athletes had slightly higher resting blood pressure levels on average than female athletes. The studies of Pressler et al. (7) and Al-Sendi et al. (22) confirmed these gender differences in resting blood pressure levels. These gender differences are thought to be hormonal (23). The blood pressure progression of our subjects until exhaustion is similar to that described in previous studies (7, 24). The systolic blood pressure increases linearly with the load in all subjects. Diastolic blood pressure, on the other hand, flattened by a maximum of 22 mmHg from start to the last stage (25). However, diastolic blood pressure under maximum stress is often measured incorrectly too low using the Riva Rocci method (26)

TABLE 8 Age dependent blood pressure percentiles of female younior athletes.

| Percentiles | Age | | | |
|----------------|----------------|----------------|----------------|----------------|
| | 10–12-year old | 13–15-year old | 16–17-year old | 18–20-year old |
| 75. percentile | 171 mmHg | 190 mmHg | 190 mmHg | 190 mmHg |
| 90. percentile | 183 mmHg | 200 mmHg | 200 mmHg | 200 mmHg |
| 95. percentile | 190 mmHg | 208 mmHg | 200 mmHg | 217 mmHg |
| 99. percentile | — | 238 mmHg | — | — |

The sex, the age, the proportion of fat-free mass and the amount of training exercise influence the development of the maximum systolic blood pressure. Male athletes had higher mSBD levels and an equally higher fat-free mass than their female counterparts did. It is believed that the higher weight associated with increased muscle mass allows for higher performance on the ergometer. The extent of the increase in systolic blood pressure is also related to the mass of muscles involved (25). However, if one looks at the relative workload per kilogram, the difference between the sexes is virtually non-existent. As the relative workload increases, so does the maximum blood pressure.

That the age component has a decisive influence on the development of maximum systolic blood pressure under stress (27), was confirmed in this study. In the Kiel EX. PRESS study (27) reference values for systolic blood pressure limits derived from bicycle ergometry tests were described. In addition, our study found an influence of age on the development of maximum systolic blood pressure values. The oldest study group with an age of 18–20 years had the highest maximum blood pressure values (196 ± 22 mmHg), the youngest subjects (10–12 years) the lowest. Since this data is collected on high-performance athletes who are (almost) daily exposed to their sports specific training, our values are significantly higher than previously described (27)

The training frequency parameter had a significant influence on the maximum systolic blood pressure development of young athletes. It confirmed the results of the meta-analysis of Berge et al. (2015). The research team around Berge classified the training amount into two categories: >10 h per week and 10 h of training effort per week. We were able to confirm the fact that those athletes who exercised more frequently (>10 h/week) had higher blood pressure development. With a four-part classification of the training frequency (1–5 h, 6–10 h, 11–15 h, 16–20 h), a linear relationship between training frequency and mSBD development could be revealed across genders. However, what is striking about both sexes is that the mSBD forms a plateau from a training range of 6–10 h and then only increases minimally with increasing training scope. The maximum values are reached for both sexes with a training range of 16–20 h. These results suggest that from a training volume of 6 h per week, sports-related adjustment reactions of blood pressure occur.

Influence of the sports type can be found as well. The technical-acrobatic sports results were significantly different compared to endurance athletes and sprint/strength and strength sports. In both cases, the technical-acrobatic athletes had a lower maximum systolic blood pressure level. This result differs from other study results (3, 28). We assume that a very different composition of sports can explain these divergent results. In addition, in some sports, unattained maximal cardiovascular stress due to premature muscular fatigue must be assumed. Premature test cancellation meant that maximum blood pressure might not have been reached.

When looking at the subjects with the highest systolic blood pressure values in the exhaustion, significant abnormalities could be identified in single athletes. A canoeist and a cross-country skier had a maximum value of 260 and 250 mmHg, by far the highest systolic values. Both athletes also performed correspondingly high but not the highest relative workloads. However, it is also noticeable that in both athletes, the systolic resting blood pressure was above the age-appropriate limit. Excitement, but also cardiovascular regulation disorders, can be the cause of this.

On the contrary, by looking at those athletes who stand out with a particularly good relative performance, it becomes clear that in these subjects, the maximum systolic blood pressure only assumes values of 200 mmHg. All these athletes are even in their 90th percentile of upper systolic blood pressure levels. As a result, no direct linear relationship can be determined between an excessive blood pressure reaction and a higher relative performance on the bicycle ergometer.

Overall, the majority of the athletes studied ($n = 610$, 82,5%) are in the range of the calculated mean value plus the simple standard deviation in terms of the maximum systolic blood pressure reaction (<206 mmHg). According to the calculated percentiles, this corresponds to the 90th and 95th percentiles in all age ranges. Ninety-one subjects had mSBD values above the mean up to twice the standard deviation (up to 229 mmHg).

In 38 of the 739 athletes studied, the systolic maximum values during the maximal workload were even higher than a deviation of twice the SD of the mean (>229 mmHg). Thirteen of the 38 athletes with excessive blood pressure reaction above systolic values of 229 mmHg correspondingly had norm-deviating systolic rest values of >140 mmHg. Whether these deviations are excitement-related increases, or already an indication of arterial hypertension, is to be discussed.

Those young athletes who had a maximum systolic blood pressure value >229 mmHg showed significant different (higher) values for inner left ventricular dimension in the systolic and diastolic and the left ventricular posterior wall thickness. Therefore these athletes might need to be monitored more closely during their career, if both cardiac function and dimensions and blood pressure regulation show any signs of pathologic development.

Due to the increase in blood pressure and the associated increase in intramyocardial pressure, there may be increased wall stress on the heart during a training career [see, among others (29)]. Echocardiography is an important examination methodology for assessing and demarcating a pathological adaptation from a physiological (sport-related) adaptation. Overall, the results from the statistical calculations show a few echocardiographic abnormalities, although the vast majority of athletes are within the norm values for excluding pathological cardiac changes.

Limitations

However, since this work is only a cross-sectional analysis, the long-term effects of stress-induced hypertension are neglected. Mainly the long-standing competitive sport with numerous intensive training sessions and competitions provokes functional and structural changes to the musculoskeletal system and the cardiovascular system (30). To rule out pathological changes due to intensive training, further, regular examinations must take place for each athlete during his/her career. Only in this way, can the influence of a long-term high training workload reveal individual cardiovascular risks and provide information about how the body deals with high blood pressure provoked by training in the long term.

The presented blood pressure percentiles for children and adolescents are related to sex and age to simplify the graphic representation. The aspect of body height, which is known to effect the age dependent resting blood pressure was not taken into account and might lead to misinterpreting the exercise blood pressure children and adolescents.

Another limitation of this work is that a pre-test activity, the nutritional status of the athletes, but also temporary emotional and psychological stress, such as in school or family, were not taken into account. However, all these factors can influence athletic performance and resulting blood pressure. The extent to which they have an impact remains debatable.

To define cluster of paediatric subjects by age may not be appropriate when investigating the influence of physical maturity on parameters. Unfortunately no data of puberty was gathered during the regular clinical assessment. Therefore the influence of pre- or late puberty on exercise related blood pressure regulation cannot be assessed in this study.

Conclusion

In this study, the blood pressure behavior of young athletes aged 10–20 years was investigated and evaluated retrospectively. Overall, the young athletes show standard-compliant systolic and diastolic rest blood pressure values in the results of the

examination. Only the level of maximum blood pressure values differs between athletes and non-athletes.

For the first time, blood exercise related pressure percentiles were defined for junior athletes across sports. Competitive athletes in the junior field achieve higher blood pressure values due to physiological training adaptations and tolerate overall higher maximum blood pressure values than subjects who do not exercise.

However, children and adolescents who are above these limits during exercise exposure may be at higher risk of cardiovascular disease.

Overall, a large proportion of the athletes studied already show cardiac adaptation reactions that have a positive effect on performance. However, those subjects who had excessive BD reactions showed significantly higher chamber septum thicknesses during the diastole and higher left ventricular posterior wall thicknesses compared to their colleagues. Although these values are still within the range of the athlete-specific norm values, a tendency related to blood pressure is visible. Regular medical examinations should further be carried out to ensure the long-term health of all athletes.

In general, there are only a few studies devoted to blood pressure behavior in childhood and adolescence. There is a need for research, especially to the question of what causes stress-induced hypertension in the long term. For this purpose, further longitudinal studies should be done examining children and adolescents from junior performance sports several times during their sporting careers for abnormalities.

Data availability statement

The raw data supporting the conclusions of this article will be made available by the authors, without undue reservation.

Ethics statement

Ethical review and approval was not required for the study on human participants in accordance with the local legislation and institutional requirements. Written informed consent to participate in this study was provided by the participants' legal guardian/next of kin.

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Author contributions

JW: initial research idea, implementation of the medical examinations and stress tests, author of the text, monitoring of the research project, Data analysis and data interpretation. FB: Data analysis and data interpretation, assistance in text writing. PR: Data analysis and data interpretation, implementation of the stress tests. WB: supervising of the research project, Data analysis and data interpretation. CP: data interpretation, assistance in text writing. All authors contributed to the article and approved the submitted version.

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Conflict of interest

The handling Editor [I.S] declared a past co-authorship with the author [J.W]. The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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2.3. Different habitus but similar electrocardiogram: Cardiac repolarization parameters in children; Comparison of elite athletes to obese children.

Fortführend zu den Überlegungen zur Veröffentlichung 2.2., dass es insbesondere in der sportmedizinischen Begutachtung von Kindern und Jugendlichen häufig nur eingeschränkte Daten zu Kindern und Jugendlichen und noch weniger Daten aus dem jeweiligen Elitebereich ihrer jeweiligen Sportart gibt, beschäftigt sich die dritte vorgestellte Arbeit mit einer der zentralen Fragestellungen der kardiologisch orientierten (Hochleistungs-) Sportmedizin: den sportbedingten EKG-Veränderungen. Gemäß den Ausführungen in der Einleitung hat es in den vergangenen Jahrzehnten umfangreiche Untersuchungen bei den verschiedensten Sportlern unterschiedlicher Sportarten, Alters- und Leistungsstufen, Trainingsumfängen etc. gegeben, die letztlich zu den oben genannten Empfehlungen zur Begutachtung von „Sportler-EKGs“ geführt haben (vgl. „Seattle Kriterien“ etc). Allerdings liegen vergleichbare Empfehlungen zur Interpretation von „Sportler-EKGs“ bei Leistungssporttreibenden Kindern- und Jugendlichen nur sehr eingeschränkt vor. Es ist wenig darüber bekannt, ob es „normale, physiologische“ EKG-Veränderungen in ähnlichem Ausmaß bei hochleistungssportlichen Kindern wie bei Leistungssportlern im Erwachsenenalter gibt. Dabei bot es sich an, dieses Kollektiv von Kindern und Jugendlichen mit einem weiteren Kollektiv der pädiatrischen Medizin zu vergleichen, die ebenfalls Veränderungen im Ruhe-EKG aufweisen könnten, hier jedoch aufgrund einer zugrundeliegenden bekannten Pathologie (Adipositas). Gemäß der Literatur wird als Ursache der bekannten EKG-Veränderungen (insbesondere der QT Zeit) bei adipösen Kindern neben einer fettbedingten erhöhten hormonellen Aktivität, ein deutlich erhöhter parasympathischer Einfluss angenommen. Ein deutlich erhöhter parasympathischer Einfluss wird jedoch auch durch intensives Ausdauertraining erreicht. Insofern lag die Vermutung nahe, dass es aufgrund des erhöhten parasympathischen Einflusses bei jungen Elitesportlern zu vergleichbaren EKG Veränderungen, wie bei adipösen Kindern kommt. Während, wie erwartet, die längsten QTc Zeiten bei den adipösen Kindern gefunden wurden, zeigten die Athleten relativ kurze QTc Zeiten. Des Weiteren wurde untersucht, ob innerhalb der Sporttreibenden Unterschiede hinsichtlich ihrer ausgeübten Hauptbelastungsart (Ausdauer- vs. Kraft-/Schnellkraftsportarten) bestehen. Allgemein konnte gezeigt werden, dass es in beiden Gruppen keine pathologischen Auffälligkeiten gab, jedoch vorzugsweise in der Gruppe der ausdauersporttreibenden Kinder, die weiter oben beschriebenen kürzere QT-Zeiten im Vergleich zu anderen Kollektiven an Kindern und Jugendlichen vorherrschen. Im Gegensatz zu erwachsenen Sportlern, bei denen eine QT-Zeit Verlängerung, bzw. Verlängerung der korrigierten QT-Zeit bekanntermaßen auftritt und bereits vielfach beschrieben wurde [98-101], konnte dies bei den jungen Sportlern nicht nachgewiesen werden.

Different habitus but similar electrocardiogram: Cardiac repolarization parameters in children – Comparison of elite athletes to obese children

Christian Paech¹, Janina Moser¹, Ingo Dähnert¹, Franziska Wagner¹, Roman Antonin Gebauer¹, Toralf Kirsten², Mandy Vogel², Wieland Kiess², Antje Körner², Bernd Wolfarth³, Jan Wüstenfeld³

¹Department for Pediatric Cardiology, University of Leipzig - Heart Center, Leipzig, Germany, ²LIFE Child (Leipzig Research Center for Civilization Diseases), University of Leipzig, Leipzig, Germany, ³University of Leipzig, Institute for Applied Scientific Training, Leipzig, Germany

ABSTRACT

- Introduction** : The standard 12-lead electrocardiogram (ECG) remains a widely used tool in the basic cardiac evaluation of children and adolescents. With the emergence of inherited arrhythmia syndromes, the period of cardiac repolarization has been the focus of attention. So far, data on cardiac repolarization and its normal variants in healthy children are scarce. This may cause uncertainties in the differentiation between pathologies and normal variants. As abnormal autonomic regulation seems to be a major influencing factor on cardiac repolarization, this study aimed to evaluate the parameters of cardiac repolarization of children in extremely good physical shape to obese children to improve knowledge about cardiac repolarization in these subgroups of pediatric patients that are vastly affected by the alterations of autonomic regulation.
- Methods** : A total of 426 pediatric volunteers (84 lean, healthy controls; 130 obese healthy pediatric volunteers; and 212 elite athletes) were enrolled in the study, and the parameters of cardiac repolarization were determined in 12-lead ECG.
- Results** : Most importantly, there were no pathological findings, neither in the healthy controls nor in the obese or athletes. Athletes showed overall shorter corrected QT intervals than children from the other groups. This is also true if a correction of the QT interval is performed using the Hodges formula to avoid bias due to a tendency to lower heart rates in athletes. Athletes showed the shortest Tpeak-to-end ratios between the groups. The comparison of athletes from primarily strength and power sports versus those from endurance sports showed endurance-trained athletes to have significantly longer QT intervals.
- Conclusions** : This study suggests that neither obesity nor extensive sports seems to result in pathological cardiac repolarization parameters in healthy children. Therefore, pathology has to be assumed if abnormal repolarization parameters are seen and might not be simply attributed to the child's habitus or an excellent level of fitness.
- Keywords** : Athlete, cardiac repolarization, children, long QT, obesity

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Address for correspondence: Dr. Christian Paech, Department for Pediatric Cardiology, University of Leipzig - Heart Center, Strümpellstr 39, 04289 Leipzig, Germany. E-mail: christian.paech@med.uni-leipzig.de

INTRODUCTION

The standard 12-lead electrocardiogram (ECG) remains a widely used tool in the basic cardiac evaluation of children and adolescents. With the emerging focus of present medicine to the preventive aspect and risk stratification, the inherited arrhythmia syndromes and thereby the period of cardiac repolarization have been the focus of attention.^[1-5] While the interpretation of cardiac arrhythmias is well established, the period of cardiac repolarization provides more challenges to the physicians.

To date, there are various clearly pathological phenomena of cardiac repolarization reported. Still, the interpretation of repolarization disturbances remains complex, as cardiac repolarization underlies variable influencing factors such as autonomic nervous system regulation. Nevertheless, the clinical consequence of missing or falsely diagnosing a potentially lethal disease as an inherited arrhythmia syndrome such as long QT syndrome demands a distinct differentiation of pathological phenomena and normal variants. Especially in children, data on these phenomena are scarce. In addition, autonomic regulation is more variable in children than that in adults, and hormonal influences during puberty add other influencing factors. While recent studies added some data on normal values of cardiac repolarization in healthy children, the influence of conditions such as obesity or extensive physical fitness remains less defined.^[6-14]

The aim of this study is, therefore, to compare the parameters of cardiac repolarization in children at the extremes of physical fitness and to increase knowledge about cardiac repolarization in these subgroups of pediatric patients that are vastly affected by deviations of autonomic regulation.

METHODS

Study population

After analysis of the LIFE Child Study at the Leipzig Research Centre for Civilization Diseases (LIFE) from 2011 to 2014 database and the database from the Leipzig Institute for Applied Training Sciences, a total of 426 pediatric volunteers (84 lean, healthy controls; 130 obese healthy pediatric volunteers; and 212 elite athletes) were enrolled in the study.^[15] All participants underwent a thorough cardiac evaluation including medical history, physical examination, anthropometric evaluation, 12-lead ECG, echocardiography, and blood samples. Written consent of the parents was obtained with inclusion into the study.

Definitions

Childhood obesity was classified as adjusted body mass index (BMI) >1.28 standard deviation score (SDS), and

children with an adjusted BMI ≤ 1.28 were classified as lean.

Children and adolescents were considered as elite athletes, if being either part of a German youth national team or equivalent in their specific sports.

Age adjustment

All anthropometric parameters were adjusted for age and sex, based on the national reference values of German children from Kromeyer-Hauschild *et al.* (2001) and accordingly were presented in SDS. Participants were stratified into overweight/obese and lean children applying an adjusted BMI 1.28/1.88 SDS as a cutoff according to the current German guidelines.^[16]

Electrocardiogram analysis

For each child, a 12-lead ECG was recorded using an electrocardiograph (General Electrics, MAC 5000). The frequency response of this recorder is flat to 150 Hz. A paper speed of 50 mm/s and amplitude of 10 mm/mV was used. The same technician recorded the ECGs throughout the study.

All ECGs were anonymized by pseudonymization and analyzed by two physicians with extensive experience in the analysis of pediatric ECGs. Both physicians had no access to demographic or clinical data.

Parameters of cardiac repolarization were measured in leads II and V5 of a standard 12-lead ECG. All parameters were determined from the measurements of at least six consecutive beats in lead II and V5. Minimum, maximum, and average values were acquired. Tpeak to end (TPE) was measured in milliseconds from the peak of the T wave to the end of the T wave. The end of the T wave was defined as the return of the descending limb to the TP baseline when not followed by a U wave or if distinct from following the U wave. If there was a terminal low-amplitude signal interrupting the terminal portion of the T wave, the downslope of the T wave was extended by drawing a tangent to the steepest proportion of the downslope until it crosses the TP segment to determine the end of the T wave. The QT interval was then corrected for heart rate using the Bazett's formula (QT/\sqrt{RR}) (QTc) and in addition the Hodges's formula in patients with a resting heart rate below 50 bpm as suggested in literature.^[17-19] The RR interval was measured in seconds and taken as the immediate RR interval preceding the TPE interval from the same lead, in which the TPE interval was measured. QT dispersion was calculated as the difference between maximum and minimum QT interval within the measurement of six consecutive beats.

Statistical analysis

Statistical analysis was carried out with SPSS 21.0 software (SPSS Statistics, IBM, Ehningen, Germany). Continuous data were assessed for normality, and the Student's *t*-test

was used for normally distributed data. To characterize the influence between continuous variables, bivariate correlation was used, and the Pearson’s correlation coefficient (*r*) was reported. For this study, α was set at 0.05; thus, $P < 0.05$ (two-sided) was considered statistically significant.

RESULTS

Patients’ characteristics

Table 1 shows the patients’ basic characteristics of the stratified groups: lean, overweight/obese, and elite athletes including anthropometric data. All patients had structurally normal hearts in echocardiography.

Parameters of cardiac repolarization

Table 2 shows the parameters of cardiac repolarization of the three groups: lean, overweight/obese, and athletes. Although there were significant differences between the groups, no pathological findings were recorded. First of all, there is a trend toward shorter corrected QT intervals in athletes despite insignificant differences in average absolute QT intervals. This is also true if a correction of the QT interval is performed using the Hodges’s formula to avoid bias due to a tendency to lower heart rates in

athletes. Second, athletes showed statistically significant shorter TPE ratios compared to the other groups, albeit clinically probably not leading to a significant difference.

Comparison of athletes from strength and power sports versus athletes from endurance sports

Table 3 shows the comparison of sportsmen from strength and power sports versus endurance sports. As expected, the different physiognomy can be seen in the anthropometric data. Comparing the absolute QT intervals of both groups, endurance-trained athletes tend to have significantly longer QT intervals. When the QT intervals are corrected for heart rate, this effect persisted only in the corrections using the Hodges’s formula and was not found to be statistically significant when using the common Bazett’s formula. There were no statistically significant differences in the parameters of cardiac inhomogeneity (TPE) between the groups.

DISCUSSION

The current study evaluated the influence of obesity and extreme physical fitness on parameters of cardiac repolarization in children. The results of the current study demonstrate one major finding. In this pool of otherwise healthy children, there were no pathological phenomena of

Table 1: Patient’s characteristics

| | Lean control | Obese | Athlete | P |
|-------------|----------------------|--------------------|---------------------|--------|
| Age (years) | 10.6 (6, 4; 17, 3) | 12.3 (5, 5; 19, 1) | 15 (9, 6; 17, 9) | <0.001 |
| Gender | | | | |
| Male | 40 | 70 | 125 | 0.197 |
| Female | 44 | 60 | 87 | |
| Height (cm) | 146 (116; 180) | 157 (120; 188) | 167 (129; 203) | <0.001 |
| Height ADJ | 0.05 (-2.17; 1.88) | 0.72 (-2.03; 3.84) | 0.17 (-2.13; 3.45) | <0.001 |
| Weight (kg) | 36.2 (19; 71.7) | 70.33 (31.7; 136) | 56.85 (27.1; 121.8) | <0.001 |
| Weight ADJ | -0.049 (-2.59; 1.46) | 2.31 (0.83; 4.53) | 0.18 (-2.55; 3.31) | <0.001 |
| BMI | 16.91 (10.6; 22.6) | 27.81 (20.1; 43.7) | 20.05 (15.1; 38.4) | <0.001 |
| BMI ADJ | -0.23 (-5.86; 1.26) | 2.28 (1.41; 3.85) | 0.1 (-2.43; 3.06) | <0.001 |

All numbers are depicted as median and range. BMI: Body mass index, ADJ: Adjusted to age

Table 2: Parameters of cardiac repolarization

| | Lean control | Obese | Athlete | P |
|----------------------|----------------|----------------|----------------|--------|
| HR (bpm) | 73 (56; 103) | 78 (48; 118) | 60 (42; 94) | <0.001 |
| PR interval | 128 (87; 196) | 132 (57; 213) | 140 (80; 190) | 0.001 |
| QRS width II | 80 (58; 107) | 82 (57; 108) | 100 (80; 130) | <0.001 |
| QT max II | 370 (310; 410) | 360 (300; 460) | 370 (285; 455) | <0.001 |
| QT avrg II | 360 (305; 405) | 355 (295; 455) | 370 (285; 455) | 0.476 |
| QT max V5 | 360 (290; 400) | 360 (300; 450) | 380 (300; 470) | <0.001 |
| QT avrg V5 | 355 (285; 395) | 352 (295; 440) | 375 (295; 455) | <0.001 |
| TPE II | 70 (50; 100) | 70 (50; 100) | 60 (40; 80) | <0.001 |
| TPE V5 | 60 (50; 80) | 70 (50; 100) | 60 (40; 90) | <0.001 |
| QTc max II (Bazett) | 410 (350; 425) | 410 (340; 455) | 380 (290; 430) | <0.001 |
| QTc avrg II (Bazett) | 400 (350; 410) | 400 (340; 440) | 370 (290; 420) | <0.001 |
| QTc max II (Hodges) | // | // | 380 (330; 440) | // |
| QTc avrg II (Hodges) | // | // | 370 (325; 430) | // |
| QTc max V5 (Bazett) | 395 (355; 425) | 405 (340; 460) | 385 (300; 450) | <0.001 |
| QTc avrg V5 (Bazett) | 390 (350; 410) | 400 (330; 450) | 370 (300; 435) | <0.001 |
| QTc max V5 (Hodges) | // | // | 380 (325; 445) | // |
| QTc avrg V5 (Hodges) | // | // | 375 (320; 435) | // |

All numbers are depicted as median and range. The unit of the HR is depicted in beats per minute, all other parameters in milliseconds. HR: Heart rate, TPE: Tpeak to end, //: No value available

Table 3: Parameters of cardiac repolarization in athletes - endurance versus strength and power sports

| | Endurance | Strength | P |
|----------------------|--------------------|---------------------|--------|
| Age (years) | 15.01 (9.6; 17.2) | 15.15 (9.9; 17.9) | 0.461 |
| Gender | | | |
| Male | 64 | 61 | 0.93 |
| Female | 44 | 43 | |
| Height (cm) | 171 (145; 203) | 165 (129; 199) | 0.001 |
| Height ADJ | 0.68 (-1.64; 3.45) | -0.18 (-2.13; 2.74) | <0.001 |
| Weight (kg) | 58.8 (34.5; 103.3) | 55.2 (27.1; 121.8) | 0.714 |
| Weight ADJ | 0.29 (-1.78; 2.52) | 0.01 (-2.55; 3.31) | 0.088 |
| Weekly training h | 15 (3; 32) | 10 (0; 30) | <0.001 |
| HR (bpm) | 59 (42; 76) | 65 (43; 94) | <0.001 |
| PR interval II | 140 (90; 180) | 140 (80; 190) | 0.017 |
| QRS width II | 375 (310; 440) | 100 (80; 120) | 0.196 |
| QT max II | 390 (320; 460) | 360 (290; 460) | <0.001 |
| QT avrg II | 380 (315; 450) | 355 (285; 455) | <0.001 |
| QT max V5 | 390 (320; 470) | 370 (300; 460) | <0.001 |
| QT avrg V5 | 380 (315; 455) | 360 (295; 455) | <0.001 |
| TPE II | 60 (40; 80) | 60 (40; 80) | 0.044 |
| TPE V5 | 60 (40; 90) | 60 (40; 80) | 0.330 |
| QTc max II (Bazett) | 380 (315; 430) | 380 (290; 420) | 0.397 |
| QTc avrg II (Bazett) | 370 (310; 420) | 370 (290; 410) | 0.214 |
| QTc max II (Hodges) | 385 (335; 440) | 370 (330; 430) | <0.001 |
| QTc avrg II (Hodges) | 380 (335; 430) | 365 (325; 425) | <0.001 |
| QTc max V5 (Bazett) | 385 (325; 450) | 390 (300; 435) | 0.578 |
| QTc avrg V5 (Bazett) | 375 (320; 435) | 370 (300; 425) | 0.361 |
| QTc max V5 (Hodges) | 385 (340; 445) | 375 (325; 435) | <0.001 |
| QTc avrg V5 (Hodges) | 380 (340; 435) | 370 (320; 430) | <0.001 |

All numbers are depicted as median and range. The units of the heart rate, age, weight, and height are presented in brackets, all other parameters in milliseconds. HR: Heart rate, TPE: Tpeak to end, ADJ: Adjusted to age

cardiac repolarization, neither in the group of elite athletes nor in the group of obese patients or healthy controls.

Yet, data analysis showed significant differences from the literature in adults.

First of all, the data showed no lengthening of the QT or corrected QT interval in elite athletes compared to lean probands. This finding stands in contrast to the findings in literature concerning adults.^[20-24] To rule out the Bazett's formula for the correction of QT interval in the group of elite athletes with generally lower heart rates as a possible bias, we compared the results to a correction with the Hodges's formula that might be less biased by lower heart rates. The results were independent from the formula used for the correction of QT interval to the heart rate. As reported by D'Ascenzi *et al.*, the reason for this incoherence of pediatric data with data from adult patient collectives may be because puberty seems to be a strong influencing factor regarding QT interval prolongation. This might explain why these changes are probably not yet reported in adolescents, but may develop in adult athletes. In addition, it may be speculated that physical adaption to extensive sports is not fully developed in adolescence, and the full effects of physical adaption will only be present in adulthood.

Second, the overall data demonstrated the longest corrected QT intervals in obese patients, as has been demonstrated in studies before.^[13,14] Those studies reported an increased parasympathetic tone in obese patients and the hormonal activity of body fat deposits as causative mechanisms. Particularly, the hormonal activity

of body fat leads to elevated estrogen levels which are assumed to promote QT interval prolongation. As this finding might not be unexpected, it gives some hints to the etiology of QT interval prolongation in children. Both athletes and obese children are supposed to have a rather increased parasympathetic tone, often referred to as a possible influencing factor for QT interval prolongation. Nevertheless, the presented cohort showed relatively long QT intervals in the obese and relatively short QT intervals in the athletes, making the basic parasympathetic tone a rather minor influencing factor in children.

When looking at the parameters of cardiac electrical inhomogeneity, there are only minor differences between the groups. In particular, there were no statistically significant differences between athletes and lean probands. Taking a closer look at the group of obese patients, statistically significant changes in the parameters of cardiac electrical inhomogeneity (TPE interval) could be demonstrated. Primarily implying a possibly elevated risk for cardiac arrhythmia, the actual data, although statistically significant, demonstrate only differences of a few milliseconds that are rather unlikely to represent a parameter of clinical significance or to conclude on an elevated arrhythmogenic risk. Yet, some more data are needed for an appropriate evaluation of this topic.

CONCLUSION

This study suggests that neither obesity nor extensive sports seems to result in pathological cardiac

repolarization parameters in healthy children. Therefore, pathology has to be assumed if abnormal repolarization parameters are seen and might not be simply attributed to the child's habitus or an excellent level of fitness.

Limitations

The main limitation of this study is the difference in median ages between the groups. As the parameters of cardiac repolarization might be influenced by puberty, this might have an effect on the reported data. Yet, with patients of the lean control group being the youngest, a possible age-related effect on parameters of cardiac repolarization should only be important if pathological parameters would have been found in the older participants of the two other groups.

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Conflicts of interest

There are no conflicts of interest.








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2.4. Orofacial conditions and oral health behavior of young athletes: A comparison of amateur and competitive sports.

Die gesamte vorliegende Habilitationsschrift fokussiert ihren Schwerpunkt auf die sportmedizinische Vorsorgeuntersuchung in den Bereichen bisher weniger beachteter Kollektive (neue olympische Sportarten, Kinder- und jugendliche Elitesportler) oder bisher sportmedizinisch wenig beachteter vorsorgerelevanter Organsysteme. Diese vorgelegte Arbeit stellt somit eine Schnittmenge aus dem bereits oben genannte Sportlerkollektiv (junge Spitzensportler) und einer „jungen“ sportmedizinischen Teildisziplin der Sportzahnmedizin und deren Implikationen auf die körperliche Leistungsfähigkeit und Gesunderhaltung bei Hochleistungssportlern dar. Ähnlich den bereits vorangegangenen Arbeiten wurde auch hier zunächst versucht, den bisher nicht oder wenig bekannten Ist-Zustand (hier der Zahnstatus und das Zahngesundheitsverhalten) bei jugendlichen Hochleistungssportlern im Vergleich zu Amateursportlern herauszustellen. Zahlreiche Überlegungen ließen postulieren, dass sich in dem untersuchten Kollektiv der Spitzensportler Unterschiede im Zahnstatus feststellen lassen. Die physiologisch, belastungsbedingten Veränderungen in der Mundhöhle, veränderte Essgewohnheiten, vermehrte Aufnahme säurehaltiger Sportgetränke, verstärkte intracorporale Flüssigkeitsschwankungen und viele andere Ursachen, die hypothetisch einen Einfluss auf die Zahngesundheit haben können, gaben den Anlass in dem speziellen Kollektiv der jungen Hochleistungssportler den oralen Gesundheitsstatus genauer zu untersuchen. Um zudem den Einfluss des individuellen, oralen Gesundheitsverhaltens zu untersuchen, welches sich aufgrund eines bei Spitzensportlern möglicherweise gesundheitsbewussteren Lebensstils von dem bei Amateursportlern unterscheiden könnte, wurde dieser Aspekt zusätzlich beleuchtet. Dabei zeigte sich, dass insbesondere im Bereich der gingivalen Inflammation sowohl bei Hochleistungssportlern als auch bei Amateursportlern Zeichen eines Behandlungsbedarfs bestanden. Darüber hinaus fanden sich bei den Hochleistungssportlern vermehrte Auffälligkeiten kariöser, periodontaler und funktioneller Art bei im Wesentlichen gleichem oralen Gesundheitsverhalten. Somit ließ sich herausstellen, dass bei Hochleistungssportlern ein erhöhter Aufwand zur oralen Gesundheitsvorsorge gerechtfertigt scheint.

Orofacial conditions and oral health behavior of young athletes: A comparison of amateur and competitive sports

Cordula Leonie Merle¹  | Lisa Richter¹ | Nadia Challakh¹ | Rainer Haak¹  | Gerhard Schmalz¹  | Ian Needleman^{2,3}  | Bernd Wolfarth^{4,5,6}  | Dirk Ziebolz¹  | Jan Wüstenfeld^{4,6} 

¹Department of Cariology, Endodontology and Periodontology, University of Leipzig, Leipzig, Germany

²Centre for Oral Health and Performance, UCL Eastman Dental Institute, London, UK

³UK IOC Research Centre for Prevention of Injury and Protection of Athlete Health, London, UK

⁴Department of Sports Medicine, Humboldt-University, Charité University Medicine, Berlin, Germany

⁵Institute of Sports Science, Humboldt University, Berlin, Germany

⁶Department of Sports Medicine, Institute for Applied Scientific Training, Leipzig, Germany

Correspondence

Cordula Leonie Merle, Department of Cariology, Endodontology and Periodontology, University Medical Center Leipzig, Liebigstr. 12, Leipzig D-04103, Germany.
Email: cordulaleonie.merle@medizin.uni-leipzig.de

Funding information

Leipzig University

Purpose: This retrospective cross-sectional study aimed to evaluate oral health status (dental, periodontal, and functional) and oral health behavior in young German athletes including the comparison of competitive (CA) and amateur sports (AA).

Methods: Data of CA (German national teams, perspective, and youth squads) and AA aged between 18 and 30 years with an available oral examination in 2019 were included. Clinical examination: caries experience (DMF-T), non-cariou wear (erosion, BEWE), partially erupted wisdom teeth, gingival inflammation (PBI), plaque index, periodontal screening (PSI), and temporomandibular dysfunction (TMD) screening. Questionnaires: oral health behavior and periodontal symptoms.

Results: 88 CA (w = 51%, 20.6 ± 3.5 years) of endurance sports and 57 AA (w = 51%, 22.2 ± 2.1 years) were included. DMF-T was comparable (CA: 2.7 ± 2.2, AA: 2.3 ± 2.2; *p* = 0.275) with more D-T in CA (0.6 ± 1.0) than AA (0.3 ± 0.7; *p* = 0.046; caries prevalence: CA: 34%, AA: 19%; *p* = 0.06). Both groups had low severity of erosion (BEWE about 3.5). CA had more positive TMD screenings (43% vs. 25%; *p* = 0.014). In both groups, all athletes showed signs of gingival inflammation, but on average of low severity (PBI <1). More CA needed complex periodontal treatment than AA (maximum PSI = 3 in 40% vs. 12%; *p* < 0.001). Oral health behavior was comparable (daily tooth brushing; regular dental check-ups in >70%).

Conclusions: Young German athletes (CA and AA) generally showed signs of gingival inflammation and needed to improve their oral health behavior. CA showed slightly increased oral findings (more D-T, periodontal and TMD screening findings) than AA, but similar oral health behavior. This may imply an increased dental care need in competitive sports.

Cordula Leonie Merle and Lisa Richter should be considered joint first author.

Dirk Ziebolz and Jan Wüstenfeld should be considered joint senior author.

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KEYWORDS

athletes, biathlon, oral health, oral hygiene, prevention, sports dentistry

1 | INTRODUCTION

In competitive sport, protecting athletes' health is of primary importance to support wellbeing and performance.¹ Illness and pain can lead to interruption of training or even to missing important sports events. Furthermore, each small impact could affect the performance. Such possible influence factor is the oral health status: Between 17%² and 30%³ of athletes in various sports report pain in the oral region with 4%⁴ to 8%³ declaring actual discomfort. Three percent of all athletes in the 2004 Olympic Games visited the dental clinic in Olympic Village.⁵ First studies even revealed a possible link between oral health status and increased risk for muscle injuries.^{2,6} Thus, orofacial health should be of sports medical and athletes' particular interest as by now several international associations have stated, for example, the International Olympic Committee,⁷ the Fédération Dentaire Internationale⁸ and the Union of European Football Associations.⁹

It is well known that more than one-quarter of contact and combat athletes experience dental injuries^{10,11} and that mouthguards can be effective for their prevention.¹² However, as previous studies have reported, athletes commonly have oral health problems:¹³ About 46% have dental caries¹⁴ with a higher prevalence than controls.^{15,16} Gingival inflammation is very common with 58% to 85%^{2-4,15,17,18} and even periodontitis is reported in 5% to 41%.^{3,4,17-19} Erosion affects nearly every second athlete.²⁰ Moreover, between 3% and 18% of athletes declared that oral health affected their training or performance.^{3,4,17,18}

Numerous potential risk factors for oral diseases in competitive sports have been discussed, for example, special nutrition habits (usually high carbonate, partially as a gel),²¹ mental stress,¹⁵ changes in saliva,^{16,22} and oral microbiome changes.¹⁵ Overall, oral health behaviors of athletes might also compromise oral health with irregular or even no dental check-up visits,^{2,4,15,18} deficits in daily toothbrushing and missing interdental cleaning.²¹

While there have been recommendations to implement oral health screening in athletes,^{23,24} this is not yet standard and the number of epidemiological studies is low. The available studies were conducted in different healthcare systems with different organization types of competitive sports. The few existing studies with comparison to control groups^{15,16} as well as the comparison to population-based data^{3,17} indicate an increased prevalence of oral diseases for athletes.

The current study aimed to investigate the orofacial health status in German sports within a retrospective evaluation. It was hypothesized that young elite athletes would show worse oral health findings and behavior than young recreational athletes.

2 | METHODS

2.1 | Study design

This retrospective monocentric cross-sectional study was reviewed and approved by the Ethics Committee of the medical faculty of Leipzig University, Germany (No. 091/20-ek). All athletes were informed verbally and in writing about the scientific use of their clinical data and gave their general written informed consent (independent of this study). The recommendations for strengthening the reporting of cross-sectional studies (STROBE) were considered.²⁵

2.2 | Participants

Competitive athletes (CA) were compared to amateur athletes (AA). Inclusion criteria were male and female athletes (AA and CA) aged between 18 and 30 years that provided agreement to scientific use of data, with performed standardized sport dental examination in the Institute for Applied Scientific Training (IAT) (CA) or dental clinic (AA) in 2019. CA were defined as members of the German national teams and perspective and youth squads. AA were defined as being sporting active each week, typically organized in sport clubs, but without regularly participating in competitions. Athletes with missing signed agreement to data use for scientific evaluation or incomplete clinical examination were excluded.

Data of CA were collected in a collaboration between the Dept. of Cariology, Endodontology and Periodontology, University of Leipzig and the Institute for Applied Scientific Training (IAT), Leipzig. The dental examinations were performed as a supplement to the annual sports-medical performance diagnostics between May and December 2019. AA presented themselves for the specialized sports dentistry consultation at the Dept. of Cariology, Endodontology and Periodontology, University of Leipzig. This service for dental check-up had been initiated in September 2019 and had been promoted at the

faculty of sports science, University of Leipzig, and to several local sport clubs.

2.3 | Data collection

Data were extracted from participants' dental records. General characteristics of the CA group were obtained from the sport medicine records. Both groups had been examined by one single skilled dentist who was trained in the performed dental parameters and screening instruments. For CA, all examinations were conducted using a headlight on an examination couch in the IAT. AA were examined in a dentist's chair in the dental clinic.

For CA and AA, the same examination protocol had been completed: It included dental findings, oral hygiene indices, periodontal and functional screening. Questionnaires about oral health behavior and periodontal symptoms were administered to all participants.

2.3.1 | Orofacial examination

Dental findings: Dental status was assessed with mirror and probe. Decayed, missing and filled teeth were quantified by the DMF-T.²⁶ All teeth with a clearly visible or suspected cavitation reaching the dentine layer were rated as decayed (D-T), teeth extracted due to caries (no agenesis or orthodontic indication upon request) were rated as missing (M-T) and teeth with restorations as filled (F-T). Non-carious wear due to erosion, abrasion, attrition or combination was evaluated using the basic erosive wear examination (BEWE).²⁷ The highest dental wear observed in each sextant was scored either with 0 (no tooth wear), 1 (initial loss of surface texture), 2 (distinct defect, hard tissue loss <50% of the surface area, dentin often is involved), or 3 (hard tissue loss ≥50% of the surface area, dentin often is involved). The cumulative BEWE score of all sextants was analyzed. Risk level and severity of wear were estimated as follows: none (0 – 2), low (3 – 8), medium (9 – 13), and high (14 and more).

Oral hygiene: Plaque accumulation was assessed by the plaque index (PI) by Silness and Loe.²⁸ All teeth were rated from vestibular by visual inspection and probe. Plaque was quantified with the scores 0 (no plaque), 1 (plaque only detectable with the probe), 2 (moderate visible plaque at gingival margin), and 3 (extensive plaque filling interdental triangles). Gingival inflammation was assessed by the papillary bleeding index (PBI), whereby the marginal gingival sulcus was spread out with a periodontal probe (PCP/UNC 15, Hu-Friedy, Chicago, IL, USA) and bleeding was recorded after 15 seconds (0: no bleeding; 1: one bleeding point; 2: fine line of blood; 3: blood

fills interdental triangle; 4: profuse bleeding).²⁹ Plaque and PBI index were calculated per patient by dividing their total sum by the number of teeth/papillae.

Periodontal screening: Periodontal situation was examined using the Periodontal Screening Index (periodontal probe: PCP/UNC 15, Hu-Friedy, Chicago, IL, USA):^{30,31} Score 0 to 2 has probing depths less than 3.5 mm. Score 0 shows no bleeding, no calculus, score 1 bleeding on probing and score 2 calculus. A score of 3 or 4 indicates increased probing depths (3: pocket depth 3.5 – 5.5 mm; 4: pocket depth >5.5 mm). Participants with at least one sextant scored 3 or 4 were stated as having more complex periodontal treatment need (including detailed periodontal assessment and professional debridement of periodontal pockets ≥4 mm).³²

Wisdom teeth: Third molars were not included in indices or screenings unless they were present more anterior. However, the status of wisdom teeth was recorded per participant (presence, absence, or partially erupted).

TMD screening: Functional screening was performed from pain due to muscle palpation (M. masseter, M. temporalis, Venter anterior M. digastricus), mouth opening capability and movement, signs of traumatic occlusion, joint, and occlusal sounds.³³ Patients with two or more positive findings were classified as positive TMD screening and should be examined in more detail by functional analysis.

2.3.2 | Questionnaires

Standardized questionnaires about oral health behavior and periodontal symptoms were used as established in various previous studies by this working group.^{34–36} All athletes were asked to complete them self-administered on-site. Questions were asked about dentist visits (control or complaint-oriented check-ups, regular professional teeth cleaning), the importance of oral health, oral hygiene behavior (frequency of toothbrushing, interdental hygiene, use of fluoride gels), and received instructions about oral hygiene. Questioned periodontal symptoms concerned the gums (swelling, pain, sensitivity, and bleeding), sensitive or mobile teeth, changes in tooth position or bite, bad breath or taste, toothache, and periodontal therapy in the past.

2.4 | Statistical analysis

Data were collected in a Microsoft Excel spreadsheet (Microsoft, Redmond, WA, USA). Statistical analysis was performed with SPSS Statistics for Windows (version 23.0, IBM Corp. Armonk, NY, USA). As no normal-distribution was given (tested with Kolmogorov–Smirnov

test), Mann–Whitney U test and Fisher's exact test were used. The significance level was defined with $p < 0.05$. Subgroup analyses were carried out with Pearson's chi-squared statistics for all disciplines with more than ten participants. Per group, potential associations between oral findings (D-T, maximum PSI ≥ 3) and oral health behavior (interdental cleaning, control-oriented dental check-ups, professional dental cleaning) were analyzed.

3 | RESULTS

3.1 | Patients

Patient records of 88 CA ($w = 51\%$, 20.6 ± 3.5 years) and 57 AA ($w = 51\%$, 22.2 ± 2.1) were included for retrospective evaluation. Table 1 shows the characteristics of the athletes in both groups with subdivision to disciplines (CA), respectively, discipline group (AA). In total, 100 CA had been invited to present themselves for a dental check-up in 2019; 88% attended.

3.2 | Orofacial findings

The mean DMF-T of the groups showed no significant difference (CA: 2.7 ± 2.2 vs. AA: 2.3 ± 2.2 ; $p = 0.275$). 34%

TABLE 1 Characteristics of the study participants

| | <i>n</i> | Age in years [mv \pm SD] | Gender female [<i>n</i> (%)] |
|-----------------------------|----------|-------------------------------|----------------------------------|
| Competitive athletes | | | |
| Total | 88 | 20.6 ± 3.5 | 45 (51.1) |
| Running | 39 | 22.7 ± 3.4 | 13 (33.3) |
| Biathlon | 27 | 18.0 ± 1.5 | 15 (55.6) |
| Cross-country skiing | 10 | 18.4 ± 0.7 | 5 (50.0) |
| Rowing | 8 | 23.8 ± 1.2 | 8 (100.0) |
| Triathlon | 4 | 17.0 ± 2.0 | 4 (100.0) |
| Amateur athletes | | | |
| Total | 57 | 22.2 ± 2.1 | 29 (50.9) |
| Sport students ^a | 23 | 21.8 ± 2.1 | 10 (43.5) |
| Endurance | 15 | 23.3 ± 1.4 | 8 (53.3) |
| Team sports | 9 | 21.8 ± 2.6 | 3 (33.3) |
| Power | 7 | 22.4 ± 2.1 | 5 (71.4) |
| Other | 3 | 21.0 ± 1.7 | 3 (66.6) |
| <i>p</i> -Value | | <0.001 | 1.000 |

Abbreviations: mv, mean value; n, number of participants; SD: standard deviation

^aSport students at the university with several disciplines.

of CA and 19% of AA had untreated caries ($p = 0.06$). Nevertheless, CA had in average statistically significantly more untreated decayed teeth (0.6 ± 1.0) than AA (0.3 ± 0.7 ; $p = 0.046$). Both groups showed erosive tooth wear of low severity (BEWE about 3.5) and mean plaque levels below 1. All athletes showed signs of gingival inflammation, but on average of low severity (PBI <1). In CA, a higher prevalence of increased probing depths indicating signs of periodontitis (maximum PSI score 3 in 40% vs 12%; $p < 0.001$) was revealed. A higher prevalence of signs of TMD was stated for CA (43%) than AA (25%; $p = 0.014$). More detailed further data are shown in Table 2.

The disciplines running and biathlon were analyzed in more detail by subgroup analysis (Table 3): Runners (R) showed compared to biathletes (B) an increased mean of filled teeth (R: 2.4 ± 1.9 , B: 1.3 ± 1.5 ; $p = 0.017$) as well as a higher mean of a cumulative score of non-carious wear (R: 4.3 ± 2.8 , B: 3.1 ± 2.2 ; $p = 0.036$). PI was statistically significantly higher in biathletes than in runners (B: 1.0 ± 0.4 , R: 0.7 ± 0.4 ; $p = 0.003$). For all disciplines of CA separately, oral health findings are presented in Table S1.

3.3 | Oral health behavior and periodontal symptoms

74 CA and 57 AA completed the questionnaires about oral health behavior and periodontal symptoms. The results are presented in Figure 1. For all participants, tooth brushing was reported to be a daily routine, and nearly all participants rated a clean/healthy dentition as important or very important. However, less than 80% of the athletes (CA: 75.7%, AA: 71.9%; $p = 0.69$) reported regular dental check-ups. Only about a half of CA and a third of AA ($p = 0.076$) stated to go regularly for professional teeth cleaning. Less CA than AA (CA: 79.5%, AA: 93.0%; $p = 0.044$) reported having received oral hygiene instructions. Oral health behavior of biathletes and runners did not differ significantly. No group differences in reported periodontal symptoms could be found. More than a quarter of the athletes stated bleeding or sensitive gums, or sensitive teeth.

3.4 | Associations of clinical findings and oral health behavior

For both groups, no associations between decayed teeth and oral health behavior were found (Table S2). Less CA with signs of periodontitis stated regular professional teeth cleaning than those without having increased probing depths (25% vs 57%; $p = 0.009$). In AA, none associations

TABLE 2 Oral health of competitive and amateur athletes

| | Competitive athletes (n = 88) | Amateur athletes (n = 57) | p-value |
|----------------------------------------|-------------------------------|---------------------------|------------------|
| Dental findings [mv ± SD] | | | |
| DMF-T | 2.7 ± 2.2 | 2.3 ± 2.2 | 0.275 |
| D-T | 0.6 ± 1.0 | 0.3 ± 0.7 | 0.046 |
| M-T | 0.1 ± 0.5 | 0.0 ± 0.1 | 0.818 |
| F-T | 2.0 ± 1.9 | 2.0 ± 2.1 | 0.933 |
| BEWE | 3.6 ± 2.5 | 3.4 ± 1.9 | 0.859 |
| Oral hygiene [mv ± SD] | | | |
| Plaque Index (PI) | 0.8 ± 0.4 | 0.6 ± 0.3 | <0.001 |
| Gingival Inflammation (PBI) | 0.5 ± 0.3 | 0.5 ± 0.2 | 0.589 |
| Periodontal screening [mv ± SD] | | | |
| max. PSI = 1 | 3 (3.4) | 6 (10.5) | 0.155 |
| max. PSI = 2 | 50 (56.8) | 44 (77.2) | 0.013 |
| max. PSI = 3 | 35 (39.8) | 7 (12.3) | <0.001 |
| Partially erupted wisdom teeth [n (%)] | 13 (14.8) | 14 (24.6) | 0.190 |
| Positive TMD screening [n (%)] | 40 (45.5) | 14 (24.6) | 0.014 |

Abbreviations: BEWE, basic erosive wear examination; DMF-T, decayed-, missing- and filled-teeth index; D-T, decayed teeth; F-T, filled teeth; M-T, missing teeth; mv, mean value; PBI, papillary bleeding index; PSI, periodontal screening index; SD, standard deviation; TMD, temporomandibular disorder.

Bold marks significant differences ($p < 0.05$).

between clinical findings and self-reported oral health behavior were found.

4 | DISCUSSION

4.1 | Summary of the main results

In general, young athletes (AA and CA) showed a high prevalence of signs of periodontal disease, primarily gingival inflammation. Comparing CA and AA, CA showed statistically significantly more decayed teeth, a higher prevalence of positive TMD screening findings, elevated periodontal treatment need (increased probing depths). Both groups reported similar oral health behavior with potential for improvement.

4.2 | Interpretation compared to the available literature

For CA, the present study revealed less caries¹⁴ and erosion¹⁶ and similar oral health behavior,^{2,15,18,21} but more periodontal treatment need than previous studies.¹³ About one-third of CA had untreated carious lesions, and therefore, less than world-wide estimated (prevalence of caries in athletes: 46%).¹⁴ Both groups showed lower DMF-T values than other studies on elite athletes in industrialized countries (4.8 to 5.7; mean age between 21

and 27 years).^{2,15,18} Higher values observed in German triathletes (DMF-T = 9.7; medium age: 37 years)¹⁶ could be explained by the high age-dependence of this parameter.

Erosion has been researched as one key point in athletes' oral health, and its general prevalence in these individuals is estimated as 47%.²⁰ The present study determined non-carious wear (tooth surface loss) of low severity in both groups (BEWE about 3.5), comparable to another study with competitive athletes (mean: 3.02, medium age: 26 years).¹⁸ Potentially, with increasing age, higher values and group differences could become apparent as erosion is a long-term effect, for example, by intake of acid sport drinks. So, another German study described a medium level of erosive wear for triathletes (mean age: 37 years; mean BEWE: 9.6, control group: low level, 7.3).¹⁶

In both groups, mean PI and PBI were generally low (<1; Table 2), also in comparison to other studies which employed different indices.^{2,15} However, in CA, the prevalence of significant signs of periodontal diseases (40%) was higher than the values from previously reported studies of 5% to 21%^{3,4,17,18} and higher compared with AA at 12%. In this young age, moderately elevated periodontal pockets (none above 5.5 mm) could be a sign of gingivitis or initial periodontitis. Nevertheless, the data report a significant treatment need to manage periodontal health. A detailed periodontal examination in footballers (mean age: 28 years) reported a similar prevalence of periodontitis (41%), with the diagnosis of a moderate or severe stage in only two individuals (9%).¹⁹

TABLE 3 Subgroup analysis of oral health in competitive athletes for the disciplines running and biathlon

| | Running (n = 39) | Biathlon (n = 27) | p-value |
|----------------------------------------|---------------------|----------------------|--------------|
| Dental findings [mv ± SD] | | | |
| DMF-T | 2.8 ± 2.2 | 2.0 ± 2.0 | 0.162 |
| D-T | 0.5 ± 0.9 | 0.7 ± 1.2 | 0.418 |
| M-T | 0.0 ± 0.0 | 0.0 ± 0.0 | n. a. |
| F-T | 2.4 ± 1.9 | 1.3 ± 1.5 | 0.017 |
| BEWE | 4.3 ± 2.8 | 3.1 ± 2.2 | 0.036 |
| Oral hygiene [mv ± SD] | | | |
| Plaque Index (PI) | 0.7 ± 0.4 | 1.0 ± 0.4 | 0.003 |
| Gingival Inflammation (PBI) | 0.5 ± 0.3 | 0.6 ± 0.3 | 0.193 |
| Periodontal screening [mv ± SD] | | | |
| max. PSI = 3 | 11 (28.2) | 15 (55.6) | 0.214 |
| Partially erupted wisdom teeth [n (%)] | 2 (5.1) | 7 (25.9) | 0.151 |
| Positive TMD screening [n (%)] | 20 (51.3) | 12 (44.4) | 0.312 |

Abbreviations: BEWE, basic erosive wear examination; DMF-T, decayed-, missing- and filled-teeth index; D-T, decayed teeth; F-T, filled teeth; M-T, missing teeth; mv, mean value; n. a., not applicable; PBI, papillary bleeding index; PSI, periodontal screening index; SD, standard deviation; TMD, temporomandibular disorder.

Bold marks significant differences ($p < 0.05$).

Almost 15% of the CA had partially erupted wisdom teeth. The status of wisdom teeth has sports-medical relevance as they present a risk of developing symptomatic pericoronitis leading to severe pain and difficulties in both oral function and lifestyle,³⁷ and therefore, potential treatment need during competitions.^{4,38} A study on Dutch elite athletes described higher recommendation rates of wisdom tooth removal (22%).¹⁸ Furthermore, the proportion of AA with partially erupted wisdom teeth was higher (about 25%) than in CA. Consequently, this point seems already to be considered in athletes' care in Germany.

More than 40% of the CA were suspected of having TMD by screening and suggesting a need for more detailed clinical functional analysis. The literature reports a wide range of TMD in athletes (between 12% and 100%) and is difficult to compare due to different methodological approaches.³⁹ The current study showed a statistically significantly higher prevalence in CA than AA (Table 1). This is in line with a study on karate athletes (54% in competitive vs. 18% in recreational athletes).⁴⁰ Whereas available data were based on contact-sports,³⁹ the present study verified the elevated prevalence also for non-contact sports.

The determined personal oral hygiene (Figure 1A), as well as self-reported oral symptoms (Figure 1B), were

similar to the results of other studies. Daily toothbrushing was frequent,^{18,41} but less than one-half of the participants reported performing interdental cleaning,²¹ which is important to maintain periodontal health.³² Regarding the use of professional dental services, more than 20% did not regularly attend a dentist for examinations. Other studies reported comparable or lower rates of dental visits in the last 12 months.^{2,4,15,18} The association between regular professional teeth cleaning and no signs of periodontitis in CA underlines the importance of professional support for prevention. The prevalence of oral symptoms in CA (Figure 1B) was similar to reported data in literature: 8% to 10% for pain related to teeth,^{3,17} 39% to 60% for bleeding gums,^{2,3} and 23% to 27% for sensitive teeth.^{3,17,41} As the oral health status was slightly worse in CA, this could suggest a different perception of oral health in CA.

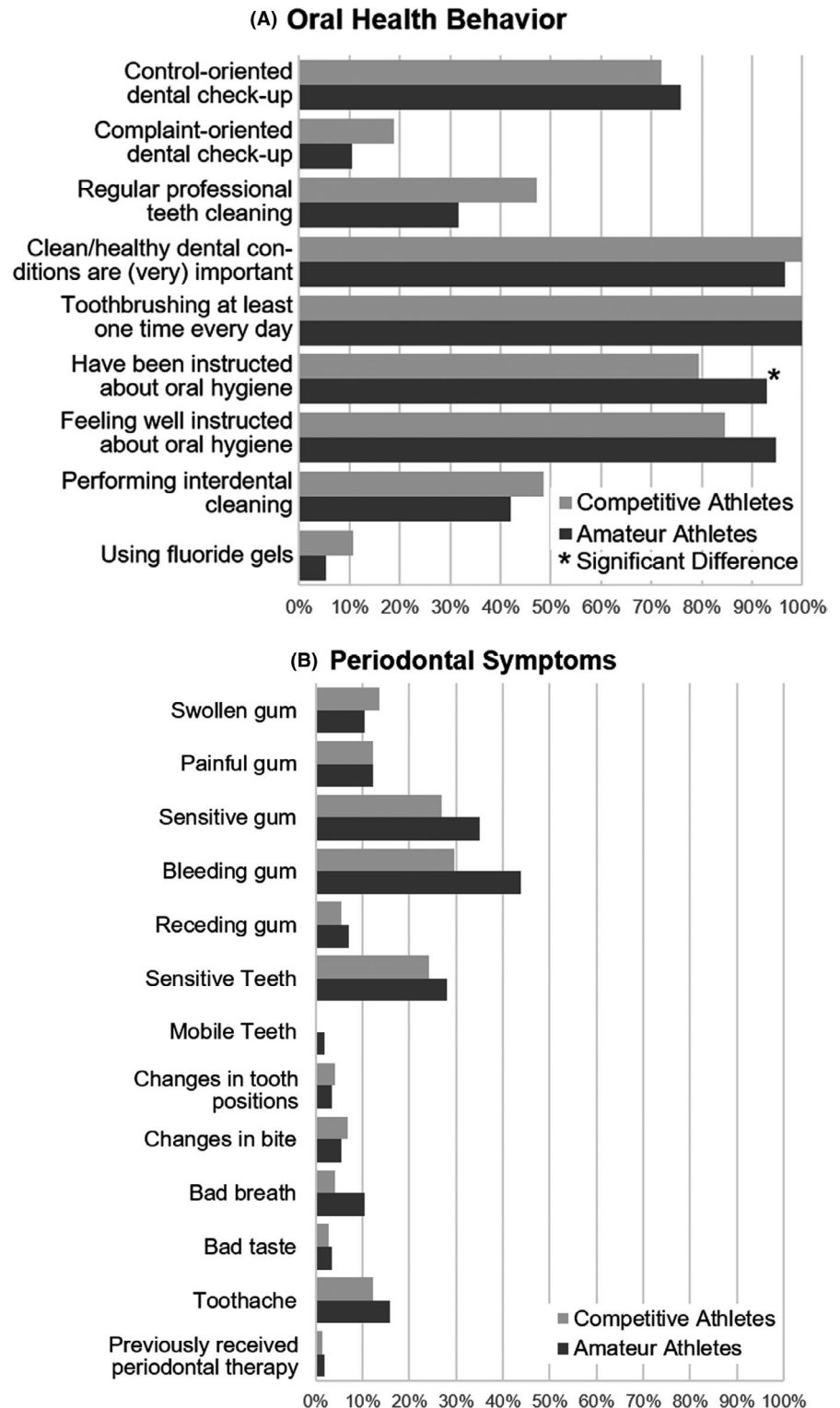
All in all, it is remarkable that neither the oral hygiene (PI, PBI) nor the self-reported oral health behavior can explain the higher prevalence of periodontal treatment need in CA. Consequently, another factor seems to affect elite athletes' periodontal tissues. Therefore, a systemic impact of the physical stress of elite sports on oral inflammation should be investigated.

Regarding the comparison between runners and biathletes, some minor differences in oral health status (F-T, BEWE) but not in oral health behavior have been observed. On the one hand, besides the disciplines, the higher mean age of the runners (on average four years older) could influence these highly age-dependent parameters. On the other hand, runners are expected to be high consumers of sport nutrition as sport drinks and carbohydrate gels leading to a high risk for both caries and non-caries wear. Further studies on risk factors specific for the different sports disciplines are necessary.

4.3 | Strengths and limitations

An apparent strength of the study is the comprehensive orofacial examination. To the authors' knowledge, this is the first study on athletes evaluating dental (caries and erosion), periodontal, and functional aspects in the same cohort. In combination with the detailed information about oral health behaviors and periodontal symptoms, an overall appraisal of the athlete's oral health is possible. The same skilled dentist performed all examinations. CA were clearly serious elite athletes as they were all current members of the German national teams and national perspective and youth squads. They were active in competitive sport for already seven years and trained about 17 h per week. The AA group can be regarded as control group because of similar age and same gender ratio, same origin and examination with the same protocol by the same

FIGURE 1 Results of Questionnaires about Oral Health Behavior (A) and Self-reported Periodontal Symptoms (B)



skilled dentist. This is important as the majority of studies on athletes do not include controls. Furthermore, AA as a control group helps to generate possible hypotheses on competitive sports as a risk factor.

Limitations of the study include the lack of a radiographic examination. As a result, the prevalence of caries could be underestimated,⁴² and no statement about totally retained or impacted wisdom teeth is possible. Neither

signs nor symptoms of pericoronitis were recorded. As no periodontal chart was determined, no information about attachment loss is available, and thus, the prevalence of periodontitis cannot be clearly stated. Furthermore, a separate evaluation for some disciplines is not meaningful regarding the very small number of athletes. Moreover, despite multiple testing no Bonferroni correction was used in this first retrospective pilot study. Consequently,

the interpretation of the statistics needs caution considering the potential risk of type 1 error. For some outcomes, differences between the different disciplines were seen (Table S1). Nevertheless, subgroup sizes were too small for drawing conclusions and also the comparison of biathletes and runners has to be interpreted with caution.

There are some potential risks for bias: Both groups have selection bias as attending dental check-ups was voluntary. Neither examination nor data extraction was blinded. Examination settings differed as AA were examined at a dentist's chair and CA at an examination couch with a headlight. In general, the comparison to AA could be criticized as they are physically active, too, and does not allow comparison to the general population. Furthermore, for interpreting the data, differences in performed sport disciplines between the groups have to be considered: While CA were all endurance athletes of five disciplines, AA included various disciplines, also power and team sports. About 40% of AA did several sports as they studied sports at university. This could lead to a less specific risk profile of AA, and therefore, could be another source of potential group difference.

Despite these limitations, these first data for elite athletes in Germany suggest differences in oral health may exist compared with recreational athletes. Further prospective and longitudinal research is needed to obtain results from a larger sample of athletes. Factors related to health status including health behaviors, knowledge, and beliefs among the athletes and their support network (parents/carers, coaches, sports scientists, etc.) should be considered. Especially, a possible systemic influence of the intensive sport should be investigated. Dental care programs for this population group could improve the oral health status.

5 | PERSPECTIVE

In general, young German athletes (CA and AA) showed signs of gingival inflammation and needed to improve their oral health behavior. CA showed more D-T and a higher prevalence of significant signs of periodontitis and positive TMD screening findings than AA, but comparable oral health behavior and oral hygiene. This may imply an additional systemic influence factor of elite sports on oral inflammation, and therefore, an increased need for dental care. Prospective studies on physical stress and oral inflammation should investigate this hypothesized interaction. Furthermore, prospective longitudinal studies with more participants of different disciplines and age groups will be important to determine risk levels and determinants of disease in competitive sports. Subsequently, special oral health promotion strategies should be discussed.

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CONFLICT OF INTEREST

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

ORCID

Cordula Leonie Merle  <https://orcid.org/0000-0002-0112-7631>

Rainer Haak  <https://orcid.org/0000-0002-3716-4231>

Gerhard Schmalz  <https://orcid.org/0000-0002-6602-377X>

Ian Needleman  <https://orcid.org/0000-0003-4696-1651>

Bernd Wolfarth  <https://orcid.org/0000-0001-6501-3003>

Dirk Ziebolz  <https://orcid.org/0000-0002-9810-2368>

Jan Wüstenfeld  <https://orcid.org/0000-0003-4730-0214>

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SUPPORTING INFORMATION

Additional supporting information may be found in the online version of the article at the publisher's website.

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2.5. Associations of Blood and Performance Parameters with Signs of Periodontal Inflammation in Young Elite Athletes

Nachdem in der Arbeit 2.4. in den Kollektiven der Spitzen- und Amateursportler insbesondere eine erhöhte gingivale und parodontale Inflammation als mögliche Folge der sportlichen Aktivität gefunden werden konnte, stellt sich die Frage inwieweit diese entzündlichen Veränderungen auch systemisch nachgewiesen werden können und v.a., ob eine gingivale bzw. parodontale Inflammationsreaktion möglicherweise in diesem Zusammenhang auch Einfluss auf die körperliche Leistungsfähigkeit haben kann. Wie bereits beschrieben ist dieser Zusammenhang bei Patienten mit chronischen Erkrankungen (beispielsweise Diabetes mellitus Typ II) bereits bekannt. Insofern galt es im globalen Aspekt der sportmedizinischen Vorsorgeuntersuchung die Relevanz einer oralen Entzündungsreaktion bei jugendlichen Leistungssportlern zu erfassen. Zusammenfassend lässt sich jedoch postulieren, dass es zum gegenwärtigen Zeitpunkt keinen klaren Hinweis darauf gibt, dass eine entzündliche Veränderung des Zahnhalteapparates sich signifikant negativ auf die Leistungsfähigkeit bei jungen Spitzensportlern auswirkt. Dennoch konnte in dieser Arbeit ein Trend hinsichtlich einer geringen maximalen Leistungsfähigkeit bei Sportlern mit vermehrter parodontaler Entzündungsreaktion nachgewiesen werden. Dabei ist jedoch herauszustellen, dass die bestehenden inflammatorischen Entzündungen zwar im Vergleich zu Normalbevölkerung und Amateursportlern deutlich häufiger vorhanden, in ihrem klinischen Ausmaß jedoch insgesamt nur gering bis mäßig ausgeprägt sind. Es finden sich vorrangig gingivale Entzündungsreaktionen. Entgegen Publikationen bei Patienten mit fortgeschrittenen Formen der parodontalen Entzündung fanden sich in dem Kollektiv der Spitzensportler im Standardlabor nur unspezifische Laborwertveränderungen ohne Nachweis von relevanten Erhöhungen spezifischer Entzündungswerte. Diese Untersuchung war somit vor dem Hintergrund einer hochleistungssportbedingten Peri-/Myokarditis hochgradig relevant, da sich zuvor postulieren ließ, dass eine sportassoziierte, vermehrte orale Inflammationsreaktion möglicherweise eine erhöhte systemische Entzündungsreaktion begünstigt, die konsekutiv die Auftretenswahrscheinlichkeit für eine kardial entzündliche Erkrankung begünstigen könnte.



Article

Associations of Blood and Performance Parameters with Signs of Periodontal Inflammation in Young Elite Athletes—An Explorative Study

Cordula Leonie Merle ^{1,2,*}, Lisa Richter ², Nadia Challakh ², Rainer Haak ², Gerhard Schmalz ², Ian Needleman ^{3,4}, Peter Rüdrieh ⁵, Bernd Wolfarth ^{5,6,7}, Dirk Ziebolz ^{2,†} and Jan Wüstenfeld ^{5,6,†}

- ¹ Department of Prosthetic Dentistry, UKR University Hospital Regensburg, 93053 Regensburg, Germany
² Department of Cariology, Endodontology and Periodontology, University of Leipzig, 04103 Leipzig, Germany
³ Centre for Oral Health and Performance, UCL Eastman Dental Institute, London WC1E 6BT, UK
⁴ UK IOC Research Centre, London WC1E 6BT, UK
⁵ Department of Sports Medicine, Institute for Applied Scientific Training, 04109 Leipzig, Germany
⁶ Department of Sports Medicine, Charité University Medicine, 10115 Berlin, Germany
⁷ Institute of Sports Science, Humboldt University, 10099 Berlin, Germany
* Correspondence: cordula.merle@klinik.uni-regensburg.de
† These authors contributed equally to this work.



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Abstract: This retrospective cross-sectional study aimed to explore interactions between signs of periodontal inflammation and systemic parameters in athletes. Members of German squads with available data on sports medical and oral examination were included. Groups were divided by gingival inflammation (median of papillary bleeding index, PBI \geq median) and signs of periodontitis (Periodontal Screening Index, PSI \geq 3). Age, gender, anthropometry, blood parameters, echocardiography, sports performance on ergometer, and maximal aerobic capacity (VO_{2max}) were evaluated. Eighty-five athletes ($f = 51\%$, 20.6 ± 3.5 years) were included (PBI < 0.42 : 45%; PSI ≥ 3 : 38%). Most associations were not statistically significant. Significant group differences were found for body fat percentage and body mass index. All blood parameters were in reference ranges. Minor differences in hematocrit, hemoglobin, basophils, erythrocyte sedimentation rates, urea, and HDL cholesterol were found for PBI, in uric acid for PSI. Echocardiographic parameters ($n = 40$) did not show any associations. Athletes with PSI ≥ 3 had lower VO_{2max} values (55.9 ± 6.7 mL/min/kg vs. 59.3 ± 7.0 mL/min/kg; $p = 0.03$). In exercise tests ($n = 30$), athletes with PBI < 0.42 achieved higher relative maximal load on the cycling ergometer (5.0 ± 0.5 W/kg vs. 4.4 ± 0.3 W/kg; $p = 0.03$). Despite the limitations of this study, potential associations between signs of periodontal inflammation and body composition, blood parameters, and performance were identified. Further studies on the systemic impact of oral inflammation in athletes, especially regarding performance, are necessary.

Keywords: performance; systemic inflammation; physical endurance; physical fitness; maximal aerobic capacity; gingivitis

1. Introduction

The high-performance standards of elite athletes are built on foundations of physical fitness, health, and wellbeing. It may be a surprise, therefore, that oral ill health is common in elite athletes and results in an increased oral inflammatory burden [1]. The prevalence of both gingivitis and periodontitis can be high [1] and differs significantly from non-elite controls [2–4]. For instance, among footballers, a periodontitis prevalence of 41% was reported [5].

Oral infections, including periodontal diseases, cause increased systemic inflammation [6], which can resolve following treatment [7], although there are inconsistencies between studies [8]. The relationship between oral health and physical activity could

be bidirectional. Some studies have reported an impairment from poor oral health on measures of physical activity and performance [9]. On the other hand, intensive physical activity leads to systemic changes: levels of (pro-)inflammatory cytokines [10,11] as well as stress hormones [12] increase. On the other side, immunoglobulin A levels decrease [13]. A transitional reduced cellular immune response [14,15] has been proposed to lead to an open window for infections [16]. However, the impact of these changes on oral inflammation is not clear.

The relationship between oral health and anaerobic capacity of athletes has received very little attention. A recent study in elite rowers did not find a relationship between dental caries and anaerobic capacity, although the study had few participants and differences in oral health status between comparison groups were small [17]. There has been no published research investigating the influence of oral inflammation on the performance of athletes or systemic biomarkers. Nevertheless, several studies have found negative impacts of poor oral health on self-reported measures of performance [18,19]. Consequently, this retrospective explorative study aimed to investigate associations between signs of periodontal inflammation and systemic parameters in elite athletes. Associations between gingival and periodontal inflammation to blood, echocardiographic, and performance parameters were investigated. It was hypothesized that these parameters would be affected in athletes with increased signs of periodontal inflammation.

2. Materials and Methods

2.1. Study Design and Participants

This pilot study was based on a retrospective data evaluation from a collaboration between the Department of Cariology, Endodontology and Periodontology and the Institute for Applied Scientific Training (IAT) Leipzig. Dental examinations were performed as a supplement to the annual sports medical and performance diagnostics.

Inclusion criteria were athletes of German national teams, perspective, or youth squads, aged between 18 and 30 years, male and female. The sports medical and standardized dental examination (performed on the same day) were conducted between May and December 2019. Participants with incomplete dental examination were excluded. A comprehensive description of the cohort and oral health status was already published elsewhere [4].

The study was reviewed and approved by the Ethics Committee of the medical faculty of Leipzig University, Germany (No. 091/20-ek). All participants were informed verbally and in writing about the scientific use of their clinical data and provided their informed consent for participation in research studies. The recommendations for strengthening the reporting of cross-sectional studies (STROBE) were considered [20].

2.2. Data Collection

Data on general characteristics, blood parameters, echocardiographic examination, and sports performance tests as part of the sports medical records were exported from the IAT database. Data on signs of periodontal inflammation were extracted from patients' dental records.

General characteristics. Recorded general characteristics were age, gender, training, and anthropometric data including body mass index (BMI), body fat percentage (BFP), lean body mass (LBM), and resting heart rate (RHR).

Blood parameters. The annual sports medical and performance diagnostics comprised extensive blood tests for all athletes. A complete blood count with the number of erythrocytes, leukocytes, thrombocytes, lymphocytes, neutrophils, basophils, eosinophils and monocytes, hematocrit, hemoglobin, mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), mean corpuscular volume (MCV), immature reticulocyte fraction (IFR), high (HFR), medium (MFR), and low-fluorescence reticulocytes (LFR) was performed. Neutrophil-lymphocyte (NLR), monocyte-lymphocyte (MLR), and platelet-lymphocyte ratios (PLR) were calculated. Further determined blood parame-

ters were erythrocyte sedimentation rates after 1 (ESR1h) and 2 h (ESR2h), iron, ferritin, sodium, calcium, potassium, magnesium, gamma-glutamyl transferase (GGT), glutamic-pyruvate-transaminase (GPT), urea, uric acid, creatine kinase, total protein, total cholesterol, low-density lipoprotein (LDL) and high-density lipoprotein (HDL) cholesterol, LDL/HDL ratio, glucose, and triglycerides.

Echocardiographic examination. Additionally, if available, sport-specific and performance-related measurements of transthoracic echocardiographic examination were exported: absolute heart volume (HV_abs), relative heart volume (HV_rel) (calculated by the equation of Dickhuth) [21], left atrial size (LA), left ventricular end-diastolic dimension (LVEDd), and tricuspid annular plane systolic excursion (TAPSE).

Sports performance. Maximal aerobic capacity (VO_{2max}) by spiroergometry was extracted if available. If not, it was estimated by the equation of Rexhepi and Brestoci [22]. Furthermore, data from sports performance tests with incremental exercise tests on running or cycle ergometer were considered: RHR, heart rates (HF), lactate, and power, respectively, and speed were extracted for analysis. Besides minimum, maximum, and differences, the speed/power output at individual anaerobic threshold (IAT), lactate threshold 1 (LT1, initial rise after basal lactate), lactate threshold 2 (LT2, Dickhuth model: basal lactate + 1.5 mmol/L), without load ($p = 0$), and maximal load (P_{max}) at the ergometer were tested.

Signs of periodontal inflammation. Data for both gingival and periodontal inflammation were extracted from patients' dental records. A comprehensive (standardized) orofacial examination was performed using a headlight on an examination couch at the IAT. A single skilled dentist that was trained in these periodontal parameters examined all athletes ($\kappa > 80\%$). Gingival inflammation was assessed by the papillary bleeding index (PBI) [23] which discriminates five scores after probing (0: no bleeding; 1: single bleeding point; 2: several bleeding points or fine line; 3: interdental triangle filled with blood, 4: profuse bleeding). The PBI index was calculated per patient by division of the total sum by the total number of interdental papillae. Periodontal conditions (= sign of periodontitis/periodontal treatment need) were examined using the Periodontal Screening Index [24]: score 0 to 2 has probing depths less than 3.5 mm. Score 0 shows no bleeding, no calculus, score 1 bleeding on probing, and score 2 calculus. A score of 3 or 4 indicates increased probing depths (3: pocket depth 3.5–5.5 mm; 4: pocket depth > 5.5 mm) as a sign of periodontitis. Third molars were not included in this evaluation despite they took a more anterior position.

2.3. Statistical Analysis

Statistical analysis was performed with SPSS Statistics for Windows (version 23.0, IBM Corp., Armonk, NY, USA). Possible associations from signs of periodontal inflammation to anthropometric data, blood, echocardiographic, and exercise test parameters were examined. For analyzing associations to gingival inflammation, the athletes were divided into two groups by median of the PBI (PBI $<$ median vs. PBI \geq median). Regarding signs of periodontitis, group division was based on having increased probing depths (≥ 3.5 mm) or not (PSI $<$ 3 vs. PSI \geq 3). Quantitative variables were presented by mean and standard deviation (SD). Independent, normal-distributed samples were analyzed with a *t*-test. For non-normal distributed samples, the Mann–Whitney U test was used. All tests were performed two sided, with a significance level at $p < 0.05$ and under exclusion of missing data. Normal distribution was verified by Kolmogorov–Smirnov test. For parameters with an association ($p < 0.1$) and plausible link to PBI or PSI, a multivariate analysis of variance (MANOVA) and, for significant models, linear regression were planned.

3. Results

3.1. Athletes

Records of 85 athletes from the German national elite, perspective, and youth squads ($f = 51\%$, 20.6 ± 3.5 years) were included for retrospective evaluation. Table 1 shows their characteristics, training, and anthropometric data.

Table 1. Characteristics of the athletes (entire cohort).

| | <i>n</i> | % |
|-----------------------------------------|-------------|------------|
| <i>n</i> —All disciplines | 85 | 100.0 |
| -Running | 39 | 45.9 |
| -Biathlon | 24 | 28.2 |
| -Cross-country skiing | 10 | 11.8 |
| -Rowing | 8 | 9.4 |
| -Triathlon | 4 | 4.7 |
| Female gender | 43 | 50.6 |
| | mean | SD |
| Age (years) | 20.6 | ± 3.5 |
| Training sessions per week | 9.8 | ± 2.9 |
| Training time (h) per week | 17.3 | ± 4.8 |
| Training history (years) | 7.2 | ± 2.9 |
| Body mass index (kg/m ²) | 20.7 | ± 2.1 |
| Body weight (kg) | 65.9 | ± 10.5 |
| Body height (cm) | 177.8 | ± 9.6 |
| Resting heart rate ^a (bpm) | 49.0 | ± 8.2 |
| Body fat percentage (by impedance) (%) | 8.7 | ± 3.5 |
| Body fat percentage (by skin folds) (%) | 13.1 | ± 4.7 |
| Lean body mass (%) | 57.3 | ± 9.2 |
| VO _{2max} (mL/min/kg) | 58.02 | ± 7.02 |

Abbreviations: *n*: number of participants; VO_{2max}: maximal aerobic capacity. ^a Missing data for eight participants ($n = 77$).

3.2. Signs of Periodontal Inflammation

Mean gingival inflammation (PBI) was 0.48 ± 0.29 and the median was 0.42 (IQR: 0.31; 0.69). The subgroup PBI < 0.42 contained 40 and the subgroup PBI ≥ 0.42 45 athletes. As such, 53 athletes had a PSI < 3 and 32 a PSI ≥ 3 with 11 having a PSI ≥ 3 in more than one sextant. No athlete showed a PSI score of 4. The associations between body composition and performance with periodontal health are shown in Table 2. Most associations were not statistically significant at $p < 0.05$. BFP was significantly lower in PBI ≥ 0.42 (PBI < 0.42: 14.4 ± 4.8 vs. PBI ≥ 0.42 : 11.9 ± 4.3 ; $p = 0.02$) but significantly higher in PSI ≥ 3 (PSI < 3: 12.4 ± 4.9 vs. PSI ≥ 3 : 14.3 ± 4.2 ; $p = 0.047$). Athletes with signs of periodontitis also had a higher BMI (PSI < 3: 20.3 ± 2.0 vs. PSI ≥ 3 : 21.5 ± 2.0 ; $p = 0.01$).

3.3. Blood Parameters

Results of the complete blood count (Table 3) and further blood parameters (Table 4) are presented for the entire cohort and separately for the divided groups by PBI and PSI. Again, most associations were not statistically significant. However, statically significant differences between athletes with a lower and those with a higher PBI were found for hematocrit (PBI < 0.42: $41.5 \pm 2.8\%$ vs. PBI ≥ 0.42 : $42.6 \pm 2.4\%$; $p = 0.04$), hemoglobin (14.2 ± 1.2 g/dL vs. 14.7 ± 0.9 g/dL; $p = 0.04$), basophils ($0.5 \pm 0.2\%$ vs. $0.4 \pm 0.2\%$; $p = 0.03$), ESR1h (5.1 ± 3.3 mm vs. 3.8 ± 2.8 mm; $p = 0.01$), ESR2h (10.6 ± 7.2 mm vs. 8.0 ± 5.7 mm; $p = 0.04$), urea (6.3 ± 1.7 mmol/L vs. 5.5 ± 1.4 mmol/L; $p = 0.04$), and HDL

cholesterol (1.9 ± 0.3 mmol/l vs. 1.7 ± 0.2 mmol/L; $p = 0.02$). In relation to periodontitis based on $PSI \geq 3$, statistically significant differences were found only for uric acid ($PSI < 3$: 251.3 ± 74.1 μ mol/L vs. $PSI \geq 3$: 283.1 ± 60.8 μ mol/L; $p = 0.04$). Multivariate linear regression was performed for urea, uric acid, HDL cholesterol, thrombocytes, and iron, whereby ANOVA revealed significance for two different models, including urea, uric acid, and thrombocytes, however, showing a small effect size (Supplementary Materials Table S1).

3.4. Echocardiographic Parameters

An echocardiographic examination was performed on a subgroup of 40 athletes. The results of the quantitative measurements are presented in Supplementary Materials Table S2. HV_rel was, on average, 12 mL/kg, LA 3.6 cm, and TAPSE 2.5 cm. There were no statistically significant associations with PBI or PSI.

3.5. Performance Parameters

Spiroergometric data were available for 41 athletes; 30 completed further performance diagnostics with incremental exercise tests (Table 5). Ergometer types were running ($n = 20$, biathletes) or cycling ($n = 10$, cross-country skiers). Overall, in athletes, those with signs of periodontitis had lower VO_{2max} values (55.9 ± 6.7 mL/min/kg vs. 59.3 ± 7.0 mL/min/kg; $p = 0.03$). Detailed data on power on the ergometer are presented in Table 6; the group with less gingival inflammation achieved a higher relative maximal load on the cycling ergometer ($PBI < 0.42$: 5.0 ± 0.5 W/kg vs. $PBI \geq 0.42$: 4.4 ± 0.3 W/kg; $p = 0.03$).

Table 2. Characteristics of the athletes (entire cohort) and their associations with periodontal health (PBI and PSI).

| | Association to PBI | | | | | | Association to PSI | | | | | | | |
|--------------------------------------|--------------------|------|------------|-------|---------|---------|--------------------|---------|------|---------|-------|---|------|-------------|
| | PBI < 0.42 | | PBI ≥ 0.42 | | p-Value | PSI < 3 | | PSI ≥ 3 | | p-Value | | | | |
| | n | % | n | % | | n | % | n | % | | | | | |
| n | 40 | 47.1 | 45 | 52.9 | | 53 | 62.4 | 32 | 37.6 | | | | | |
| Female gender | 25 | 62.5 | 18 | 40.0 | 0.05 | 28 | | 15 | | | | | | |
| | mean | ± | SD | mean | ± | SD | p-Value | mean | ± | SD | mean | ± | SD | p-Value |
| Age (years) | 21.0 | ± | 3.0 | 22.0 | ± | 3.8 | 0.35 | 21.4 | ± | 3.2 | 21.6 | ± | 3.9 | 0.81 |
| Training sessions per week | 10.4 | ± | 3.2 | 9.2 | ± | 2.4 | 0.15 | 9.9 | ± | 2.7 | 9.5 | ± | 3.1 | 0.51 |
| Training time (h) per week | 16.8 | ± | 4.0 | 17.7 | ± | 5.4 | 0.58 | 17.4 | ± | 5.1 | 17.1 | ± | 4.3 | 0.99 |
| Training history (years) | 6.7 | ± | 3.0 | 7.6 | ± | 2.9 | 0.12 | 6.9 | ± | 2.8 | 7.7 | ± | 3.1 | 0.23 |
| BMI (kg/m²) | 20.9 | ± | 2.0 | 20.5 | ± | 2.1 | 0.36 | 20.3 | ± | 2.0 | 21.5 | ± | 2.0 | 0.01 |
| Body weight (kg) | 65.9 | ± | 10.5 | 66.0 | ± | 10.5 | 0.99 | 64.5 | ± | 10.2 | 68.3 | ± | 10.6 | 0.11 |
| Body height (cm) | 176.8 | ± | 9.4 | 178.6 | ± | 9.9 | 0.35 | 177.8 | ± | 9.7 | 177.6 | ± | 9.6 | 0.96 |
| RHR (bpm) | 48.5 | ± | 8.4 | 49.3 | ± | 8.1 | 0.59 | 47.9 | ± | 6.7 | 50.7 | ± | 10.1 | 0.25 |
| BFP (by impedance) (%) | 9.5 | ± | 3.7 | 7.9 | ± | 3.1 | 0.07 | 8.1 | ± | 3.7 | 9.6 | ± | 2.8 | 0.01 |
| BFP (by skin folds) (%) | 14.4 | ± | 4.8 | 11.9 | ± | 4.3 | 0.02 | 12.4 | ± | 4.9 | 14.3 | ± | 4.2 | 0.05 |
| LBM (%) | 56.4 | ± | 9.0 | 58.0 | ± | 9.3 | 0.43 | 56.4 | ± | 8.5 | 58.7 | ± | 10.1 | 0.37 |
| VO_{2max} (mL/min/kg) | 56.9 | ± | 6.3 | 59.0 | ± | 7.5 | 0.44 | 59.3 | ± | 7.0 | 55.9 | ± | 6.7 | 0.03 |

Abbreviations: BMI: body mass index, RHR: resting heart rate; BFP: body fat percentage; LBM: lean body mass; n: number of participants; PBI: Papillary Bleeding Index; PSI: Periodontal Screening Index with PSI ≥ 3 indicating increased probing depths as a sign of probable periodontitis; VO_{2max}: maximal aerobic capacity. Bold marks significant differences (*p* < 0.05).

Table 3. Complete blood count (BC) of the athletes (entire cohort) and their associations with periodontal health (PBI and PSI).

| | Total n = 85 | | | Reference Ranges | Association to PBI | | | | Association to PSI | | | | | | | | | |
|---------------------------------------------|-----------------|---|------|---------------------|--------------------|---|------------|-------|--------------------|---------|-------------|---------|---|---------|-------|---|------|------|
| | | | | | PBI < 0.42 | | PBI ≥ 0.42 | | p-Value | PSI < 3 | | PSI ≥ 3 | | p-Value | | | | |
| Erythrocytes (× 10 ⁶ /μL) | 4.8 | ± | 0.4 | 3.9–6.1 | 4.8 | ± | 0.4 | 4.9 | ± | 0.4 | 0.15 | 4.8 | ± | 0.4 | 4.8 | ± | 0.4 | 0.86 |
| Hematocrit (%) | 42.1 | ± | 2.6 | 34.1–44.9 | 41.5 | ± | 2.8 | 42.6 | ± | 2.4 | 0.04 | 42.0 | ± | 2.6 | 42.2 | ± | 2.7 | 0.71 |
| Hemoglobin (g/dL) | 14.5 | ± | 1.1 | 12–18 | 14.2 | ± | 1.2 | 14.7 | ± | 0.9 | 0.04 | 14.5 | ± | 1.1 | 14.5 | ± | 1.1 | 0.89 |
| MCH (fmol) | 1.9 | ± | 0.1 | 1.5–2.1 | 1.9 | ± | 0.1 | 1.9 | ± | 0.1 | 0.52 | 1.9 | ± | 0.1 | 1.9 | ± | 0.1 | 0.93 |
| MCHC (mmol/L) | 21.4 | ± | 0.5 | 20.0–22.7 | 21.3 | ± | 0.6 | 21.4 | ± | 0.5 | 0.30 | 21.4 | ± | 0.6 | 21.3 | ± | 0.5 | 0.71 |
| MCV (fl) | 87.3 | ± | 3.3 | 79.4–100 | 87.2 | ± | 3.5 | 87.5 | ± | 3.1 | 0.65 | 87.3 | ± | 3.8 | 87.4 | ± | 2.4 | 0.84 |
| IRF (%) | 3.7 | ± | 2.0 | 2.1–17.5 | 3.9 | ± | 2.3 | 3.4 | ± | 1.7 | 0.23 | 3.7 | ± | 2.3 | 3.6 | ± | 1.5 | 0.70 |
| HFR (%) | 0.2 | ± | 0.3 | 0–2.4 | 0.2 | ± | 0.4 | 0.2 | ± | 0.3 | 0.71 | 0.2 | ± | 0.4 | 0.2 | ± | 0.3 | 0.75 |
| MFR (%) | 3.4 | ± | 1.9 | 1.8–14.4 | 3.7 | ± | 2.2 | 3.2 | ± | 1.7 | 0.24 | 3.5 | ± | 2.2 | 3.4 | ± | 1.4 | 0.75 |
| LFR (%) | 96.3 | ± | 2.0 | 87.8–99.5 | 96.1 | ± | 2.3 | 96.6 | ± | 1.7 | 0.23 | 96.3 | ± | 2.3 | 96.4 | ± | 1.5 | 0.70 |
| Leukocytes (/nl) | 5.9 | ± | 1.3 | 3.6–9.8 | 5.7 | ± | 1.2 | 6.0 | ± | 1.4 | 0.19 | 5.8 | ± | 1.2 | 6.0 | ± | 1.5 | 0.35 |
| Lymphocytes (%) | 39.0 | ± | 7.3 | 19–53 | 40.5 | ± | 7.8 | 37.8 | ± | 6.5 | 0.08 | 39.1 | ± | 7.0 | 39.0 | ± | 7.7 | 0.96 |
| Neutrophils (%) | 46.7 | ± | 7.8 | 34–71 | 45.5 | ± | 8.7 | 47.8 | ± | 6.7 | 0.17 | 46.5 | ± | 7.3 | 47.1 | ± | 8.6 | 0.75 |
| Basophils (%) | 0.5 | ± | 0.2 | 0.1–1.2 | 0.5 | ± | 0.2 | 0.4 | ± | 0.2 | 0.03 | 0.5 | ± | 0.3 | 0.4 | ± | 0.2 | 0.62 |
| Eosinophils (%) | 3.3 | ± | 2.7 | 1–7 | 3.2 | ± | 2.0 | 3.3 | ± | 3.2 | 0.95 | 3.4 | ± | 3.1 | 3.1 | ± | 1.7 | 0.88 |
| Monocytes (%) | 10.5 | ± | 2.1 | 5.0–12.0 | 10.3 | ± | 1.8 | 10.7 | ± | 2.3 | 0.43 | 10.6 | ± | 2.2 | 10.4 | ± | 1.9 | 0.74 |
| Thrombocytes (/nl) | 236.7 | ± | 48.3 | 150–361 | 247.2 | ± | 49.2 | 227.4 | ± | 45.9 | 0.06 | 239.6 | ± | 47.1 | 232.1 | ± | 50.5 | 0.49 |
| NLR | 1.3 | ± | 0.5 | 0.1–3.2 | 1.22 | ± | 0.5 | 1.34 | ± | 0.5 | 0.09 | 1.27 | ± | 0.5 | 1.31 | ± | 0.6 | 0.84 |
| MLR | 0.3 | ± | 0.1 | 2.0–8.6 | 0.26 | ± | 0.1 | 0.30 | ± | 0.1 | 0.18 | 0.28 | ± | 0.1 | 0.28 | ± | 0.1 | 0.91 |
| PLR | 110.3 | ± | 32.3 | 46.8–218.0 | 113.8 | ± | 29.1 | 107.2 | ± | 35.0 | 0.14 | 112.8 | ± | 32.0 | 106.2 | ± | 33.0 | 0.33 |

Abbreviations: MCH: mean corpuscular hemoglobin (MCH); MCHC: mean corpuscular hemoglobin concentration; MCV: mean corpuscular volume (MCV); HFR: high fluorescence reticulocytes; IRF: immature reticulocyte fraction (IRF), LFR: low fluorescence reticulocytes; MFR: medium fluorescence reticulocytes; MLR: monocyte-lymphocyte ratio; n: number of participants; NLR: neutrophil-lymphocyte ratio; PBI: Papillary Bleeding Index; PLR: platelet-lymphocyte ratio; PSI: Periodontal Screening Index with PSI ≥ 3 indicating increased probing depths as a sign of probable periodontitis. Bold marks significant differences (*p* < 0.05).

Table 4. Further blood parameters of the athletes (entire cohort) and their associations to with periodontal health (PBI and PSI).

| | Total <i>n</i> = 85 | | | Reference Ranges | Association to PBI | | | | | Association to PSI | | | | | | | | |
|-----------------------------------------|------------------------|---|-------|---------------------|--------------------|---|------------|-------|-----------------|--------------------|-------------|---------|---|-----------------|-------|---|--------|-------------|
| | | | | | PBI < 0.42 | | PBI ≥ 0.42 | | <i>p</i> -Value | PSI < 3 | | PSI ≥ 3 | | <i>p</i> -Value | | | | |
| ESR1h (mm) | 4.4 | ± | 3.1 | <10 | 5.1 | ± | 3.3 | 3.8 | ± | 2.8 | 0.01 | 4.7 | ± | 3.3 | 4.0 | ± | 2.7 | 0.32 |
| ESR2h (mm) | 9.2 | ± | 6.5 | <20 | 10.6 | ± | 7.2 | 8.0 | ± | 5.7 | 0.04 | 9.7 | ± | 7.0 | 8.5 | ± | 5.7 | 0.76 |
| Iron (µmol/L) | 16.8 | ± | 7.1 | 6.6–30.1 | 15.6 | ± | 6.6 | 17.9 | ± | 7.3 | 0.13 | 16.6 | ± | 7.3 | 17.1 | ± | 6.9 | 0.75 |
| Ferritin (µg/L) | 62.2 | ± | 39.0 | 15–280 | 56.4 | ± | 32.7 | 67.4 | ± | 43.6 | 0.32 | 62.1 | ± | 41.3 | 62.5 | ± | 35.6 | 0.73 |
| Sodium ^a (mmol/L) | 139.9 | ± | 2.3 | 15–280 | 140.0 | ± | 2.3 | 139.9 | ± | 2.3 | 0.64 | 140.0 | ± | 2.5 | 139.8 | ± | 2.1 | 0.78 |
| Calcium (mmol/L) | 2.4 | ± | 0.1 | 2.2–2.6 | 2.4 | ± | 0.1 | 2.4 | ± | 0.1 | 0.35 | 2.4 | ± | 0.1 | 2.4 | ± | 0.1 | 0.21 |
| Potassium (mmol/L) | 4.2 | ± | 0.3 | 3.6–5.5 | 4.2 | ± | 0.2 | 4.2 | ± | 0.3 | 0.95 | 4.2 | ± | 0.3 | 4.2 | ± | 0.3 | 0.75 |
| Magnesium (mmol/L) | 0.8 | ± | 0.1 | 15–280 | 0.8 | ± | 0.1 | 0.8 | ± | 0.1 | 0.95 | 0.8 | ± | 0.1 | 0.8 | ± | 0.1 | 0.30 |
| GGT ^b (U/L) | 22.3 | ± | 5.7 | 0–55 | 22.2 | ± | 4.4 | 22.4 | ± | 6.7 | 0.74 | 23.1 | ± | 6.6 | 21.3 | ± | 4.3 | 0.29 |
| GPT ^b (U/L) | 40.2 | ± | 41.5 | 10–50 | 36.1 | ± | 18.5 | 43.5 | ± | 53.4 | 0.58 | 36.5 | ± | 16.9 | 45.2 | ± | 61.3 | 0.80 |
| Urea (mmol/L) | 5.9 | ± | 1.6 | 2.6–8.9 | 6.3 | ± | 1.7 | 5.5 | ± | 1.4 | 0.04 | 5.8 | ± | 1.6 | 6.0 | ± | 1.6 | 0.62 |
| Uric acid (µmol/L) | 263.3 | ± | 70.7 | 120–416 | 247.6 | ± | 65.8 | 277.2 | ± | 72.7 | 0.05 | 251.3 | ± | 74.1 | 283.1 | ± | 60.8 | 0.04 |
| Creatine kinase (U/L) | 427.5 | ± | 799.6 | 24–350 | 360.2 | ± | 412.5 | 487.3 | ± | 1030.6 | 0.07 | 340.9 | ± | 317.8 | 571.0 | ± | 1236.5 | 0.64 |
| Creatinine ^b (µmol/L) | 79.5 | ± | 11.7 | 44–97 | 78.76 | ± | 11.3 | 80.1 | ± | 12.1 | 0.64 | 80.5 | ± | 11.7 | 78.1 | ± | 11.8 | 0.41 |
| Total Protein (g/L) | 70.6 | ± | 3.5 | 66–88 | 70.8 | ± | 3.5 | 70.5 | ± | 3.5 | 0.70 | 70.2 | ± | 3.4 | 71.4 | ± | 3.5 | 0.13 |
| Total Cholesterol ^b (mmol/L) | 4.5 | ± | 0.8 | <5.2 | 4.7 | ± | 0.8 | 4.3 | ± | 0.7 | 0.30 | 4.5 | ± | 0.9 | 4.5 | ± | 0.6 | 0.79 |
| LDL Cholesterol ^b (mmol/L) | 2.3 | ± | 0.6 | <4.1 | 2.3 | ± | 0.7 | 2.3 | ± | 0.6 | 0.74 | 2.3 | ± | 0.7 | 2.3 | ± | 0.5 | 0.91 |
| HDL Cholesterol ^b (mmol/L) | 1.8 | ± | 0.3 | >0.9 | 1.9 | ± | 0.3 | 1.7 | ± | 0.2 | 0.02 | 1.8 | ± | 0.2 | 1.8 | ± | 0.3 | 0.51 |
| LDL/HDL Ratio ^b | 1.3 | ± | 0.4 | <3.5 | 1.3 | ± | 0.4 | 1.4 | ± | 0.4 | 0.41 | 1.3 | ± | 0.4 | 1.4 | ± | 0.4 | 0.53 |
| Glucose (mmol/L) | 4.7 | ± | 0.6 | 3.4–5.6 | 4.8 | ± | 0.4 | 4.6 | ± | 0.7 | 0.29 | 4.7 | ± | 0.6 | 4.7 | ± | 0.5 | 0.78 |
| Triglycerides ^b (mmol/L) | 0.9 | ± | 0.4 | <2.3 | 1.0 | ± | 0.5 | 0.8 | ± | 0.3 | 0.06 | 0.9 | ± | 0.5 | 0.9 | ± | 0.3 | 0.89 |

Abbreviations: ESR1h: erythrocyte sedimentation rate after 1 h; ESR2h: erythrocyte sedimentation rate after 2 h; GGT: gamma-glutamyl transferase; GPT: glutamic-pyruvate-transaminase; HDL: high-density lipoprotein; LDL: low-density lipoprotein; *n*: number of participants; PBI: Papillary Bleeding Index; PSI: Periodontal Screening Index with PSI ≥ 3 indicating increased probing depths as a sign of probable periodontitis. Bold marks significant differences (*p* < 0.05); ^a Missing data for two participants (*n* = 83); ^b Missing data for 18 participants (*n* = 67).

Table 5. Performance test parameters, heart frequencies, and lactate values during incremental exercise test and their association with periodontal health (PBI and PSI).

| | Total | | | Association to PBI | | | Association to PSI | | | | | | | | | | |
|----------------------|--------|---|------|--------------------|------------|---------|--------------------|---------|---------|------|-------|---|-------|-------|---|------|------|
| | n = 30 | | | PBI < 0.46 | PBI ≥ 0.46 | p-Value | PSI < 3 | PSI ≥ 3 | p-Value | | | | | | | | |
| RHR (bpm) | 52.0 | ± | 9.3 | 51.5 | ± | 9.3 | 52.5 | ± | 9.5 | 0.77 | 50.3 | ± | 7.3 | 53.8 | ± | 10.9 | 0.30 |
| HF_LT (bpm) | 143.9 | ± | 12.7 | 142.8 | ± | 14.1 | 144.8 | ± | 11.7 | 0.67 | 145.2 | ± | 12.20 | 142.5 | ± | 13.5 | 0.57 |
| HF_IAnT (bpm) | 174.4 | ± | 12.2 | 173.4 | ± | 13.4 | 175.2 | ± | 11.5 | 0.70 | 176.5 | ± | 11.1 | 172.3 | ± | 13.3 | 0.36 |
| HF_Pmax (bpm) | 194.6 | ± | 8.9 | 193.8 | ± | 7.3 | 195.4 | ± | 10.2 | 0.63 | 196.1 | ± | 7.4 | 193.2 | ± | 10.2 | 0.39 |
| HF_max (bpm) | 201.9 | ± | 1.3 | 202.1 | ± | 1.2 | 201.6 | ± | 1.4 | 0.39 | 201.7 | ± | 1.3 | 202.0 | ± | 1.4 | 0.51 |
| Lactate_LT1 (mmol/L) | 1.2 | ± | 0.3 | 1.2 | ± | 0.3 | 1.3 | ± | 0.3 | 0.31 | 1.2 | ± | 0.3 | 1.3 | ± | 0.4 | 0.52 |
| Lactate_LT2 (mmol/L) | 2.7 | ± | 0.3 | 2.7 | ± | 0.3 | 2.8 | ± | 0.3 | 0.31 | 2.7 | ± | 0.3 | 2.8 | ± | 0.4 | 0.52 |
| Lactate_max (mmol/L) | 9.5 | ± | 2.2 | 10.1 | ± | 1.9 | 8.9 | ± | 2.3 | 0.13 | 9.5 | ± | 2.1 | 9.4 | ± | 2.3 | 0.97 |

Abbreviations: IAnT: individual anaerobic threshold; HF: heart frequency LT1: lactate threshold 1, LT2: lactate threshold 2; max: maximal value; n: number of participants; PBI: Papillary Bleeding Index; PSI: Periodontal Screening Index with PSI ≥ 3 indicating increased probing depths as a sign of probable periodontitis; Pmax: maximal load; RHR: resting heart rate.

Table 6. Power on ergometer during incremental exercise tests and their association with periodontal health (PBI and PSI).

| | Total | | | Association to PBI | | | Association to PSI | | | | | | | | | | |
|---------------------------------|-------|---|------|--------------------|------------|---------|--------------------|---------|---------|-------------|-------|---|------|-------|---|------|------|
| | | | | PBI < 0.46 | PBI ≥ 0.46 | p-Value | PSI < 3 | PSI ≥ 3 | p-Value | | | | | | | | |
| Power (Running) (n = 20) | | | | | | | | | | | | | | | | | |
| P_la = 2 mmol/L (km/h) | 12.0 | ± | 1.5 | 11.6 | ± | 1.1 | 12.3 | ± | 1.7 | 0.30 | 12.3 | ± | 1.1 | 11.8 | ± | 1.8 | 0.49 |
| P_LT2 (km/h) | 13.3 | ± | 1.4 | 12.9 | ± | 1.0 | 13.6 | ± | 1.6 | 0.28 | 13.4 | ± | 1.2 | 13.3 | ± | 1.7 | 0.92 |
| P_LT1 (km/h) | 9.8 | ± | 1.1 | 9.4 | ± | 0.8 | 10.0 | ± | 1.2 | 0.22 | 9.7 | ± | 1.0 | 9.8 | ± | 1.3 | 0.85 |
| P_max (km/h) | 16.4 | ± | 1.7 | 16.1 | ± | 1.3 | 16.5 | ± | 2.0 | 0.55 | 16.6 | ± | 1.6 | 16.1 | ± | 1.9 | 0.58 |
| Power (Cycling) (n = 10) | | | | | | | | | | | | | | | | | |
| P_la = 2 mmol/L (W) | 217.9 | ± | 62.8 | 244.5 | ± | 52.9 | 178.0 | ± | 60.3 | 0.10 | 220.2 | ± | 69.3 | 215.6 | ± | 63.8 | 0.92 |
| P_LT2 (W) | 238.4 | ± | 65.9 | 263.3 | ± | 60.5 | 201.0 | ± | 62.0 | 0.09 | 245.8 | ± | 68.6 | 231.0 | ± | 70.3 | 0.92 |
| P_LT1 (W) | 150.3 | ± | 47.4 | 165.0 | ± | 46.5 | 128.3 | ± | 45.5 | 0.29 | 157.8 | ± | 47.6 | 142.8 | ± | 51.6 | 0.53 |
| P_max (W) | 326.5 | ± | 77.1 | 360.5 | ± | 65.3 | 275.5 | ± | 70.4 | 0.09 | 332.8 | ± | 74.1 | 320.2 | ± | 88.3 | 0.81 |
| P_max_rel (W/kg) | 4.8 | ± | 0.5 | 5.0 | ± | 0.5 | 4.4 | ± | 0.3 | 0.03 | 4.9 | ± | 0.7 | 4.6 | ± | 0.4 | 0.25 |

Abbreviations: P_la = 2 mmol/l: power on ergometer when having lactate value of 2 mmol/L; P_LT1: power at lactate threshold 1; P_LT2: power at lactate threshold 2; PBI: Papillary Bleeding Index; PSI: Periodontal Screening Index with PSI ≥ 3 indicating increased probing depths as a sign of probable periodontitis; P_max: maximum power on ergometer; P_max_rel: relative maximum power. Bold marks significant differences (p < 0.05).

4. Discussion

Overall, young athletes showed low mean gingival inflammation ($PBI = 0.48 \pm 0.29$) but, importantly, signs of periodontitis ($PSI \geq 3$) were present in 38% of the athletes. Group differences between athletes with lower or higher gingival inflammation were found for several blood parameters (hematocrit, hemoglobin, basophils, ESR1h, ESR2h, and urea), maximal aerobic capacity (VO_{2max}), and maximum load on the cycling ergometer. Athletes with signs of periodontitis differed in body composition (BMI, BFP), uric acid, and VO_{2max} .

One explanation for the differences between groups of different oral health status is that increased oral inflammation affects systemic parameters. Despite controversial discussion [8], various changes in blood values have been observed in periodontitis patients, including inflammation markers, cytokines, and changes in both white and red blood cell counts [25–29]. Furthermore, periodontal treatment that reduces local inflammation also reduces these systemic effects [7,30,31]. In the presented cohort of young athletes, the prevalence of signs of periodontitis was quite high (38%) in comparison to the overall population (1.7%) at this young age [32]. Moreover, this cohort of elite athletes showed a higher prevalence for signs of periodontitis than amateur athletes, despite similar oral health behavior [4]. Moderately elevated periodontal pockets (PSI score 3: none above 5.5 mm) were assessed. This low severity is in line with a previous study on periodontitis in footballers that reported overall mild periodontitis and a similar prevalence of periodontitis [5]. Even though the extent of systemic changes depends on the severity of periodontitis [28], increased CRP values have also been stated due to experimental gingivitis caused by cessation of oral hygiene [33]. Consequently, a systemic impact is possible, even for mild periodontitis and gingivitis. Regarding the gingival inflammation status in the present study, the PBI per papilla was below one (median: 0.42, IQR: 0.31;0.69), indicating mild or localized gingivitis.

Interestingly, the current study also revealed differences in the anthropometric data depending on periodontal status: individuals with probable signs of periodontitis showed higher BMI and BFP (Table 2). In contrast, another study could not reveal such differences between athletes, with and without periodontitis [5]. The values of BMI and BFP of the athletes were generally at a low level. For low BMI (18 to 22), a negative correlation between BMI and generalized aggressive periodontitis was already described [34] as well as in athletes, between BFP and periodontal probing depths [5]. In athletes with lower BMI and BFP, the phenomena of ‘Relative Energy Deficiency in Sport’ must be considered [35]. However, the results of the current study are inconclusive between the groups of gingival and periodontal inflammation: athletes with higher gingival inflammation showed lower BFP measured by skin folds (Table 2).

Some blood parameters showed significant differences: basophils, hematocrit, hemoglobin, ESR1, ESR2, urea, HDL cholesterol (by PBI), and uric acid (by PSI). The detected extensions were not of clinical relevance, as all investigated blood markers were within the reference ranges and the differences were small. As the direction of the group differences was inconsistent between the groups of gingival and periodontal inflammation and partly even in the same comparison (ESR1 and ESR 2), the significance of these differences is questionable in general. Nevertheless, the direction and extent of the revealed differences for uric acid, hemoglobin, and hematocrit would be in line with the results of a study in blood donors with increased probing depths compared to periodontally “healthy” ones [36]. In contrast to the stated difference in HDL cholesterol in the present study, experimental gingivitis did not lead to differences in cholesterol fractions [33].

Regarding the results of the performance tests, on the cycling ergometer, athletes with a lower level of signs of periodontal inflammation consistently reached higher power. Despite the small subgroup size, several trends for gingival inflammation became apparent and athletes with less gingival inflammation reached a significantly higher relative maximum power (Table 6). The revealed differences are relevant, especially as the subgroup is a homogeneous elite group from one sport discipline. Furthermore, in general, athletes with signs of periodontitis achieved lower VO_{2max} values (Table 2). These results are in line with the stated negative influence of periodontitis on physical fitness in other

population cohorts [9]. Athletes with higher oral inflammation could be compromised in their performance due to a systemic effect. In contrast, no impact of caries on the anaerobic capacity of athletes was found by another study [17]. However, this does not contradict a potential influence of oral inflammation as superficial caries generally have less systemic impact. The possibility of such systemic influence of oral health in athletes is underlined by potential associations between poor oral health and injuries [5,37,38].

Strengths and limitations: This explorative study was, to the best of the author's knowledge, the first published on possible associations between signs of periodontal inflammation and systemic parameters in competitive athletes. Including data from 85 athletes from the German national elite, perspective, or youth squads, allowed us to evaluate a considerable cohort. The limitation in athletes between 18 and 30 years indicates to include the typical age of elite athletes. With the resulting medium age of 21 years, this study presents the stage of young elite athletes. Moreover, a detailed description of the oral health status and oral health behavior of this cohort of elite athletes is available [4]. A major strength of the current study is the comprehensive number of available parameters, including blood parameters, echocardiographic parameters, as well as performance parameters. One limitation of the present study is the multiple statistical testing. Nevertheless, due to the explorative character, data were not adjusted [39]. Therefore, all statistical differences should be interpreted with caution. Overall, this applies to the performance and echocardiographic examinations, as only small subgroups could be analyzed. In addition, a potential selection bias must be considered, because it cannot be excluded that athletes with more severe signs of periodontal inflammation were more strongly affected and could not fulfill the squad levels for inclusion. In addition, the methods for the assessment of signs of periodontal inflammation must be discussed. The evaluated data originate from oral examinations that were part of the annual sports medical diagnostics and aimed to detect treatment need. Regarding the PSI, it must be considered that this screening index only indicates gingival inflammation and/or increased probing depths as a sign of probable periodontitis [23] and could also be caused by local swelling due to gingivitis. However, the stated prevalence of signs of periodontitis (38%) complies with the prevalence of a study with comprehensive periodontal examination, according to the current classification (41%, initial periodontitis, stage I, in all but two athletes) [5]. The current classification of periodontal disease (staging/grading matrix) [40] allows for the correct diagnosis with periodontitis. Nevertheless, these diagnoses are mainly based on attachment loss and may be in a stable status without inflammation [40]. The question of current periodontal inflammation and stability depends on periodontal probing depths and bleeding on probing (BOP) [40] but the BOP is not integrated in the basis diagnosis (stage/grade) of periodontitis. For the precise identification to periodontitis and/or periodontal inflammation, a complete periodontal chart (periodontal probing depths, clinical attachment loss for stage, and grade as well as BOP) would be necessary. The concept of the periodontal inflammation surface area (PISA) [41] could quantify the resulting inflammatory burden. These data were not available in the present study. This should be taken into account for interpretation of the presented data and for future studies. Nevertheless, despite not exactly identifying the diagnosis of periodontitis, the PSI identifies elevated periodontal probing depths in the case of full mouth and all-around-the-tooth examination [42]. Thus, it can detect current signs of periodontal inflammation (= inflammatory burden) and periodontal treatment need (PSI Score ≥ 3). Regarding the periodontal attachment loss, under- and, in young age groups, overestimation by the PSI have been discussed [43]. For gingival inflammation, such strict group definition (health vs. presence of inflammation) was not possible, as all athletes showed bleeding as a sign of gingivitis or periodontitis (no PSI score 0) [4]. The performed PBI is a gingivitis index that evaluates the gingival inflammation by the intensity of bleeding on probing at the interdental sites [23]. Generally, gingival inflammation as well as signs of periodontitis were only mild or localized. Due to the resulting small inflammation (PBI: median: 0.42, IQR: 0.31;0.69; PSI ≥ 3 in 38%, localized in 34% of them), the group size could still be too small for detecting these slight systemic effects. Further

limitations must be addressed regarding the compared subgroups. The group differences of gingival inflammation (PBI < 0.42 vs. PBI \geq 0.42) were small and might have limited the ability to assess the differences in the systemic effects. As, in addition to PSI score 1 to 2, score 3 could indicate the status of gingivitis due to localized swelling, the group division by PSI might not distinguish clearly enough between those athletes with and those without periodontal inflammation. A larger sample size as well as comprehensive periodontal examination might improve the identification of the small, but potentially important, systemic effects for both initial periodontitis and gingival inflammation. In addition, cohorts with more severe periodontal inflammation or experimental gingivitis are further interesting research possibilities. The blood parameters investigated in this study were those from routine medical tests due to the retrospective nature of the project. Thus, the available blood parameters are an unspecific part of the routine diagnostics. Even though, for periodontitis patients, some studies could reveal such differences [26,28,29], these parameters are probably not sensitive enough for such localized, mild inflammatory group differences. Furthermore, VO_{2max} was determined by spiroergometry in only less than half of the participants. The used formula for VO_{2max} in the others is based on age, body mass, and RHR. Nevertheless, it can be considered an appropriate estimation in case of missing exercise tests [22].

5. Conclusions

The present study supports the hypothesis for an influence of oral inflammation in athletes; body composition, blood, and performance test parameters differed slightly between athletes with different levels of signs of periodontal inflammation. A potential systemic impact of oral inflammation on athletic performance should be investigated.

This explorative study identifies some aspects for future research; prospective studies during a uniform exercise test with spiroergometry of all participants should be carried out. Blood analysis should include more sensitive inflammatory parameters, such as CRP and interleukins. As a marker for the oral status, the PISA and salivary biomarkers would be recommendable. A cohort with a higher level of inflammation burden could simplify the discrimination. Similarly, a larger sample size, based on an appropriate power calculation with consideration for the variability in outcome measures, will be important. Furthermore, an intervention study could prove the connection by showing the systemic effect of periodontal treatment in athletes.

Supplementary Materials: The following supporting information can be downloaded at: <https://www.mdpi.com/article/10.3390/jcm11175161/s1>, Table S1: Multivariate linear regression analysis of the influence of some blood parameters on gingival inflammation (PBI); Table S2: Echocardiographic parameters and their associations with periodontal health (PBI and PSI).

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3. Diskussion

Die sportmedizinische Betreuung von jungen Spitzensportlern unterscheidet sich in vielen Facetten von anderen medizinischen Disziplinen. Während in den meisten medizinischen Fachgebieten die kurative Medizin zur Behandlung im Mittelpunkt des ärztlichen Handelns steht, liegt der Fokus in der sportmedizinischen Tätigkeit (neben der Versorgung akuter Erkrankungen und Verletzungen) vor allem in der präventiven Versorgung [41]. Dabei geht es nicht nur darum, die Wahrscheinlichkeit für das Auftreten von Verletzungen und Überlastungsreaktionen durch die Ausübung der jeweiligen Sportart zu minimieren, sondern vor allem sportbedingte, langfristig auftretende Pathologien zu verhindern oder rechtzeitig zu erkennen. Nicht zuletzt ist die Verhinderung des sportbedingten plötzlichen Herztodes ein wesentlicher Aspekt der sportärztlichen Vorsorgeuntersuchung. Die Wirksamkeit dieser ärztlich präventiven Tätigkeit konnte bereits mehrfach international nachgewiesen werden [58, 60, 64-66]. Dabei beziehen sich die Mehrzahl aller veröffentlichten Daten auf Sportler unterschiedlichster Sportarten, Altersklassen und vor allem Leistungsklassen. Die Definition, ab wann eine Person als Sportler bzw. Leistungssportler angesehen werden kann, ist dabei uneinheitlich und bezogen auf das Kollektiv von Eliteathleten der jeweiligen Nationalkader vergleichsweise niederschwellig [102]. Dies macht die Betrachtung und Betreuung von Spitzensportlern auf der Basis gesicherter wissenschaftlicher Erkenntnisse herausfordernd, da Studien, die sich ausschließlich mit Eliteathleten auf internationalem Topniveau ihrer jeweiligen Sportart befinden, allein schon aufgrund der geringen Anzahl von vorhandenen Athleten begrenzt sind und somit auch immer mit dem Problem der begrenzten, wissenschaftlichen Aussagefähigkeit bei geringer Fallzahl der untersuchten Athleten umgehen müssen. Vor diesem Hintergrund ist es umso erstrebenswerter, in Zentren, die sich sportmedizinisch vorrangig mit Hochleistungssportlern ihrer jeweiligen Sportart beschäftigen, sportmedizinisch wissenschaftliche Untersuchungen in diesem elitären und speziellen Kollektiv durchzuführen. Darüber hinaus liegen, bezogen auf die Klientel der jüngeren Elite-Nachwuchssportler, noch wenige Erkenntnisse über sportbedingte Adaptationserscheinungen vor. Hinsichtlich der hohen körperlichen Anforderungen, die bereits in den Aufbau- und Anschlusstrainingsphasen an die heranwachsenden Elitesportler gestellt werden, ist eine umfangreiche Kenntnis der sportbedingten, physischen Veränderungen aller Organsysteme für eine möglichst effektive, präventive, sportmedizinische Betreuung dieser Athleten immanent. Auf der Grundlage dieser Anforderungen konnte in der ersten vorgestellten Arbeit dargestellt werden, inwieweit die Ausübung spezieller Ausdauersportarten, die zwar in ihren Anforderungen und ihrer Technik vergleichsweise ähnlich sind, zu unterschiedlichen kardiovaskulären Anpassungen führt. In einer multizentrischen, retrospektiven Analyse an Hochleistungssportlern wurden echokardiografische 2D Untersuchungen und Speckle Tracking Analysen sowie die leistungsphysiologischen Daten aus Spiroergometrien verglichen. Dabei

zeigten sich bei den Skilangläufern und Biathleten im Vergleich zu den Skibergsteigern ein höherer linksventrikulärer Massenindex und insbesondere ein größerer linker Vorhof. Der Nachweis eines erhöhten Vorhofvolumens bei Ausdauerhochleistungssportlern wurde bereits mehrfach in der Literatur beschrieben und wird von manchen Autoren als ein möglicher Risikofaktor im Kontext von langfristig ausgeübten, hochleistungssportlichen Ausdauertrainings in Bezug auf die Entwicklung atrialer Kardiomyopathien angesehen. So wird das Lebenszeitrisiko für das Auftreten von Vorhofflimmern bei Leistungssportlern im Vergleich zur Normalbevölkerung in einem Metareview ca. 2,5fach erhöht beschrieben [103]. Als ein möglicher zugrundeliegender Pathomechanismus wird dabei eine vermehrte atriale, myokardiale Fibrosierung diskutiert, die vermehrt durch die biatriale Dilatation der Vorhöfe bei Leistungssportlern begünstigt wird [104]. Die in der oben genannten Studie nachgewiesenen echokardiografischen und spiroergometrischen Unterschiede, insbesondere zwischen den Gruppen der Biathleten und Skilangläufer auf der einen und Skibergsteigern auf der anderen Seite, könnten dabei auf die zugrundeliegenden unterschiedlichen Trainingsmodalitäten und Trainingsumfänge zurückzuführen sein. Ein wesentlicher Aspekt in der Betrachtung der unterschiedlichen echokardiografischen Daten muss jedoch hinsichtlich der Basisdaten zum Untersuchungszeitpunkt konstatiert werden. Während die Gruppen der Skilangläufer und Biathleten ein vergleichbares Alter und Körpergewicht hatten, waren die Skibergsteiger jünger und leichter. Somit hatten die Skibergsteiger im Vergleich weniger Trainingsstunden im intensiven Ausdauertraining bezogen auf ihre Lebensspanne. Interessanterweise spiegelten sich die erhöhten Herzvolumina bei Skilangläufern und Biathleten nicht in einer erhöhten maximalen Sauerstoffaufnahme unter Belastung wieder. Lediglich die signifikant höheren Sauerstoffpulsmaximalwerte in diesen beiden Athletengruppen im Vergleich zu den Skibergsteigern könnte ein Hinweis auf eine bessere Ausdauerspitzenleistung darstellen und gegebenenfalls durch die ausgeprägtere kardiale Adaption erklärt werden. Allerdings muss klar festgestellt werden, dass die Ausdauerleistung eines Athleten und insbesondere die Sauerstoffaufnahme und der Sauerstoffpuls, neben der kardialen Anpassung, von vielen weiteren Faktoren beeinflusst wird (zum Beispiel Hämoglobinmasse, muskuläre Kapillarisation, muskulär mitochondriale Kapazität und vieles mehr) und auf kardialer Ebene ein variables dynamisches Schlagvolumen, funktionelle Rechts-Links-Shunts und andere Faktoren denkbare Einflussfaktoren darstellen. Des Weiteren können die genannten spiroergometrischen und echokardiografischen Ruhemesswerte nur bedingt eine Aussage zur sportlichen Leistungsbeurteilung liefern, da diese nur bedingten Einfluss auf die Leistung im Wettkampf bzw. unter maximalen Belastung haben. Dennoch könnten die beschriebenen kardialen Remodeling-Vorgänge zu einer besseren sportlichen Leistung durch ein verbessertes Herzzeitvolumen beitragen, da ein positiver Zusammenhang zwischen der Vorhofentleerungsfraktion und der maximalen Sauerstoffaufnahme [105] und der maximalen und Ruheherzleistung gezeigt werden konnte [106]. Letztlich kann jedoch

nicht ausgeschlossen werden, dass in der Gruppe der Skibergsteiger die physiologischen Veränderungen im Vergleich zu Skilangläufern und Biathleten andere Ursachen haben. Aufgrund der Tatsache, dass es sich bei dieser Sportart um eine vergleichsweise „junge“ Sportart handelt, welche 2026 erstmalig in das olympische Wettkampfprogramm aufgenommen wird, könnte eine Ursache der Unterschiede darin begründet liegen, dass die trainingswissenschaftlichen Methoden und die Betreuung ungleich weniger ausgereift sind. Umso wichtiger ist es für Sportmediziner, die sportmedizinische Tauglichkeitsuntersuchungen und Leistungsdiagnostiken durchführen, die Unterschiede im kardiovaskulären System zu kennen.

Unter dem Aspekt der hochspezifischen, trainingsbedingten Anpassungsvorgänge, die sich bereits bei jungen Eliteathleten zeigen, beschäftigte sich die zweite vorgestellte Arbeit mit dem belastungsinduzierten Blutdruckverhalten auf dem Ergometer.

Arterielle Hypertonie ist eine Erkrankung der westlichen Welt, die mit steigendem Lebensalter eine zunehmende Prävalenz erfährt [107]. Als einer der bekannten kardiovaskulären Risikofaktoren für Apoplex, arterielle Aneurysmen, Vorhofflimmern und Myokardinfarkt [108] ist die rechtzeitige Erkennung und Behandlung einer arteriellen Hypertonie ein wesentlicher Baustein der kardiovaskulären Prävention. Dabei ist zu beachten, dass der arterielle Blutdruck im Kindes- und Jugendalter deutlich niedriger liegt als im Erwachsenenalter und ausgehend von einem mittleren systolischen Blutdruck von 70mmHg im frühen Kindesalter kontinuierlich im Verlauf der Kindheit und Adoleszenz bis zum Erreichen des Erwachsenenalters ansteigt [109, 110]. In diesem Zusammenhang ist besonders relevant, dass offensichtlich der Blutdruck in der Kindheit eine hohe Korrelation mit dem Blutdruck im Erwachsenenalter aufweist [111]. Somit kann ein Monitoring des Blutdrucks in Ruhe und des Blutdruckverhaltens unter Belastung bei Kindern und Jugendlichen frühzeitig einen Hinweis auf das Entstehen einer arteriellen Hypertonie liefern. Inwiefern ein erhöhter arterieller Blutdruck unter Belastung bei jungen Leistungssportlern ebenfalls einen Hinweis auf die Entstehung einer arteriellen Hypertonie im späteren Erwachsenenalter gibt ist aufgrund fehlender Langzeitbeobachtungen jedoch nicht geklärt. Ein weiterer Grund dafür ist, dass obwohl die Belastungsuntersuchung auf dem Ergometer mit Ableitung eines 12-Kanal EKGs und der Erfassung des Blutdruckverhaltens bereits seit Jahrzehnten einen wesentlichen Teil der sportmedizinischen Vorsorgediagnostik darstellt, bisher keine spezifischen Angaben zur Beurteilung eines „normalen“ Belastungsblutdruckverlaufs im Kollektiv der jungen Elitesportler existierten. Obschon diese Normwerte für Erwachsene bereits seit vielen Jahren durch die verschiedenen Fachgesellschaften definiert wurden, sind diese Empfehlungen nicht einheitlich. So empfiehlt die Amerikanische Gesellschaft für Kardiologie (American Heart Association, AHA) die Definition eines erhöhten Belastungsblutdrucks bei einem systolischen Blutdruck >210 mmHg bei Männern und >190 mmHg bei Frauen und / oder einem diastolischen Blutdruck > 90 mmHg für

beide Geschlechter [112]. Demgegenüber wird durch die Europäische Gesellschaft für Kardiologie (European Society of Cardiology, ESC) eine Belastungshypertonie ab einem systolischen Blutdruck von >220 mmHg bei Männern und über 200 mmHg bei Frauen, und / oder einem diastolischen Blutdruck über 85 mmHg bei Männern und ab 80 bei Frauen definiert [113]. Schließlich lautet die Definition einer belastungsinduzierten Hypertonie durch die Amerikanische Gesellschaft für Sportmedizin (American College of Sports Medicine, ACSM) ab einem systolischen Spitzenblutdruck über 225 mmHg und / oder über 90 mmHg für den diastolischen Blutdruck für beide Geschlechter [114, 115]. Inwieweit diese Empfehlungen auch für adulte Leistungssportler anwendbar sind, wurde durch Keiler et. al. kürzlich untersucht [116]. Dabei zeigte sich, dass bei kardiovaskulär gesunden, jungen Leistungssportlern (mittleres Alter 21 Jahre) ohne vorbestehende arterielle Hypertonie die Prävalenz von erhöhtem Belastungsblutdruck bei der Anwendung der ESC- Empfehlungen am höchsten (19,6 %) und am niedrigsten bei der Anwendung der ACSM-Richtlinien (6,8 %) war [116]. Die Autoren schlossen daher, dass die Empfehlungen der oben genannten Gesellschaften zur Beurteilung des Blutdruckverhaltens unter Belastung nur eingeschränkt tauglich sind, um sie auf das Kollektiv von Leistungssportlern zu übertragen. Empfehlungen zur Beurteilung des belastungsinduzierten Blutdruckverhaltens für Kinder und Jugendliche sind im Gegensatz zu den Empfehlungen der AHA, ESC, und ACSM rar. Dieses Wissen ist aus Sicht des Autors jedoch unabdingbar, um eine solche Untersuchung zum einen sicher durchzuführen und zum anderen hochnormale Blutdruckverläufe von pathologischen Zuständen unterscheiden zu können. Nach Kenntnis des Autors gab es zum Zeitpunkt der Schrifterstellung lediglich acht wissenschaftliche Veröffentlichungen, die sich mit der Beschreibung des belastungsinduzierten Blutdruckverhaltens bei Kindern und Jugendlichen auseinandergesetzt haben [117-124]. Lediglich eine dieser Arbeiten beschäftigte sich dabei mit dem belastungsinduzierten Blutdruckverhalten bei Athleten und Athletinnen [124], während die anderen Arbeiten Kinder und Jugendliche ohne Bezug zu regelmäßigem sportlichen Training untersuchten. Während in der Arbeit von Szmigielska et. al. zwar bisher einmalig der Fokus auf junge Sportlerinnen und Sportler gelegt wurde, waren diese bezüglich ihres Trainingsvolumens mit durchschnittlich 7,6 Trainingsstunden pro Woche als moderat leistungssportlich aktiv zu bezeichnen. Somit sind in der dargestellten Veröffentlichung dieser Schrift erstmalig Daten von Kindern und Jugendlichen präsentiert, die einem hochleistungssportlichen Trainingskontext mit durchschnittlich 14h Training pro Woche zugeordnet werden können. Im Vergleich der Daten von Szmigielska et. al. zeigt sich dabei, dass in den eigenen Untersuchungen an Leistungssportlern die maximalen systolischen Blutdruckwerte unter Belastung deutlich höher lagen. Im Unterschied zur Arbeit von Szmigielska et. al. wurden in der eigenen Studie zusätzlich vollständige echokardiografische Untersuchungen der Athleten und Athletinnen mit einbezogen. Mit der Darstellung von altersadaptierten Perzentilen des maximalen Belastungsblutdrucks (und echokardiografischer Werte) wurden nun die Voraussetzungen geschaffen

in der Gruppe der kindlichen und jugendlichen Leistungssportler eine Beurteilung des Blutdruckverhaltens auf dem Ergometer zu gewährleisten. Inwieweit jedoch ein erhöhter maximaler belastungsinduzierter Blutdruck in diesem speziellen Kollektiv junger Sportler auch mittel- oder langfristig zu einer manifesten arteriellen Hypertonie führt, kann wie bereits weiter oben diskutiert, nicht abschließend beurteilt werden, wenngleich ein Analogieschluss aus anderen Kollektiven (Erwachsene) naheliegt. Limitierend ist zudem zu beachten, dass in der vorgestellten Arbeit die Blutdruckperzentilen zwar auf das Alter und Geschlecht der Sportler bezogen wurden, der Einfluss der jeweiligen Körpergröße jedoch nicht berücksichtigt wurde. Von dieser ist jedoch aus Untersuchungen an nicht sporttreibenden Kindern und insbesondere Adoleszenten bekannt, dass diese einen Einfluss auf das Blutdruckverhalten hat[125]. Somit könnte dies zu einer Fehlinterpretation des Belastungsblutdrucks führen. Ebenso könnte eine Gruppierung adoleszenter Sportler nach ihrem Alter und nicht nach ihrer körperlichen Reife eine falsche Einschätzung des Belastungsblutdrucks bedingen. Dennoch erlaubt die nunmehr geschaffene Kategorisierung die Beurteilung eines altersgerechten Blutdruckverhaltens und gestattet dem beurteilenden Sportmediziner die Fokussierung weiterführender Diagnostik oder Folgebeobachtungen auf einzelne Sportler mit einem potentiell erhöhten kardiovaskulären Risiko.

Vor der Beurteilung und Analyse der ergometrischen Belastungsuntersuchung bei jugendlichen Sportlern ist die Erfassung und Interpretation eines 12-Kanal Elektrokardiogramm essentieller Bestandteil jeder sportmedizinischen Tauglichkeitsuntersuchung in Deutschland. Während die Erkennung und Interpretation von Herzrhythmusstörungen für den begutachtenden Arzt in der Regel keine Herausforderung darstellen dürfte, bereitet die Interpretation der Repolarisationsphase im Herzzyklus des Ruhe-EKG mitunter größere Schwierigkeiten. Diese ist jedoch vor dem Hintergrund der Erkennung von genetischen Arrhythmiesyndromen zunehmend in den Fokus der Kardiologie gerückt [126-128]. Zwar wurden bereits unterschiedliche pathologische Repolarisationsphänomene beschrieben, die auf eine zugrundeliegende Pathologie weisen, jedoch bleibt die Erkennung dieser Repolarisationsstörungen weiterhin eine Herausforderung für den behandelnden Arzt. Dies liegt daran, dass die Repolarisationsphase des Herzens zahlreichen Einflüssen unterliegt, die unter anderem durch den Einfluss des autonomen Nervensystems begründet sind. Die Unterscheidung von physiologischen Veränderungen von potentiell zum plötzlichen Herztod führenden erblichen Arrhythmiesyndromen ist jedoch hinsichtlich der begutachtenden Tätigkeit des behandelnden Arztes von fundamentaler Bedeutung. Nochmals herausfordernder wird diese in der Analyse von Ruhe-EKGs bei Kindern und Jugendlichen, da diesbezüglich weniger Daten vorhanden sind und zudem die oben genannten Einflüsse des autonomen Nervensystems bei Kindern ausgeprägter sind als bei Erwachsenen. Des Weiteren können die hormonellen Veränderungen in der Pubertätsphase zusätzlich einen Einfluss auf das kardiale Repolarisationsverhalten nehmen [129]. Während mittlerweile viele

Studien publiziert wurden, die sich mit dem kardialen Repolarisationsverhalten bei normalen Kindern beschäftigen und somit eine Einschätzung zu deren Normalverteilung erlauben, ist bisher noch selten untersucht worden inwieweit leistungssportliche Aktivität oder Adipositas einen Effekt auf Selbiges nehmen. In der weiter oben vorgestellten eigenen Arbeit zur Betrachtung der Veränderungen im Ruhe-EKG bei normalgewichtigen, adipösen und leistungssporttreibenden Kindern konnten zwar insgesamt keine relevant pathologischen Auffälligkeiten nachgewiesen werden, jedoch wurde insbesondere in der Betrachtung der elektrischen Repolarisationsphase bei adipösen Kindern eine signifikant längere QTc Zeit als bei normalgewichtigen und sporttreibenden Kindern nachgewiesen. Dabei wiesen die sporttreibenden Kinder die kürzesten QTc Zeiten auf. Dieser Trend zu kürzeren QTc Zeiten blieb auch bestehen, wenn statt der Bazett Formel (QT/VR) zur Frequenzkorrektur der QT-Zeit die Formel nach Hodges angewendet wurde. Diese sollte gemäß Literatur bei Patienten mit einer Herzfrequenz von unter 50/min Anwendung finden [130, 131], was insbesondere bei Ausdauersport treibenden Jugendlichen häufiger der Fall ist. In der Betrachtung der Zeiten von Spitze der T-Welle bis zum Ende der T-Welle (time peak to end, TPE) zeigten sich bei Sportlern signifikant niedrigere Werte als bei normgewichtigen und adipösen Kindern. Diese waren zwar statistisch signifikant, jedoch klinisch wahrscheinlich ohne Relevanz, da sich die Unterschiede nur in wenigen Millisekunden ausdrückten. Eine abschließende Beurteilung dieser Auffälligkeit hinsichtlich eines erhöhten Arrhythmierisikos ist jedoch aufgrund fehlender Verlaufsuntersuchungen nicht möglich.

Im Vergleich unterschiedlicher Sportartengruppen fand sich in der Analyse der QTc Zeiten bei Ausdauer- und Sprint-/Kraftsportlern ein Trend hinsichtlich der längeren QTc Zeiten in der Gruppe der Ausdauersportler, der nach Frequenzkorrektur nur in der Berechnung der QTc nach der Hodges Formel erhalten blieb. Ein Unterschied in den TPE Zeiten war hier nicht nachweisbar. Neben der Tatsache, dass sich in keiner der drei untersuchten Probandengruppen pathologische Veränderungen im Repolarisationsverhalten zeigten, konnten dennoch entscheidende Unterschiede im Vergleich zu publizierten Daten von Erwachsenen festgestellt werden. Im Unterschied zur Literatur bei erwachsenen Spitzensportlern konnten in unserer Untersuchung keine Verlängerung der QT oder QTc Zeit im Vergleich zu normgewichtigen Personen nachgewiesen werden [98, 100, 101]. Dabei ist wichtig zu betonen, dass unabhängig von der verwendeten Formel zur Frequenzkorrektur der QT-Zeit, die Ergebnisse konsistent blieben und nicht durch die bei sporttreibenden Kindern potentiell niedrigere Herzfrequenz beeinflusst wurden. Somit könnte die Vermutung von D'Ascenzi zutreffen, dass der Unterschied im Repolarisationsverhalten von Kindern und Jugendlichen im Vergleich zu Erwachsenen durch einen Einfluss der Pubertät auf die Verlängerung der QT-Zeit zustande kommen könnte [39]. Ebenso ist es denkbar, dass die beschriebenen sportbedingten Veränderungen des QT-Intervalls erst im Laufe eines prolongierten Anpassungsprozesses an das sportliche Training auftreten und daher im Kinder- und Jugendalter noch nicht nachweisbar sind.

Interessanterweise konnten in unserer Studie Daten aus früheren Untersuchungen bestätigt werden, wonach die längsten QTc Zeiten in der Gruppe der adipösen Kinder gefunden wurden [129, 132]. Auf der Grundlage einer angenommenen erhöhten hormonellen Aktivität des Fettgewebes mit erhöhten Östrogenspiegeln bei adipösen Kindern im Vergleich zu normgewichtigen Kindern, wurde postuliert, dass die erhöhten Östrogenspiegel im Zusammenspiel mit einem erhöhten parasymphatischen Tonus zu einer Verlängerung des QT-Intervalls führen. Der ursächlich angenommene, erhöhte parasymphatische Tonus, der als einer der Faktoren für die QTc Intervallverlängerung angenommen wurde, scheint somit jedoch einen geringen Einfluss auf diese Veränderung zu haben, da dieser aufgrund des ausgeübten leistungssportlichen Trainings bei sporttreibenden Kindern ebenso stark ausgeprägt sein dürfte und nicht zu einer Verlängerung des QT-Intervalls führte. Einschränkend ist jedoch festzuhalten, dass die nachgewiesenen Unterschiede der QTc Zeit nur wenige Millisekunden betragen. Inwieweit dies Unterschiede auf ein erhöhtes Arrhythmierisiko in der Gruppe der adipösen Kinder schließen lässt, kann auch hier nicht abschließend beurteilt werden.

Nicht zuletzt gilt es auch in dem Kollektiv der Jugendlichen und der jungen, erwachsenen Athleten zusätzliche sportmedizinisch relevante Aspekte zu beleuchten, die typischerweise nicht im Fokus der sportmedizinischen Betrachtung stehen. Am Beispiel der Interaktion von oraler Gesundheit und systemischer Inflammation im Zusammenhang mit sportlicher Höchstleistung konnte in dieser Schrift gezeigt werden, dass dieser bisher wenig beachtete Aspekt ein weiteres wichtiges Feld der sportbezogenen Prävention und Optimierung der physiologischen Leistungsentwicklung darstellt.

Um jedoch eine Einschätzung der Relevanz des Einflusses oraler Gesundheit auf die sportliche Leistungsfähigkeit abgeben zu können, ist es wichtig zunächst einen tieferen Einblick in den Zustand des Mundgesundheitszustandes bei Leistungssportlern im Vergleich zur Normalbevölkerung und Freizeitsportlern zu bekommen und mögliche Einflussfaktoren zu identifizieren, die eventuell abweichende Untersuchungsergebnisse erklären könnten. Auf der Grundlage bisher publizierter Daten muss angenommen werden, dass Sportler eine höhere Kariesprävalenz [92, 133, 134] und häufiger parodontale Veränderungen haben [91], als dies in der Normalbevölkerung der Fall ist. Als mögliche Ursachen werden dabei unterschiedlichste Faktoren diskutiert. So wird angenommen, dass die erhöhte Aufnahme von Kohlenhydraten, die zum Teil in flüssiger Form oder als Gel eingenommen werden, eine höhere Kariesprävalenz bei Sportlern begünstigt [135]. Des Weiteren werden als mögliche Risikofaktoren eine sportbedingte Veränderung der Speichelzusammensetzung [134] und ein verändertes orales Mikrobiom [89] diskutiert. Letztlich sind sich jedoch zahlreiche Autoren einig, dass insbesondere ein eingeschränktes Mundgesundheitsverhalten für die erhöhte Prävalenz von dentalen und parodontalen Pathologien anzunehmen ist [136, 137].

Um den möglichen Ursachen einer eingeschränkten Mundgesundheit bei Elitesportlern nachzugehen und den Einfluss des Mundgesundheitsverhaltens aus diese zu beleuchten, wurde in der vierten vorgestellten Arbeit zunächst der zahnmedizinische Zustand von Elitesportlern und Amateursportlern erfasst, untereinander verglichen und dies in den Kontext des individuellen Mundgesundheitsverhaltens gestellt. Bisherige Studien zur Mundgesundheit bei Sportlern wurden mehrheitlich in Ländern durchgeführt, die ein im Vergleich zu Deutschland unterschiedliches Gesundheitssystem haben und deren Daten damit nicht zwangsläufig auf Deutschland übertragen werden können [135]. In einer der vorliegenden Studie unserer Arbeitsgruppe zeigte sich in den untersuchten Gruppen nun vorrangig ein Unterschied bezüglich Vorliegen oraler Entzündungszeichen (vornehmlich parodontal), ein höherer Anteil von cranio-mandibulären Dysfunktionen (CMD) sowie kariöser Zähne. Diese waren bei Leistungssportlern im Vergleich zu Amateursportlern vermehrt festzustellen, wobei dies interessanterweise nicht auf ein unterschiedliches orales Präventionsverhalten zurückzuführen war. Im Vergleich zu bisher veröffentlichten Daten war die Häufigkeit von Karies bei Leistungssportlern in unserer Studie jedoch bei vergleichbarem Mundgesundheitsverhalten geringer [133] während parodontale Veränderungen häufiger waren [87]. Insbesondere im Vergleich zu einer deutschen Studie an Triathleten war die Anzahl kariöser Zähne deutlich weniger [134], was möglicherweise darauf zurückzuführen ist, dass die Athleten in unserer Studie im Durchschnitt jünger (Alter: 20,6 versus 37 Jahre) waren [134] und sich damit ein Hinweis auf die Altersabhängigkeit dieses Parameters bei Leistungssportlern ergibt. Inwiefern sportbedingte Ursachen einen Einfluss auf das vermehrte Auftreten von kariösen Veränderungen im Verlauf der leistungssportlichen Karriere nehmen kann nur spekuliert werden. Denkbar wäre ein erhöhter Konsum von zucker- und säurehaltigen Sportdrinks als ein möglicher Einflussfaktor, weswegen es im höheren Alter zu vermehrten kariösen Zuständen kommt. Dies ist derzeit allerdings nicht belegt. In Bezug auf erosive Veränderungen der Zahnhartsubstanz, welche in unserem Studienklientel in geringerem Grad ausgeprägt war, zeigte sich eine Übereinstimmung mit bereits publizierten Daten bei Sportlern vergleichbaren Alters [138], während Erosionen der Zahnoberfläche in der bereits genannten Studie an Triathleten höheren Alters einen höheren Score Wert aufzeigten [134] und somit die oben genannte Annahme unterstützt. Während das Vorliegen kariöser Veränderungen der Zähne bei Sportlern in unseren Untersuchungsgruppen als insgesamt gering ausgeprägt bezeichnet werden können, zeigte sich ein etwas anderes Bild in der Untersuchung des Zahnhalteapparates. Zwar waren auch hier die Biofilmmakkumulation (=Plaqueablagerung) und gingivale Entzündung insgesamt geringfügig ausgeprägt, jedoch fanden sich bereits initiale parodontal auffällige Befunde mit einem auffälligem PSI-Score (Periodontal Screening Index = PSI) bei 40 % der Leistungssportler, was im Vergleich zu anderen bisher publizierten Daten (5-21%)[91, 92, 135] als deutlich erhöht gewertet werden muss und auch im Vergleich zur Kontrollgruppe der Amateursportler (12 %) deutlich häufiger festzustellen war. Da die

genannten Veränderungen einen Hinweis auf etablierte Gingivitis und/oder bereits initiale Parodontitis geben, ergibt sich daraus ein signifikanter parodontaler Behandlungsbedarf in der Gruppe der Leistungssportler. Als mögliche Ursache für das erhöhte Vorkommen parodontaler Veränderungen kann ein unzureichendes Mundgesundheitsverhalten der Leistungssportler angenommen werden: während sie angeben täglich die Zähne mit einer Zahnbürste zu putzen, wird die Anwendung von Interdentalreinigungsmaßnahmen (Zahnseide, Interdentalbürsten etc.) allerdings nur von weniger als der Hälfte der Sportler angegeben; zudem geben 20% an nicht regelmäßig zahnärztliche Kontrolluntersuchung vornehmen zu lassen. Ebenso zeigte sich ein reziproker Zusammenhang zwischen parodontaler Veränderung und der Durchführung professioneller Zahnreinigungen bei Sportlern, was die Bedeutung dieser präventiven Maßnahme in diesem Kollektiv unterstreicht und den Bedarf an einer spezieller, präventionsorientierten, zahnmedizinischer Beratung bei Sportlern hervorhebt. Interessanterweise lassen sich zusammenfassend aus den erhobenen Daten der dargestellten Arbeit zum Mundgesundheitsverhalten und der oralen Hygiene bei Amateur- und Leistungssportlern die Unterschiede insbesondere in Bezug auf den parodontalen Behandlungsbedarf bei Leistungssportlern nicht fundiert erklären, sodass angenommen werden muss, dass es einen weiteren, bisher nicht eindeutig geklärten Einflussfaktor auf die parodontale Gesundheit, möglicherweise durch eine sportbedingte erhöhte orale Inflammationsneigung, bei Leistungssportlern gibt. Einschränkend muss jedoch festgehalten werden, dass in der durchgeführten Studie in der Gruppe der Leistungssportler ausschließlich Ausdauersportler eingeschlossen waren, während die Gruppe der Amateursportler zusätzlich Kraft und Mannschaftsspielsportler beinhaltete, beziehungsweise diese mitunter unterschiedlichen Sportarten ausübten und somit ein sportartenspezifischer Einfluss nicht ausgeschlossen werden kann.

Da in der vierten vorgestellten Untersuchung ein deutlich erhöhter parodontaler Behandlungsbedarf in der Gruppe der Leistungssportler im Vergleich zu Amateursportlern bei vergleichbarem Mundgesundheitsverhalten aufgezeigt werden konnte, lag es nahe anzunehmen, dass diese parodontalen Erkrankungszeichen möglicherweise durch leistungssportlich induzierte Inflammation bedingt wird. Ebenso ist ein bidirektionaler Zusammenhang denkbar, wobei ebenfalls das erhöhte orale Inflammationsgeschehen Auswirkungen auf das systemische Inflammationsgeschehen und letztlich auf die Trainingsreizwirksamkeit und somit Leistungsfähigkeit haben könnte. Inwiefern jedoch eine erhöhte orale Inflammation, wie sie einer parodontalen Erkrankung zugrunde liegt und deren systemische Wirkung bereits vielfältig im Kontext verschiedener Erkrankungen wie zum Beispiel Diabetes mellitus bekannt ist [139] und Einfluss auf die körperliche Leistungsfähigkeit bei Spitzensportlern hat, ist bisher unklar.

Dass es eine Kausalität zwischen oraler Inflammation, wie sie der Parodontitis zugrunde liegt, auf die systemische Inflammationsaktivität gibt, konnte bereits aufgezeigt werden [139]. Im Zusammenhang mit körperlicher Leistung könnte dies wie bereits beschrieben jedoch wechselseitig bedingt sein, da zum einen körperliche Aktivität im Sport zu einer erhöhten systemischen Inflammation [81] und Abnahme der Immunglobulin A Spiegel im Speichel führt [83], zum anderen eine schlechte Mundgesundheit als Ursache für eine verminderte körperliche Leistungsfähigkeit beschrieben wurde [140]. Dabei ist anzumerken, dass der Zusammenhang zwischen körperlicher Leistungsfähigkeit und oraler Gesundheit bei Leistungssportlern bisher nur sehr spärlich untersucht wurde. Lediglich in einer Studie an Ruderern wurde bisher die anaerobe Kapazität im Zusammenhang mit Kariesprävalenz beschrieben [93]. Ähnlich der bereits zuvor vorgestellten Arbeit fand sich auch in dieser Studie bei insgesamt niedriger gingivaler Entzündungslast bereits erhöhte Anzeichen einer initialen Parodontitis im auffällig erhöhtem PSI-Scores ≥ 3 (38%) (Peridontaler Entzündungscore, PSI. Die PSI Scores waren vor allem auch im Vergleich zu gleichaltrigen Nichtsportlern, bei welchen dieser Wert in der Literatur mit 1,7 % angegeben wird, deutlich erhöht [141], wobei die Schwere der Parodontitis in der Gruppe der Leistungssportler als eher gering ausgeprägt angesehen werden muss. Bezogen auf die Betrachtung der systemischen Auswirkungen einer vorliegenden Parodontitis (parodontalen Behandlungsbedürftigkeit) auf diverse Blutparameter, spiegelt sich dies in der Gruppe mit erhöhten PSI Werte in einer Erhöhung der Blutsenkungsgeschwindigkeit(BSG). In Bezug auf die Leistungsfähigkeit fand sich in dieser Gruppe im Vergleich zu den Athleten mit niedrigem PSI Wert auch eine geringere maximale Leistung auf dem Ergometer und niedrigerer maximaler Sauerstoffaufnahme, welche jedoch das Signifikanzniveau nicht erreichte. Hierbei ist jedoch zu beachten, dass in der dargestellten Untersuchung die gingivale Inflammation bei allen Sportlern insgesamt niedrig war. Somit ist auch weiterhin nicht auszuschließen, dass sich eine höhergradige entzündliche Veränderung des Zahnhalteapparates, wie bei einer schweren Parodontitis, sich bei Sportlern negativ auf die Leistung auswirken kann. Da die Teilnahme an den sportzahnmedizinischen Untersuchungen auf Freiwilligkeit beruhte und die Untersuchungen nicht verblindet waren, kann der Einfluss eines Selektions- oder Verblindungsfehlers in dieser Studie nicht ausgeschlossen werden. Ebenso ist die Generalisierung der getroffenen Aussagen auf die Gesamtheit aller Spitzensportler eingeschränkt, da in unseren Studien in den Untersuchungsgruppen der Leistungssportler vorrangig Ausdauersportler eingeschlossen wurden. Inwieweit eine höhergradige orale Entzündungslast einen solch ausgeprägten Einfluss auf die körperliche Leistungsfähigkeit hat, dass ein Sportler aufgrund dessen erst gar nicht die Voraussetzungen zur Aufnahme in eine Auswahlmannschaft erreicht, bleibt spekulativ.

Schlussendlich bleibt zu resümieren, dass wissenschaftliche Untersuchungen, die an einer sehr selektiven Zielgruppe wie Hochleistungsathleten durchgeführt werden, fast immer der Problematik der kleinen Fallzahl unterliegen, da naturgemäß nur wenige Athleten den Sprung in den Elitebereich

ihrer Sportart schaffen. Somit ergeben sich zwangsläufig fast immer Einschränkungen hinsichtlich der statistischen Power, der möglichen statistischen Verzerrung und der eingeschränkten Generalisierbarkeit. Dem entgegen ist jedoch anzumerken, dass die vorgestellten Untersuchungen an Elitesportlern (fast) ausnahmslos am Institut für Angewandte Trainingswissenschaft in Leipzig durchgeführt wurden (lediglich die Untersuchungen der Skibergsteiger fand in der Universitätsklinik Erlangen-Nürnberg statt), in dessen sportmedizinischer Abteilung ausschließlich und in sehr hoher Fallzahl - bezogen auf aktive Leistungssportler verschiedener Bundeskaderzugehörigkeit - Elitesportler betreut werden. Somit konnte im Vergleich zu vielen anderen Studien sichergestellt werden, tatsächlich die Topsportler ihrer jeweiligen Sportart in vergleichsweise hoher Fallzahl zu untersuchen.

4. Zusammenfassung

Die vorgelegte Habilitationsschrift befasst sich thematisch mit der Relevanz und Notwendigkeit sportmedizinischer, präventiver Diagnostik insbesondere junger Spitzensportler. Während in der Literatur umfangreiche Arbeiten zur präventiven Diagnostik von Erwachsenen und Sportlern im Allgemeinen vorliegen, liegt hier der Fokus auf der speziellen Klientel der Elitesportler ihrer jeweiligen Sportart bzw. auf den heranwachsenden Spitzensportlern. Insbesondere letztere gehören zu der Gruppe von Spitzensportlern, die in der Vergangenheit aus wissenschaftlicher Sicht weniger in Bezug auf sportmedizinisch präventive Diagnostik berücksichtigt wurden. Um eine effektive präventive Betreuung dieser Athleten gewährleisten zu können, ist es jedoch wichtig, die physischen Veränderungen des gesamten Organismus im Zusammenhang mit hohen körperlichen Anforderungen zu verstehen. Mit dem Fokus auf weniger beachtete Sportarten (hier beispielsweise Skibergsteigen), Kinder und jugendliche Spitzenathleten sowie den bisher eher als Randbereiche der sportmedizinischen Vorsorge diagnostik wahrgenommen Themen (hier die orale Gesundheit, gingivale Entzündungsreaktionen und deren Auswirkungen auf die sportliche Leistungsfähigkeit) bemüht sich diese Arbeit, das Wissen und Verständnis rund um den weiten Komplex der sportmedizinischen Vorsorgeuntersuchungen zu erweitern.

Dazu konnten in der vergleichenden Arbeit leistungsdiagnostischer und echokardiografischer Daten bei 31 Weltklasse-Wintersportlern (Biathleten, Skilangläufern und Skibergsteigern) signifikante echokardiografische Unterschiede zwischen den teilnehmenden Disziplinen festgestellt werden. Bei exzellenten Leistungsdaten aller Athleten zeigten sich signifikante Unterschiede zwischen den Skilangläufern und Biathleten einerseits und den Skibergsteigern andererseits. Um validiert Belastungsuntersuchungen auf dem Ergometer bei Kindern und jugendlichen Spitzensportlern durchzuführen, lagen bisher keine ausreichenden Erkenntnisse zum belastungsinduzierten Blutdruckverhalten dieser trainierten Kinder und Adoleszenten vor. Altersangepasste Perzentilen wurden daher in einer Arbeit für den maximalen Belastungsblutdruck erstellt, um eine Normbasis für begutachtende Ärzte zu schaffen. Ebenso wurde bisher nur spekuliert, ob ein erhöhter parasympathischer Einfluss, wie er bei spitzensporttreibenden Kindern angenommen wird, zu ähnlichen EKG-Veränderungen führt wie bei adipösen Kindern, bei denen als eine mögliche Ursache gleichfalls ein erhöhter parasympathischer Tonus angenommen wird. Obwohl weder bei normalgewichtigen, adipösen oder Leistungssporttreibenden Kindern relevant pathologische Befunde festgestellt wurden, zeigten die sporttreibenden Kinder im Gegensatz zu adipösen Kindern eine signifikant kürzere QTc-Zeit. Letztlich wurde in zwei weiteren Arbeiten die Zahngesundheit und individuelles orales Gesundheitsverhalten sowie die Interaktion von oraler Gesundheit und systemischer Entzündung in Bezug auf sportliche Leistung bei Leistungssportlern untersucht. Es zeigte

sich, dass dieser Aspekt wichtig für die Prävention und Optimierung der Leistungsentwicklung ist. Eine vermehrte Entzündung in der Mundhöhle hatte jedoch keinen signifikanten Einfluss auf die individuelle Leistungsfähigkeit. Systemische Entzündungsprozesse standen nicht im Zusammenhang mit geringen oralen Entzündungen bei den untersuchten Sportlern.

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7. Eidesstattliche Versicherung

Erklärung

§ 4 Abs. 3 (k) der HabOMed der Charité

Hiermit erkläre ich, dass

- weder früher noch gleichzeitig ein Habilitationsverfahren durchgeführt oder angemeldet wurde,
- die vorgelegte Habilitationsschrift ohne fremde Hilfe verfasst, die beschriebenen Ergebnisse selbst gewonnen sowie die verwendeten Hilfsmittel, die Zusammenarbeit mit anderen Wissenschaftlern/Wissenschaftlerinnen und mit technischen Hilfskräften sowie die verwendete Literatur vollständig in der Habilitationsschrift angegeben wurden,
- mir die geltende Habilitationsordnung bekannt ist.

Ich erkläre ferner, dass mir die Satzung der Charité – Universitätsmedizin Berlin zur Sicherung Guter Wissenschaftlicher Praxis bekannt ist und ich mich zur Einhaltung dieser Satzung verpflichte.

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Datum

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Jan C. Wüstenfeld