TESIS DOCTORAL

APORTACIÓN DE LOS PUNTOS GATILLOS MIOFASCIALES Y LOS PROCESOS DE SENSIBILIZACIÓN DOLOROSA EN EL DOLOR DE HOMBRO

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AMPARO HIDALGO LOZANO
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DEPARTAMENTO DE FISIOTERAPIA

E.U. CIENCIAS DE LA SALUD

UNIVERSIDAD DE GRANADA

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Aportación de los puntos gatillo miofasciales y los procesos de sensibilización dolorosa en el dolor de hombro

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Que la Tesis Doctoral titulada *Aportación de los puntos gatillo miofasciales y los procesos de sensibilización dolorosa en el dolor de hombro*, que presenta Dª. AMPARO HIDALGO LOZANO al superior juicio del tribunal que designa la Universidad de Granada, ha sido realizada bajo mi dirección durante los años 2008-2011, siendo expresión de la capacidad técnica e interpretativa de su autora en condiciones que le hacen merecedora del Título de Doctora, siempre y cuando así lo considere el citado Tribunal.

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CONTENIDOS

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- **Hidalgo-Lozano A**, Fernández-de-las-Peñas C, Díaz-Rodríguez L, González-Iglesias J, Palacios-Ceña D, Arroyo-Morales M. Changes in pain and pressure pain sensitivity after manual treatment of active trigger points in patients with unilateral shoulder impingement: A case series.
- **Hidalgo-Lozano A,** Fernández-de-las-Peñas C, Calderón-Soto C, Domingo-Cámara A, Pascal M, Arroyo-Morales M. Elite swimmers with and without shoulder impingement: mechanical hyperalgesia and trigger point in neck-shoulder muscles.
- **Hidalgo-Lozano A**, Fernández-de-las-Peñas C, Alonso-Blanco C, Ge HY, Arendt-Nielsen L, Arroyo-Morales M. Muscle trigger points and pressure pain hyperalgesia in the shoulder muscles in patients with unilateral shoulder impingement: a blinded, controlled study.
- **Hidalgo-Lozano A**, Calderón-Soto C, Domingo-Cámara A, Fernández-de-las-Peñas C, Pascal M, Arroyo-Morales M. Elite swimmers with unilateral shoulder pain exhibit bilateral higher cervical muscle activity during a functional upper limb task.

ABREVIATURAS

TrPs	Puntos gatillo
ATP	Adenosín trifosfato
EMG	Electromiografía
SCM	Esternocleidomastoideo
UP	Trapecio superior
SCL	Escaleno anterior
PPT	Umbral doloroso a la presión

INTRODUCCIÓN

El dolor de hombro es un problema de salud muy común, que se presenta en forma de distintas patologías, causadas por diversos factores; y caracterizado por asociarse a una gran cantidad de pacientes y por tanto, a un alto coste social[1]. Como ejemplo, en el año 2000, el coste directo para el tratamiento de las afecciones de hombro en EEUU fue de siete billones de dólares[2].

Este síntoma, que a menudo persiste o recurre, tiene una prevalencia durante el primer año del 20-50%, en función de las condiciones y los rasgos sociodemográficos. En cuanto a la incidencia de las afecciones de hombro, se registran de 7 a 25 por cada 1000 consultas de medicina general[3]. Sin embargo, los mecanismos patofisiológicos que hay bajo el dolor de hombro no han sido clarificados en la actualidad[4]. Existen evidencias respecto a este déficit de conocimiento sobre la etiología de este síntoma, aunque podemos apuntar que el síndrome subacromial es una de las principales causas potenciales de dolor de hombro, siendo el juicio diagnóstico más prevalente (13%)[5].

La etiología del síndrome subacromial tampoco está completamente desarrollada ni esclarecida, pero sí que hay evidencias que muestran la presencia de desequilibrios musculares en el hombro, como factor potencial relacionado con este síndrome[6]. Diferentes estudios han demostrado la presencia de estos desequilibrios musculares en la región del hombro, que cursan con dolor, y que sugieren que los puntos gatillo pueden jugar un papel relevante en el síndrome subacromial del hombro[7].

Puntos gatillo

La importancia clínica de los puntos gatillo miofasciales para los diferentes profesionales ha sido descrita en la literatura por acupuntores, anestesiólogos, especialistas en dolor crónico, dentistas, médicos de familia, ginecólogos, neurólogos, enfermeros, traumatólogos, pediatras, fisioterapeutas, rehabilitadores, reumatólogos y veterinarios[8,9]. Aún así, los músculos en general y los puntos gatillo en particular reciben poca atención como una de las principales fuentes de dolor y disfunción en las modernas facultades de medicina y en los textos médicos[10]. Los puntos gatillo, son una causa primordial, aunque descuidada, de dolor y disfunción en el mayor órgano del cuerpo, la musculatura voluntaria (esquelética) que representa casi el 50 % del peso corporal[11].

Los puntos gatillo (trigger points) son definidos como una zona hiperirritable en un músculo esquelético asociada con un nódulo palpable hipersensible, localizado en una banda tensa[12]. La zona afectada por el punto gatillo es dolorosa a la compresión, a la contracción, al estiramiento y/o a la palpación o estimulación manual[12]. El punto gatillo puede dar lugar a un dolor referido característico[12], a un dolor reconocido como familiar[13]. Así mismo, esta alteración puede acompañarse de diferentes alteraciones sensitivas y motoras como hipersensibilidad a la presión referida[14], disfunción motora, respuesta de espasmo local, limitación dolorosa de la amplitud de movilidad al estiramiento, pérdida de fuerza y fenómenos autonómicos[5]. Además de la exploración manual de los puntos gatillo, existen exploraciones complementarias para demostrar la presencia de los mismos, como la electromiografía de superficie[8], la algometría[15,16], y la termografía[17]. En estudios recientes se ha podido visualizar la banda tensa muscular que alberga un punto gatillo o el propio nódulo mediante técnicas de elastografía por resonancia magnética[18,19] o ultrasonidos[20], lo cual augura la posibilidad de diagnóstico y/o confirmción de los puntos gatillo con pruebas de imagen.

Dado que los puntos gatillo ejercen influencia sobre los músculos asociados y se acompañan de una pérdida en el recorrido de movilidad de los tejidos que albergan, los músculos asociados con una articulación en que el movimiento se encuentra restringido deben ser examinados en búsqueda de participación de los puntos gatillo en dichas restricciones. Si bien esto puede ocurrir en cualquier articulación, existen ejemplos de la articulación del hombro como el estudio de Kuchera[21].

Los TrPs se diagnostican mediante palpación manual en el contexto de una exploración física detallada. El diagnóstico de los TrPs se basa en la presencia de una serie de síntomas y signos, entre los que destacan: a) presencia de una banda tensa palpable dentro de un músculo esquelético; b) presencia de un nódulo doloroso a la palpación dentro de la banda tensa; c) obtención de la llamada respuesta de espasmo local (contracción súbita e involuntaria de la banda tensa) con la palpación, y d) presencia de dolor referido a distancia con la palpación[12]. El diagnóstico de los TrPs necesita de una habilidad y entrenamiento manual con objeto de alcanzar un grado suficiente de precisión y fiabilidad[22]. En algunos músculos la exploración resulta más fiable que en otros. Gerwin *et al* recomendaron como criterios mínimos la presencia de un nódulo doloroso dentro de una banda tensa de un músculo esquelético, y la provocación de dolor referido con la exploración[23]. Estos criterios han mostrado una buena concordancia inter-observador, con índices kappa (k) entre 0,84 y 0,88[23]. No obstante, distintas revisiones sistemáticas han concluido con la necesidad de una estandarización de los criterios diagnósticos, ya que en la literatura se emplean criterios variados[24,25,26].

Las características clínicas más relevantes de los puntos gatillo son una historia clínica de dolor en relación con la actividad muscular[27]. Desde el punto de vista clínico, los puntos gatillo pueden ser activos o latentes. Los puntos gatillo activos son los que dan dolor local y referido, responsables directos de los síntomas de los pacientes[28] y cuyo dolor referido es reconocido por éste tanto en lo que respecta a su localización como en cuanto a la calidad del dolor[12]. Esta evidencia preliminar sugiere que el dolor referido de los puntos gatillo activos puede implicar la imagen clínica del síndrome subacromial del hombro. Los puntos gatillo latentes tienen los mismos hallazgos que los puntos gatillo activos pero no reproducen los síntomas, y su dolor referido no reproduce ningún síntoma del paciente y, por tanto, no es un dolor familiar[12]. La distinción clínica entre los puntos gatillo activos y latentes está sostenida por hallazgos histoquímicos referentes a los niveles de sustancias algogénicas y de mediadores químicos como la sustancia P, que son más altos en los puntos gatillo activos[29].

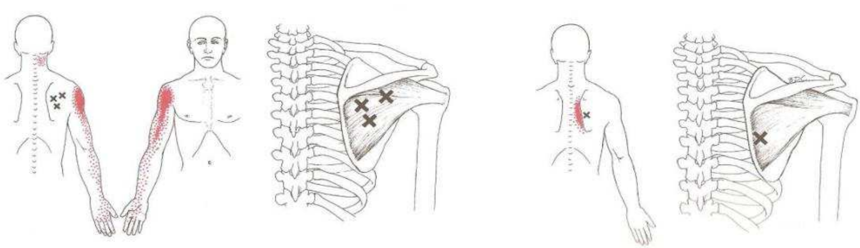
Naturaleza y etiopatogenia del trigger point (TrP)

La formación de un TrP puede resultar de factores diversos (estrés, sobrecarga por actividad física, posturas forzadas, traumatismos), capaces de generar tensión sobre el músculo. El dolor originado en los TrPs está vehiculizado por fibras nerviosas finas, amielínicas (C) y poco mielinizadas (Aδ)[30]. Un estudio realizado en ratas encontró que las conexiones medulares de los TrPs eran similares a las del tejido muscular normal, pero que las neuronas conectadas con TrPs tendían a ser de menor diámetro (principalmente, neuronas nociceptivas)[31]. Dos características de los TrPs son la hiperalgesia y la provocación de dolor referido. Como cualquier dolor referido, el dolor a distancia generado desde los TrP miofasciales puede explicarse por la convergencia de fibras aferentes de procedencias más o menos distantes sobre las mismas neuronas del sistema nervioso central[32]. La hiperalgesia se puede explicar por fenómenos de sensibilización. Mediante resonancia magnética funcional se ha comprobado que la estimulación de TrPs en pacientes con dolor miofascial desencadena una activación cerebral más extensa en áreas corticales implicadas en el procesamiento del dolor (corteza somatosensitiva y sistema límbico) que la aplicación del mismo tipo de estímulo en controles sanos[33].

En realidad, los mecanismos exactos que llevan a la aparición de TrPs son desconocidos. Se han formulado distintas hipótesis. La más difundida es la llamada “hipótesis integrada”, que sugiere que el núcleo de los TrPs lo constituyen placas motoras en disfunción, en un estado de “crisis energética” por falta de ATP[34] y la influencia de un proceso de sensibilización[35]. La teoría de la “hipótesis integrada” sugiere que el proceso de formación de los TrPs se iniciaría con un daño o una sobrecarga de la musculatura, que conduciría a una disfunción de la placa motora, con incremento de la liberación o disminución de degradación de la acetilcolina, dando lugar a una despolarización mantenida de la membrana post-sináptica. En la fibra muscular, esta despolarización provocaría la liberación de iones de calcio desde el retículo sarcoplásmico local, que a su vez causaría un acortamiento de los sarcómeros próximos a la placa motora[36]. Este acortamiento, mantenido en el tiempo, provocaría una pérdida del aporte de oxígeno y de nutrientes, asociada a un aumento de las demandas metabólicas[37]. Eventualmente se llegaría a una situación de “crisis energética” con déficit de ATP, que a su vez ocasionaría la liberación de sustancias algógenas responsables de la aparición de dolor. Al mismo tiempo, la liberación excesiva de sustancias neuroactivas podría potenciar la liberación de acetilcolina en la placa motora, generándose un círculo vicioso. El sistema nervioso simpático parece perpetuar todo este proceso[38,39,40,41]. Un estudio muy reciente ha puesto de manifiesto un estado de vasoconstricción en el TrP[42].

En estudios histológicos se ha podido demostrar que el TrP contiene numerosos nodos de contracción, que pueden ser segmentos de fibras musculares con sarcómeros extremadamente contraídos[12]. Estos hallazgos estructurales apoyan la idea de que el acortamiento de los sarcómeros en determinados puntos es un elemento fundamental para la formación de TrPs. Por otra parte, en estudios EMG se ha detectado actividad eléctrica espontánea en el seno de los TrPs, consistente en actividad de bajo voltaje con espigas superpuestas[43]. Esta actividad parece corresponder con lo que los neurofisiólogos denominan “ruido de placa motora”, que se atribuye a despolarización post-sináptica por la liberación de paquetes de acetilcolina desde las terminaciones nerviosas. Estos hallazgos electromiográficos apoyan la idea de que en el núcleo de los TrPs existan placas motoras en disfunción[44]. Precisamente la estimulación nociceptiva en las regiones donde se sitúan las placas motoras induce niveles más altos de dolor que la de otras zonas de los músculos[45]. Además, el dolor evocado desde esas regiones se ha descrito como profundo, opresivo y quemante, características similares a las descritas para el dolor referido provocado por TrPs. La existencia de mayor densidad de nociceptores musculares en la vecindad de las placas motoras podría explicar las diferencias topográficas en la intensidad y en las características del dolor de origen muscular[46]. No obstante, todavía son necesarios más estudios con objeto de confirmar estos resultados.

Los puntos gatillo activos pueden proporcionar una explicación alternativa para el dolor de hombro, lo cual es independiente de la presencia de anormalidades subacromiales morfológicas[47]. De acuerdo con Travell y Simons[48], los puntos gatillo dentro del músculo infraespinoso, el más prevalente de esta región[49], causan dolor en las regiones del deltoides medio y anterior, el cual se expande en la parte frontal y superior del brazo, refiriendo sensación de dolor en la muñeca y la mano[50].



Esquema de los puntos gatillo del músculo infraespinoso, llamado por Simons y Travell como el " músculo del dolor articular del hombro" (51).

Además, la rotación interna con addución del brazo (como alcanzar el bolsillo de atrás del pantalón, abrocharse el sujetador, etc) puede estar limitada en presencia de puntos gatillo en este músculo[52], lo cual es frecuente en pacientes con dolor de hombro[53]. Los puntos gatillo miofasciales, pues, pueden ofrecer una explicación alternativa para los mecanismos patofisiológicos que subyacen al dolor de hombro.

Dolor de hombro y puntos gatillo

El dolor muscular experimental[54], el dolor clínico muscular[55], y los puntos gatillo[56] han sido asociados a la alteración de los patrones de activación motora del hombro. Estas alteraciones han mimetizado las alteraciones cinemáticas que se han visto en el dolor de hombro en los pacientes, que a menudo han sido identificadas como síndrome subacromial[57]. Hasta la fecha, el dolor unilateral de hombro ha sido principalmente propuesto como consecuencia de alteraciones morfológicas en la anatomía del complejo articular del hombro como la inflamación de los tendones o la bursa[58], o rupturas degenerativas del manguito de los rotadores[59]. Aunque estas estructuras patológicas pueden causar dolor, se conoce que anormalidades similares han sido halladas en hombros asintomáticos[60]. El desequilibrio hallado en la activación muscular, como el enlentecimiento del reclutamiento de las fibras en músculos escapulares durante la elevación del brazo[61], sí lo hayamos en el síndrome subacromial y no en hombros asintomáticos[62]; poniendo a los puntos gatillo miofasciales en el centro de este síndrome[63].

Dolor de hombro y síndrome subacromial

El síndrome subacromial cuenta con un 44 a un 65 % de diagnósticos en visitas clínicas, cuyo motivo de consulta es el dolor de hombro[64]. Primero descrito por Neer, el síndrome subacromial ha sido clasificado en dos categorías principales: estructural y funcional[65]. El síndrome subacromial puede ser causado por un estrechamiento del espacio subacromial, resultante de una reducción del espacio debido al crecimiento óseo o a una inflamación del tejido blando (estructural), o por una migración superior de la cabeza del húmero causada por debilidad y/o desequilibrio muscular (funcional). También es posible que algún síndrome subacromial resulte de una combinación de ambos factores, estructural y funcional[66].

El síndrome subacromial funcional está más relacionado con la inestabilidad glenohumeral, y a veces es descrita como 'inestabilidad funcional', que ocurre la mayoría de las veces en deportistas de menos de 35 años de edad[67]. Hay que partir de que la articulación glenohumeral es una articulación relativamente inestable, cuya estabilidad depende de los ligamentos circundantes, músculos, y cápsula, en los que se incluyen el manguito de los rotadores y los músculos que adducen y abducen el hombro, que pueden ser responsables del origen del síndrome subacromial y del dolor de hombro, principalmente en deportistas[68]. La coordinación entre las distintas porciones funcionales de los músculos del trapecio es especialmente crucial[69]. El incremento de actividad del trapecio inferior y el superior, el descenso de la actividad del serrato anterior, y la inadecuada coordinación entre los músculos, va a incrementar la inclinación y va a aumentar la rotación externa de la escápula, durante la elevación del hombro[70]. A la larga, con este control muscular alternativo, el espacio subacromial se restringe significativamente lo que lleva al síndrome subacromial. El incremento del desequilibrio entre el deltoides anterior y los músculos del manguito de los rotadores provocan la migración superior humeral como otro factor que causa síntomas del mismo síndrome[71]. Janda explica este patrón, como el 'síndrome cruzado superior', matizando que existe también un descenso de actividad en el músculo infraespinoso y un incremento de tensión en el pectoral y el elevador de la escápula[72].

No se puede obviar, pues, que el complejo del hombro depende especialmente de esos músculos para mejorar la estabilidad dinámica durante el amplio rango de movilidad. El propio equilibrio de los músculos circundantes del complejo del hombro es además necesario para la flexibilidad y el estiramiento; un déficit en la flexibilidad y el estiramiento de la musculatura agonista puede ser compensado por la musculatura antagonista, provocando una disfunción[73]. Estos desequilibrios musculares llevan a cambios artrocinemáticos y movimientos discapacitantes, lo cual a la larga puede ser causa de daños estructurales[74].

Estas son una serie de evidencias que muestran la presencia de desequilibrios musculares en el hombro, como factor potencial relacionado con este problema de salud, aunque la etiología del síndrome subacromial no queda completa y claramente identificada[75]. Diferentes estudios han mostrado la presencia de estos desequilibrios musculares en la región del hombro[76], que cursan con dolor, y que sugieren que los puntos gatillo pueden jugar un papel relevante en el síndrome subacromial del hombro[77]. Como ejemplo, el estudio de Ingber, que describió tres pacientes con síndrome subacromial, tratados satisfactoriamente con inyección el punto gatillo del subescapular[78]. Otro ejemplo de ello es un estudio reciente en el que se ha hallado que el dolor local y referido provocados por puntos gatillo activos en el elevador de la escápula, supraespinoso, infraespinoso, subescapular, pectoral mayor y bíceps braquial, reproducían el patrón de dolor en individuos con síndrome subacromial[5]. Estos estudios apoyan que hay un rol del punto gatillo del músculo activo en el hombro con síndrome subacromial en la población general[79].

Varios estudios han demostrado que los puntos gatillo activos también están relacionados con diferentes síndromes dolorosos tales como el dolor mecánico de cuello[80], la cefalalgia tensional crónica[81], la epicondilalgia lateral[82] y la migraña[83]. El dolor referido provocado por puntos gatillo activos en la musculatura reproducen patrones de dolor asociados con estas patologías. Esta evidencia preliminar sugiere que los puntos gatillo pueden estar implicados en el cuadro clínico del síndrome doloroso de hombro. Un estudio reciente encuentra la presencia de puntos gatillo activos en la musculatura infraespinosa en pacientes con dolor de hombro unilateral[84]. Además, dos diseños de estudios promueven la relevancia del tratamiento de los puntos gatillo en el síndrome subacromial, que ha sido publicada. Sin embargo, para el mejor conocimiento de los autores no hay estudios que investiguen la presencia de puntos gatillo miofasciales en pacientes con síndrome subacromial unilateral[85].

Los resultados de estudios previos nos llevan a la conclusión de que los puntos gatillo activos pueden ser relevantes en el dolor de hombro, así como que la presencia de hipersensibilidad al dolor mecánico está relacionada con la presencia de los mismos[5]. Para inactivar estos puntos gatillo y acabar con sus factores de perpetuación[86,87] existen técnicas manuales como la compresión isquémica de los mismos[88,89]. Esta técnica, que está dentro de la terapia miofascial, va a reducir los síntomas de los pacientes que padecen dolor crónico de hombro[90]. Por tanto, estos estudios acerca del tratamiento, también sugieren que el dolor referido de los puntos gatillo activos puede ser relevante en el dolor de hombro, pudiendo estar implicados en mecanismos de sensibilización en individuos con síndrome subacromial.

Natación y dolor de hombro

La natación es un deporte donde existe un elevado caso de practicantes que presentan dolor en el hombro. La prevalencia del dolor de hombro en nadadores es ligeramente superior que en la población general, con un rango entre el 42 y el 73%, similar a la de los jugadores de voleibol[91]. Estos datos se clarifican si tenemos en cuenta que un nadador realiza alrededor de 10.000 movimientos a nivel de cada uno de sus hombros, durante las 20-30 horas que un nadador de élite puede entrenar semanalmente[92]. Tal cantidad de repeticiones año tras año se une a los desequilibrios musculares propios de la cintura escapular, para resultar una serie de factores etiológicos del desarrollo del hombro del nadador (hombro doloroso por sobreuso)[93]. Un factor que puede agravar ese desequilibrio muscular es el uso de palas en el entrenamiento[94]*.* Consecuencia normal de esto, entre el 40 y el 80% de los nadadores de competición han presentado al menos una vez en su carrera, dolores de hombro[95].

Aunque esta etiología no se ha consolidado completamente, el conflicto subacromial ha sido hipotetizado como la causa más frecuente de los problemas de hombro en el nadador[96,97]. Refiere un fenómeno mecánico en el que el contacto entre la tuberosidad mayor del húmero y el arco del acromio crean una fuerza compresiva en las estructuras subacromiales[98]. Los movimientos del hombro que provocan este compromiso fueron descritos por Neer[99,100] y consisten en: una elevación activa y máxima del brazo y una elevación lateral con el brazo rotado internamente[98]. En concreto en el nadador estos movimientos se describen asociados a la respiración unilateral, una mala posición de la cabeza, una rotación del cuerpo asimétrica y la entrada de la mano en el agua con el pulgar hacia abajo[95,101].

En cualquiera de los casos, con la evolución van a aparecer puntos gatillo miofasciales a lo largo de la región de la cintura escapular, miembro superior y región cervical[102].

Implicación de los puntos gatillo miofasciales en el dolor de hombro en nadadores

Los nadadores de élite, especialmente expuestos a las alteraciones del control motor en el hombro, han mostrado una disminución en la rotación interna después de su sesión de entrenamiento, lo cual puede estar asociado a un incremento del control neuromuscular de músculos de la escápula con otra función motora (distinta de la rotación interna)[103]. Entre las diversas alteraciones del control motor de la zona se han encontrado una disminución en la rotación ascendente de la escápula en nadadores con síndrome subacromial[104]. Se mostró también una variabilidad significativa en el tiempo de activación del serrato anterior, y trapecio superior e inferior en nadadores con síndrome subacromial, así como un incremento de la actividad del músculo trapecio superior e inferior durante la abducción del brazo[105]. El síndrome subacromial de hombro resulta pues, considerado la causa intrínseca más común del dolor y de la inestabilidad de hombro en nadadores. Sin embargo, para mejorar el conocimiento, hay que anotar que no existe información consistente sobre la posible influencia de los puntos gatillo activos en nadadores de élite con / sin síndrome subacromial[79]. En los nadadores de élite pues, el dolor de hombro es una parte inherente de la biomecánica del gesto deportivo, que fomenta los desequilibrios musculares en el nadador, estresando las estructuras cápsulo-ligamentosas que contribuyen con la inestabilidad del hombro[106].

Se proponen diferentes hipótesis sobre la etiologia del conflicto subacromial en nadadores. La inestabilidad intrínseca del complejo articular del hombro, sugiere que el gesto de la natación puede causar un estiramiento gradual de las estructuras cápsulo-ligamentosas antero-inferiores predominando la laxitud, inestabilidad y el conflicto subacromial[107,108]. Sin embargo, ningún estudio ha confirmado esta hipótesis aún. El conflicto subacromial es la causa más común del dolor de hombro y de la inestabilidad en nadadores de élite, como producto de los movimientos repetitivos y encadenados de este conjunto de articulaciones durante esta práctica deportiva, y ha sido propuesto como responsable del incremento de la laxitud de la articulación y de la tendinopatía del supraespinoso[109]. Una técnica de brazada inapropiada y la sobrecarga de entrenamientos, están relacionadas con que se promueva que la tendinopatía del supraespinoso, asociada al síndrome subacromial[110]. En términos generales, tanto el dolor de hombro como el incremento de la sensibilidad al dolor mecánico, son comunes después del entrenamiento intensivo en natación[111]. Sin embargo, estos datos han sido cuestionados por otro estudios que no encontraron diferencias cinemáticas, latencias o recidivas por orden de los músculos del hombro durante la elevación del hombro en el plano escapular entre los nadadores con síndrome

subacromial[112]. Además, un estudio reciente ha descrito la alta prevalencia del movimiento anormal escapular durante una sesión de entrenamiento normal en nadadores libres de dolor[113].
En deportes como este, cuya actividad física se focaliza en movimientos del brazo continuos y repetitivos, las lesiones de hombro que acontecen con dolor son muy frecuentes[114,115]. El dolor de hombro llega a ser una de las causas más comunes de discapacidad física en nadadores de élite como consecuencia de una biomecánica asociada estrechamente a los desequilibrios musculares que van a estresar el complejo cuello-hombro, no sólo el complejo articular del hombro[116].

Estos hallazgos, en definitiva, sugieren que los problemas de control motor están más relacionados con el dolor de hombro en nadadores de élite. Como el complejo del hombro opera con un balance preciso en sintonía entre el hombro y la columna cervical, es posible que los problemas en el control motor de los músculos cervicales pudieran estar envueltos en el desarrollo del dolor de hombro en nadadores de élite[117]. De hecho, la evidencia de los problemas de control motor en la musculatura cervical ha sido documentada en pacientes con dolor mecánico de cuello y latigazo cervical asociado a dolor de cuello durante los tests motores prescritos[118]. Algunos autores han investigado los patrones de activación muscular del cuello-hombro en individuos con latigazo cervical asociado al dolor de cuello o al dolor crónico de cuello con una carga baja, a través de tests funcionales del miembro superior[117]. Ellos describen que los sujetos con dolor de cuello exhibían un incremento de la actividad en el esternocleidomastoideo y el trapecio superior comparado con los voluntarios sanos, bajo la presencia de patrones alterados de activación muscular en estas condiciones de dolor; además de mostrar un descenso en la habilidad de relajar los músculos y volver a los patrones de activación normal después del ejercicio físico[118]. Signos electromiográficos musculoesqueléticos evidencian que el comienzo del dolor inicia respuestas neuromusculares (y comportamentales)[117]. Por ello, las valoraciones cuantitativas de las adaptaciones funcionales motoras pueden servir de referencia para el estado de dolor y ayudar a identificar signos indicando el desarrollo de desórdenes musculoesqueléticos, o lo que es lo mismo, que el dolor de hombro pueda dar lugar a alteraciones motoras cervicales asociadas.

RESUMEN

Objetivos del estudio

Los objetivos de esta serie de estudios son:

- Investigar la presencia de hiperalgesia a la presión en pacientes con síndrome subacromial respecto a sujetos sanos.
- Describir las diferencias en la presencia de puntos gatillo en la musculatura del hombro entre los pacientes con síndrome subacromial unilateral y sujetos sanos.
- Analizar si el umbral doloroso a la presión está relacionado con la presencia de puntos gatillo en la musculatura del hombro de pacientes con dolor de hombro.
- Analizar las diferencias en el comportamiento de los músculos cervicales entre los nadadores de élite con dolor de hombro y aquellos sin dolor, durante un test funcional del miembro superior.
- Investigar los cambios en el dolor y en la sensibilidad del dolor a la presión después del tratamiento manual de los puntos gatillo activos en la musculatura del hombro en pacientes con síndrome subacromial unilateral.

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Artículo	Diseño del estudio	Participantes	Intervención	Principales variables del estudio	Métodos
Changes in pain and pressure pain sensitivity after manual treatment of active trigger points in patients with unilateral shoulder impingement: A case series	Serie de casos	12 pacientes (siete hombres, cinco mujeres), diagnosticados con síndrome de atrapamiento unilateral de hombro.	Terapia manual (presión e intervención neuromuscular) sobre los puntos gatillo activos.	Dolor unilateral de hombro. Puntos gatillo y umbral doloroso a la presión en: elevador de la escápula, supraespinoso, infraespinoso, pectoral mayor, subescapular y tibial anterior.	Test de Neer y Test de Hawkins. Exploración manual y algometría de presión.
Muscle trigger points and pressure pain hyperalgesia in the shoulder muscles in patients with unilateral shoulder impingement: a blinded, controlled study	Estudio ciego de casos y controles	12 pacientes con síndrome de atrapamiento unilateral, y 10 sujetos- controles.	No aplicable.	Dolor unilateral de hombro. Intensidad del dolor. Puntos gatillo y umbral doloroso a la presión.	Test de Neer y Test de Hawkins. Escala VAS Exploración manual y algometría de presión.
Elite swimmers with and without shoulder impingement: mechanical hyperaglsia and trigger point in neck-shoulder muscles	Estudio de casos-control	17 nadadores de élite (nueve hombres y ocho mujeres) con síndrome unilateral de atrapamiento; 18 nadadores de élite (nueve hombres y nueve mujeres) sin síndrome de atrapamiento; y 15 atletas de élite (siete hombre y ocho mujeres) como controles.	No aplicable.	Dolor unilateral de hombro. Intensidad del dolor. Puntos gatillo y umbral doloroso a la presión.	Test de Neer y Test de Hawkins. Escala VAS Exploración manual y algometría de presión.
Elite swimmers with unilteral shoulder pain exhibit higher cervical muscle activity during a functional upper limb task	Estudio de casos-control	17 nadadores de élite (nueve hombres y ocho mujeres) con dolor unilateral de hombro; y 17 nadadores de élite sin dolor de hombro.	Test funcional de baja carga, de miembro superior.	Actividad electromiográfica en los siguientes músculos: trapecio superior, esternocleidomastoideo y escaleno anterior (bilateralmente)	Electromiografía de superficie.

Cambios en el dolor y el la sensibilidad al dolor después de un tratamiento manual de puntos gatillo actives en pacientes con síndrome subacromial unilateral: serie de casos

(Artículo I)

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CASE SERIES

Changes in pain and pressure pain sensitivity after manual treatment of active trigger points in patients with unilateral shoulder impingement: A case series

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KEYWORDS
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Summary The aim of this case series was to investigate changes in pain and pressure pain sensitivity after manual treatment of active trigger points (TrPs) in the shoulder muscles in individuals with unilateral shoulder impingement. Twelve patients (7 men, 5 women, age: 25 ± 9 years) diagnosed with unilateral shoulder impingement attended 4 sessions for 2 weeks (2 sessions/week). They received TrP pressure release and neuromuscular interventions over each active TrP that was found. The outcome measures were pain during arm elevation (visual analogue scale, VAS) and pressure pain thresholds (PPT) over levator scapulae, supraspinatus infraspinatus, pectoralis major, and tibialis anterior muscles. Pain was captured pre-intervention and at a 1-month follow-up, whereas PPT were assessed pre- and post-treatment, and at a 1-month follow-up. Patients experienced a significant ($P < 0.001$) reduction in pain after treatment (mean ± SD: 1.3 ± 0.5) with a large effect size ($d > 1$). In addition, patients also experienced a significant increase in PPT immediate after the treatment ($P < 0.05$) and one month after discharge ($P < 0.01$), with effect sizes ranging from moderate ($d = 0.4$) to large ($d > 1$).A significant negative association ($r_s = -0.525$; $P = 0.049$) between the increase in

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PPT over the supraspinatus muscle and the decrease in pain was found: the greater the decrease in pain, the greater the increase in PPT. This case series has shown that manual treatment of active muscle TrPs can help to reduce shoulder pain and pressure sensitivity in shoulder impingement. Current findings suggest that active TrPs in the shoulder musculature may contribute directly to shoulder complaint and sensitization in patients with shoulder impingement syndrome, although future randomized controlled trials are required.

Introduction

Shoulder pain is a common health problem that has a multifactorial underlying pathology with high direct costs for the society (Meislin et al., 2005). The one-year prevalence of shoulder pain ranges from 20% to 50% in the general population (Pope et al., 1997; Luime et al., 2004). Among the different causes of shoulder pain, the most prevalent diagnosis is shoulder impingement (13%) (Pribicevic et al., 2009).

The aetiology of shoulder impingement is not completely understood, but there is evidence showing the role of the shoulder musculature as a potential factor (Tyler et al., 2005). Different studies have shown the presence of muscle imbalance of the shoulder musculature in this painful condition (Ludewig and Cook, 2000; Moraes et al., 2008). Due to this imbalance, Simons et al. (1999) suggested that muscle trigger points (TrP) can play a relevant role in shoulder impingement syndrome. TrPs are defined as hypersensible spots in a taut band of a skeletal muscle, painful on contraction, stretching or manual stimulation which give rise to a referred distant pain. Active TrPs are those which their local and referred pains are responsible for the patients' symptoms. There is preliminary evidence suggesting that referred pain from active TrPs may be implicated in the clinical picture of shoulder impingement. Ingber (2000) described 3 patients with shoulder impingement syndrome who were successfully treated with TrPs injection of the subscapularis muscle. Ge et al. (2008) described the presence of active TrPs within the infraspinatus muscle in individuals with shoulder pain, without specific diagnosis. A recent study reported that the referred pain elicited by active TrPs in the supraspinatus, infraspinatus, pectoralis mayor and subscapularis muscles reproduced the pain pattern in subjects with shoulder impingement (Hidalgo-Lozano et al., 2010). The hypothesis that active TrPs may be relevant for shoulder pain has been supported by the study of Hains et al. (2010) where myofascial therapy using ischemic compression on shoulder TrPs reduced the symptoms of patients experiencing chronic shoulder pain. Therefore, these studies suggest that referred pain from active TrPs may be relevant for shoulder pain.

Hidalgo-Lozano et al also found that patients with shoulder impingement exhibit generalized pressure pain hypersensitivity as compared to controls (Hidalgo-Lozano et al., 2010). In addition, the presence of mechanical pain hypersensitivity was related to the presence of active TrPs, suggesting that active TrPs may be involved in sensitization mechanisms in individuals with impingement syndrome (Hidalgo-Lozano et al., 2010). The aim of this case series was to investigate changes in pain and pressure pain sensitivity after manual treatment of active muscle TrPs in the shoulder musculature in patients with unilateral shoulder impingement.

Methods

Patients

Consecutive patients with diagnosis of strictly unilateral impingement syndrome stage I (acute inflammation and either tendonitis or bursitis) (Frieman et al., 1994) within the dominant-right hand were recruited. Patients were eligible if: 1) they had unilateral shoulder complaints with duration of at least 3 months; 2) an intensity of at least 4 on an 11-point numerical pain rating scale (NPRS) during arm elevation; 3) positive Neer test, that is, pain during passive abduction (Neer, 1983); and, 4) positive Hawkins, that is, pain when the arm is flexed at 90° and passively positioned in internal rotation (MacDonald et al., 2000). The sensitivity and specificity for the Neer test has been estimated as 79% and 53%, respectively, and for the Hawkins test 79% and 59%, respectively (Hegedus et al., 2008).

Patients were excluded if they exhibited any of the following criteria: 1, bilateral shoulder symptoms; 2, younger than 18 or older than 65 years; 3, history of shoulder fractures or dislocation; 4, cervical radiculopathy; 5, previous interventions with steroid injections; 6, fibromyalgia syndrome (Wolfe et al., 1990); 7, previous history of shoulder or neck surgery; or 9, any type of physical intervention for the neck-shoulder area the previous year.

The study was approved by the local Ethics Committee (UC 2009-45) conducted following the Helsinki Declaration. All participants signed an informed consent prior to their inclusion.

Outcome measures

In this study, a visual analogue scale (VAS) (Jensen et al., 1999) was used to assess the intensity of pain experienced during arm elevation pre-intervention and one month after discharge. The VAS is a 10 cm line anchored with a "0" at one end representing "no pain" and "10" at the other end representing "the worst pain imaginable". Patients placed a mark along the line corresponding to the intensity of the symptoms, which was scored to the nearest centimetre. It has been shown to be reliable and valid for assessing pain intensity (Bijur et al., 2001), and it was selected as outcome measure based on its ability to detect immediate changes in pain exhibiting a minimal clinically important difference (MCID) between 0.9 cm and 1.1 cm (Bird and Dickson, 2001; Gallagher et al., 2001).

In addition, pressure pain thresholds (Vanderweeen et al., 1996) (PPT: minimal amount of pressure where a sensation of pressure first changes to pain) over the levator scapulae (2 cm superior to the superior angle of the scapula bone), supraspinatus (middle point over the fosa of the scapula), infraspinatus (middle muscle belly), pectoralis major (middle point under the clavicle bone), and tibialis anterior (halfway between the most superior attachment and its tendon in the upper one third of the muscle belly) muscles were also assessed. To investigate general hypoalgesic effects of TrP interventions, the inclusion of PPT assessment over the tibialis anterior was needed.

In this study, a mechanical pressure algometer (Pain Diagnosis and Treatment Inc.©, Great Neck, NY) was used (kg/cm^2). The mean of 3 trials over each point was calculated and used for analysis. A 30-s resting period was allowed between each trial. The reliability of pressure algometry has been found to be high the same day (ICC = 0.91 [95% CI 0.82–0.97]) (Chesterson et al., 2007) and between 4 separate days (ICC = 0.94–0.97) (Jones et al., 2007). PPT levels were assessed pre-intervention, post-intervention and one month after discharge.

Myofascial/muscle TrP therapy

None of the patients were taking any preventive drug at the time the study was performed. Participants were asked to avoid any analgesic or muscle relaxant during which the study was conducted.

Patients were treated by a clinician with more than 6 years of clinical experience in the management of shoulder disorders. All participants attended the physical therapy clinic 2 days per week for 2 weeks (4 sessions). They received the following manual therapies depending on clinical findings related to the location of the TrP.

Subjects were examined for the presence of active TrPs in the levator scapulae, supraspinatus, infraspinatus, subscapularis, and pectoralis major muscles by a clinician with more than 5 years of experience in the management of TrPs. TrP diagnosis was conducted according to Simons et al. (1999): 1) palpable taut band in a skeletal muscle; 2) hyperirritable tender spot in the taut band; 3) local twitch response elicited by the snapping palpation of the taut band; and 4) presence of referred pain in response to TrP compression (Fig. 1). These criteria, when applied by an experience assessor, have obtained a good inter-examiner reliability (kappa) ranging from 0.84 to 0.88 (Gerwin et al., 1997). Bron et al. (2007a,b) evaluated patients with shoulder pain and found that the most reliable feature of TrP was the referred pain (percentage of pair-wise agreement ≥70%, range 63–93%).

Different manual approaches have been proposed for the management of muscle TrPs (Dommerholt and McEvoy, 2010). A recent systematic review found moderate strong evidence supporting the use of TrP pressure release for immediate pain relief of muscle TrPs (Vernon and Schneider, 2009). In the current study, patients received a TrP pressure release technique over each active TrP that was found (Fig. 2). Pressure was applied over TrPs until an increase in muscle resistance (barrier) was perceived by the clinician and maintained until the clinician perceived release of the taut band (Lewit, 1999). At this stage the pressure was increased to return to previous level of muscle tension and the process was repeated for 90 s (usually 2 to 3 repetitions).

Patients also received a neuromuscular technique (longitudinal stroke) (Chaitow, 2010) over the affected muscle, particularly supraspinatus, infraspinatus, and pectoralis major muscles. The thumb of the therapist was placed over the taut band and longitudinal strokes were applied slowly with moderate pressure which was not painful for the patient. This technique has been found to be effective for reducing TrP pressure sensitivity (Ibáñez-García et al., 2009).

TrP manual therapies were applied depending on clinical findings related to the location of the TrP on the affected arm. No pre-determined TrP location was considered.

Statistical analysis

Data were analysed with the SPSS statistical package (19.0 Version). Results are expressed as mean, standard deviation (SD) or 95% confidence interval (95% CI). Due to the small sample size and the nature of the data, the use of non-parametric tests was considered robust. The non-parametric Wilcoxon signed test was used to examine differences from baseline to each time point for VAS and PPT levels. Further,

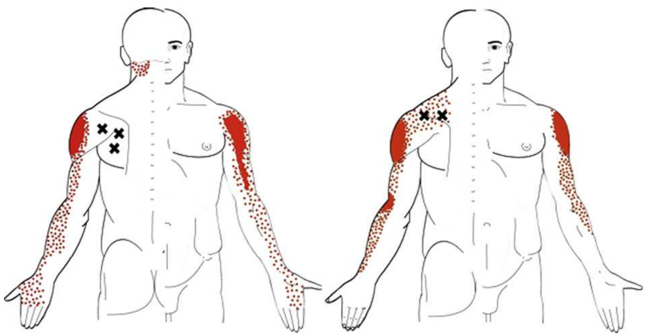

Figure 1 Referred pain from infraspinatus (left) and supraspinatus (right) muscle trigger points (TrPs) according to Simons et al. (1999).

Figure 2 TrP pressure release over infraspinatus TrPs.

changes in VAS and PPT were stratified by gender using the non-parametric U-Mann Whitney test. In addition, to further investigate if changes were clinically relevant, effect sizes were calculated using Cohen *d* coefficient (*d*) (Cohen, 1988). Effect sizes of 0.2 are considered as small, 0.5 as moderate and 0.8 large (Cohen, 1988). Finally, the Spearman's rho (r_s) was used to investigate the associated between changes in pain intensity and changes over PPT over each point at before and one month after treatment. The statistical analysis was conducted at 95% confidence level and a $P < 0.05$ was considered statistically significant.

Results

Clinical data of the participants

Twelve patients, 7 men and 5 women, aged 20–38 years (mean: 25 ± 9 years) diagnosed with unilateral shoulder impingement participated. All patients reported pain located in the anterior and posterior parts of the shoulder and the dorso-lateral aspect of the forearm in 5 patients (42%). The mean duration of shoulder pain history was 8.7 ± 4.8 months (95%CI 5–12.4), and the mean intensity of pain experienced during arm active elevation was 5.1 ± 1.9 (95% CI 3.9–6.4).

Changes in pain

The Wilcoxon signed test revealed a significant effect ($z = -2.511$; $P = 0.011$) for pain. Patients experienced a significant reduction in pain (mean ± SD: 1.3 ± 0.5, 95% CI 0.9–2.3) from pre-intervention (mean ± SD: 5.1 ± 1.9, 95% CI 3.9–6.4) as compared to one month after discharge (mean ± SD: 3.8 ± 1.3, 95% CI 2.3–5.2). The effect size for pain was large ($d > 1$). No significant differences between men and women ($t = 0.781$; $P = 0.453$) for changes in pain were found.

Changes in pressure pain sensitivity

The repeated Wilcoxon signed test revealed a significant effect for changes over the levator scapulae ($z = -2.040$; $P = 0.041$), supraspinatus ($z = -2.047$; $P = 0.042$), infraspinatus ($z = -2.353$; $P = 0.019$), pectoralis major ($z = -2.080$; $P = 0.038$), and tibialis anterior ($z = -2.041$; $P = 0.040$) muscles. Patients experienced a significant increase in PPT immediate after treatment and one month after the discharge ($P < 0.05$). Again, no significant differences for PPT difference scores between genders were found for the levator scapulae ($t = 0.622$; $P = 0.523$), supraspinatus ($t = 0.723$; $P = 0.486$), infraspinatus ($t = 1.672$; $P = 0.125$), pectoralis major ($t = 0.372$; $P = 0.718$), and tibialis anterior ($t = 0.972$; $P = 0.502$) muscles. Table 1 summarizes PPT levels at each point at pre-, post- and 1 month after discharge, whereas Table 2 shows pre-post changes for PPT data.

Relationship between changes in pain and pressure pain sensitivity

A significant negative association ($r_s = -0.525$; $P = 0.049$) between the increase in PPT over the supraspinatus muscle and the decrease in pain was found: the greater the decrease in pain, the greater the increase in pressure pain threshold.

Discussion

The current case series has shown that manual treatment of active TrPs within the shoulder muscles reduces spontaneous pain and increases PPT levels in individuals with shoulder impingement. Current results underline the importance of inspection and inactivation of active muscle TrPs in the shoulder musculature in patients with shoulder impingement syndrome as they may contribute to the overall picture of pain; however, future randomized controlled trials are required to further confirm this assumption. In fact, two randomized controlled trials have been proposed in order to elucidate the role of inactivation of muscle TrPs in patients with shoulder impingement syndrome (Bron et al., 2007a,b; Perez-Palomares et al., 2009).

The rotator cuff is formed by the supraspinatus, the infraspinatus, the teres minor and the subscapularis muscles (Keating et al., 1993). In the current case series, active myofascial TrPs in the supraspinatus, infraspinatus, and subscapularis were manually treated. A previous study found that the presence of active TrPs in the supraspinatus and infraspinatus muscles was related to a greater intensity of pain in patients with shoulder impingement, which support the role of active TrPs within the clinical pain

Table 1 Pressure pain thresholds (PPT, kg/cm^2) pre-intervention, post-interventon and one month after discharge.

	Pre-intervention	Post-intervention	One month after discharge
Levator scapulae muscle	1.9 ± 0.9 (95% CI 1.3–2.5)	2.5 ± 0.8 (95% CI 2.1–3.1)	2.8 ± 0.9 (95% CI 2.2–3.4)
Supraspinatus muscle	2.3 ± 1.0 (95% CI 1.7–3.0)	2.8 ± 0.7 (95% CI 2.4–3.3)	3.0 ± 0.8 (95% CI 2.5–3.5)
Infraspinatus muscle	2.0 ± 0.8 (95% CI 1.5–2.5)	2.9 ± 1.4 (95% CI 2.0–3.8)	2.9 ± 1.0 (95% CI 2.25–3.5)
Pectoralis major muscle	1.2 ± 0.4 (95% CI 1.0–1.4)	1.7 ± 0.6 (95% CI 1.3–2.0)	1.8 ± 0.4 (95% CI 1.5–2.1)
Tibialis anterior muscle	4.2 ± 0.9 (95% CI 3.7–4.9)	4.6 ± 1.9 (95% CI 3.4–5.9)	4.9 ± 1.9 (95% CI 3.7–6.2)

Values are expressed as means ± standard deviation (95% confidence interval)

Table 2 Pre-post and pre-follow/up change scores and effect sizes for pressure pain thresholds (PPT, kg/cm^2)

	Pre-post change scores	Pre-post effect size	Pre-follow/up scores	Pre-follow/up effect size
Levator scapulae muscle	0.6 ± 0.8 (95% CI 0.3–1.6)	0.75	0.9 ± 1.0 (95% CI 0.3–1.5)	0.90
Supraspinatus muscle	0.5 ± 0.9 (95% CI 0.2–1.0)	0.45	0.7 ± 0.9 (95% CI 0.2–1.3)	0.78
Infraspinatus muscle	0.9 ± 1.0 (95% CI 0.4–1.4)	0.90	0.9 ± 0.7 (95% CI 0.4–1.3)	1.10
Pectoralis major muscle	0.5 ± 0.7 (95% CI 0.2–0.9)	0.64	0.6 ± 0.3 (95% CI 0.4–0.7)	2.00
Tibialis anterior muscle	0.4 ± 0.8 (95% CI 0.2–1.1)	0.50	0.7 ± 1.0 (95% CI 0.4–1.6)	0.70

Values are expressed as means ± standard deviation (95% confidence interval)

picture of these patients (Hidalgo-Lozano et al., 2010). In the current case series one month after 4 sessions of treatment, patients exhibited a decrease of 1.3 cm on pain which surpassed the MCID. Nevertheless, it should also be noted that lower bound estimation for the 95% confidence interval fall in the reported MCID of 0.9–1.1 cm (Bird and Dickson, 2001; Gallagher et al., 2001). Hence, current results should be considered with caution. These findings support the view that active TrPs in the shoulder musculature may contribute directly to shoulder pain complaint in individuals with shoulder impingement syndrome, although future randomized controlled trials are required.

It has been previously reported that subjects with shoulder impingement exhibit both segmental and widespread sensitization mechanisms and that this mechanisms are related to the presence of active TrPs and pain symptoms (Hidalgo-Lozano et al., 2010). Shah et al. (2005, 2008) demonstrated that active TrPs constitutes a focus of peripheral sensitization as higher levels of algogenic substances such as bradykinin, substance *P*, or serotonin, are found in active TrPs as compared with non-TrPs. In addition, Li et al. (2009) recently demonstrated the existence of nociceptive and non-nociceptive hypersensitivity at muscle TrPs. Hence, it would be expected that treatment of active TrPs would reduce this sensitization. The current case series support this hypothesis as moderate to large increases in PPT levels were found one month after the intervention. Nevertheless, although effect sizes support a clinical effect over mechanical sensitivity; we recognize that MCID of PPT levels has not been previously studied. Our results support that muscle TrP treatment can decrease pressure pain hypersensitivity, which is in agreement with two previous studies that demonstrated that TrP treatment induces segmental anti-nociceptive effects (Srbely et al., 2008, 2010). In fact, Hsieh et al. (2007) showed that dry needling of active TrPs in the infraspinatus muscle decreased the pain intensity and mechanical pain sensitivity on the treated arm in patients with shoulder pain, supporting this anti-nociceptive effect. Additionally, the fact that PPT levels also improved in distant pain-free areas, e.g. tibialis anterior muscle, indicates a generalized anti-nociceptive effect of TrP therapy, which has been previously suggested (Niddam et al., 2007). Nevertheless, the association between the decrease in pain and the increases in PPT levels was weak.

Finally, we should recognize some limitations to the current case series. First, a study without a comparison group does not allow for inferences to be made regarding cause and effect. Therefore, as result of a lack of control group, we cannot determine if changes in pain and pressure sensitivity were due to the intervention. Second, we only include a small number of patients with shoulder impingement, which limit the results. Therefore, Future randomized clinical trials are now needed (Bron et al., 2007a,b; Perez-Palomares et al., 2009). Thirdly, we only examined the effects 1-month after discharge, so we do not know the long-term effects of the intervention. The fact that statistically significant changes occurred at short-term follow-up provides impetus for future research in this area.

Conclusion

This case series suggests that manual treatment of active TrPs may reduce spontaneous pain and increase PPT in patients with shoulder impingement. Effect sizes were large for pain and moderate-large for changes in PPT. Current findings suggest that active TrPs in the shoulder musculature may contribute to shoulder complaint and sensitization in patients with shoulder impingement syndrome. However, due to a small sample size and the absence of a control group, these assumptions should be consider with caution.

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Puntos gatillo musculares e hiperalgesia a la presión en músculos del hombro en pacientes con síndrome subacromial unilateral: un estudio ciego de casos y controles

(Artículo II)

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RESEARCH ARTICLE

Muscle trigger points and pressure pain hyperalgesia in the shoulder muscles in patients with unilateral shoulder impingement: a blinded, controlled study

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Abstract Our aim was to describe the differences in the presence of trigger points (TrPs) in the shoulder muscles and to investigate the presence of mechanical hypersensitivity in patients with unilateral shoulder impingement and healthy controls. Twelve patients with strictly unilateral shoulder impingement and 10 matched controls were recruited. TrPs in the levator scapula, supraspinatus, infraspinatus, subscapularis, pectoralis major, and biceps brachii muscles were explored. TrPs were considered active if the local and referred pain reproduced the pain symptoms and the patient recognized the pain as a familiar pain. Pressure pain thresholds (PPT) were assessed over the levator scapulae, supraspinatus, infraspinatus, pectoralis major, biceps brachii, and tibialis anterior muscles. Both explorations were randomly done by an assessor blinded to the subjects' condition. Patients with shoulder impingement have a greater number of active (mean ± SD: 2.5 ± 1; $P < 0.001$) and latent (mean ± SD: 2 ± 1; $P = 0.003$) TrPs when compared to controls (only latent TrPs, mean ± SD: 1 ± 1). Active TrPs in the supraspinatus (67%), infraspinatus (42%), and subscapularis (42%) muscles were the most prevalent in the patient group. Patients showed a significant lower PPT in all muscles when compared to controls ($P < 0.001$). Within the patient group a significant positive correlation between the number of TrPs and pain intensity ($r_s = 0.578$; $P = 0.045$) was found. Active TrPs in some muscles were associated to greater pain intensity and lower PPTs when compared to those with latent TrPs in the same muscles ($P < 0.05$). Significant negative correlations between pain intensity and PPT levels were found. Patients with shoulder impingement showed widespread pressure hypersensitivity and active TrPs in the shoulder muscles, which reproduce their clinical pain symptoms. Our results suggest both peripheral and central sensitisation mechanisms in patients with shoulder impingement syndrome.

Keywords Shoulder impingement · Trigger points · Pressure pain · Sensitization

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Introduction

Shoulder pain is a common health problem that has a multifactorial underlying pathology and is associated with high societal cost and patient burden. The 1-year prevalence of shoulder disorders ranges from 20 to 50%, depending on the definition of the condition and socio-demographic features (Pope et al. 1997; Luime et al. 2004). It is estimated that the incidence of shoulder disorders ranges from 7 to 25 per 1,000 consultations with general physicians (Van der Windt et al. 1995). A recent survey found that the prevalence of shoulder pain as reported by practitioners was 12%, with the most prevalent working diagnosis impingement syndrome (13%) (Pribicevic et al. 2009). In 2000, the direct costs for the treatment of shoulder disorders in the United States were $7 billion (Meislin et al. 2005).

Shoulder impingement syndrome is considered the most common intrinsic cause of shoulder pain and disability. The etiology of shoulder impingement is not completely understood, but there is evidence showing the role of shoulder muscles as a potential related factor to this condition (Tyler et al. 2005). For instance, patients with light to moderate shoulder impingement syndrome had late recruitment of scapular muscles during arm elevation (Moraes et al. 2008). Ludewig and Cook (2000) found an increased upper and lower trapezius muscle activity during shoulder abduction in patients with shoulder impingement syndrome. Due to this imbalance in muscle activation, some authors have suggested that myofascial trigger points (TrPs) may play a relevant role in shoulder impingement syndrome (Simons et al. 1999).

TrPs are defined as hypersensitive spots in a taut band of a skeletal muscle that are painful on contraction, stretching or manual stimulation and give rise to a referred pain distant from the spot. Muscle TrPs may be active or latent. Active TrPs are those in which both their local and referred pains are recognized by the patient as responsible for pain symptoms. Latent TrPs have the same clinical findings as active TrPs, but they are not causing clinical symptoms (Simons et al. 1999). This clinical distinction between active and latent TrPs is substantiated by histo-chemical findings because higher levels of algogenic substances and chemical mediators (i.e., bradykinin, substance P, or serotonin) have been found in active TrPs when compared with latent TrPs and non-TrPs (Shah et al. 2005, 2008).

Several studies have demonstrated that active TrPs are related to different pain syndromes such as mechanical neck pain (Fernández-de-las-Peñas et al. 2007a), chronic tension type headache (Fernández-de-las-Peñas et al. 2007b, c, d), lateral epicondylalgia (Fernández-Carnero et al. 2007), and migraine (Calandre et al. 2006; Fernández-de-las-Peñas et al. 2006). The referred pain elicited by active muscle TrPs reproduced pain patterns associated with these pathologies. There is preliminary evidence suggesting that TrPs may be implicated in the clinical picture of shoulder impingement syndrome. With a case design, Ingber (2000) described 3 patients with shoulder impingement syndrome who had not respond to traditional treatment who were successfully treated with TrPs injection of the subscapularis muscle. A recent study found the presence of active TrPs in the infraspinatus muscle in patients with unilateral shoulder pain (Ge et al. 2008). Additionally, 2 study designs promoting the relevance of TrP treatment in shoulder impingement syndrome have been published (Bron et al. 2007a; Perez-Palomares et al. 2009). Nevertheless, to the best of authors' knowledge, there are no studies investigating the presence of myofascial TrPs in patients with unilateral shoulder impingement syndrome.

The aims of the present study were: (1) to describe the differences in the presence of TrPs in the levator scapulae, supraspinatus, infraspinatus, subscapularis, pectoralis major, and biceps brachii muscles between patients with strictly unilateral shoulder impingement and healthy controls; (2) to investigate the presence of pressure pain hyperalgesia in patients with shoulder impingement; (3) to assess the relationship between active or latent TrPs and pain intensity; and (4) to analyze if pressure pain thresholds were related to the presence of TrPs in the shoulder muscles.

Materials and methods

Participants

Patients diagnosed by an orthopedic surgeon with stage I (Frieman et al. 1994) unilateral impingement syndrome (acute inflammation and either tendonitis or bursitis) on the dominant-right side were recruited. Patients were eligible if they had unilateral shoulder complaints (described as pain felt in the shoulder or upper arm) with a duration of at least 3 months and an intensity of at least 4 on an 11-point numerical pain rating scale (NPRS) during arm elevation. Patients would need to report positive Neer and Hawkins tests for the diagnosis of shoulder impingement syndrome. The Neer test is positive when the patient reports pain during passive arm elevation (Neer 1983). The Hawkins test is positive when the patient reports pain when the arm is flexed at 90° and passively positioned in internal rotation (MacDonald et al. 2000). A recent meta-analysis revealed that the pooled sensitivity and specificity for the Neer test was 79 and 53%, respectively, and for the Hawkins test was 79 and 59%, respectively (Hegedus et al. 2008).

Patients were excluded if they exhibited any of the following criteria: 1, bilateral shoulder symptoms; 2, younger than 18 or older than 65 years; 3, history of shoulder fractures or dislocation; 4, cervical radiculopathy; 5, previous interventions with steroid injections; 6, fibromyalgia syndrome (Wolfe et al. 1990); 7, any systemic disease; 8, previous history of shoulder or neck surgery; or 9, any type of physical intervention for the neck–shoulder area during the previous year.

Additionally, age-matched right-handed controls were recruited from volunteers who responded to a local announcement. They were excluded if they exhibited a history of neck, shoulder or arm pain, history of trauma or diagnosis of any systemic disease. Both the Neer and Hawkin tests were negative. The study protocol was approved by the local ethic committee (UC 45) and conducted according to the Helsinki Declaration. All participants signed an informed consent prior to their inclusion.

Muscle trigger point examination

Muscle TrPs were explored in the levator scapulae, supraspinatus, infraspinatus, subscapularis, pectoralis major, and biceps brachii muscles by an assessor who had more than 8 years' experience in muscle TrPs diagnosis and who was blinded to the subjects' condition. TrP diagnosis was performed following the criteria described by Simons et al. (1999) and by Gerwin et al. (1997): (1) presence of a palpable taut band in a skeletal muscle; (2) presence of a hyperirritable tender spot within the taut band; (3) local twitch response elicited by the snapping palpation of the taut band; and (4) presence of referred pain in response to TrP compression. These criteria, when applied by an experience assessor, have obtained a good inter-examiner reliability (kappa) ranging from 0.84 to 0.88 (Gerwin et al. 1997). Bron et al. (2007b) evaluated patients with shoulder pain and found that the most reliable feature of TrP was the referred pain (percentage of pair-wise agreement ≥70%, range 63–93%).

TrPs were considered active when both the local and the referred pain evoked by digital compression reproduced the pain symptoms (both in location and pain sensation) and the subject recognized the pain as familiar pain (Simons et al. 1999), whereas TrPs were considered latent when the local and referred pain elicited by digital compression did not reproduce symptoms familiar to the subjects. Figure 1 details the referred pain patterns evoked by TrPs in the examined shoulder muscles according to Simons et al. (1999).

TrP examination was performed in a blinded fashion. After TrP assessment in all the muscles, the participant was asked: "When I pressed these muscles, did you feel any pain or discomfort locally, and in other areas (referred

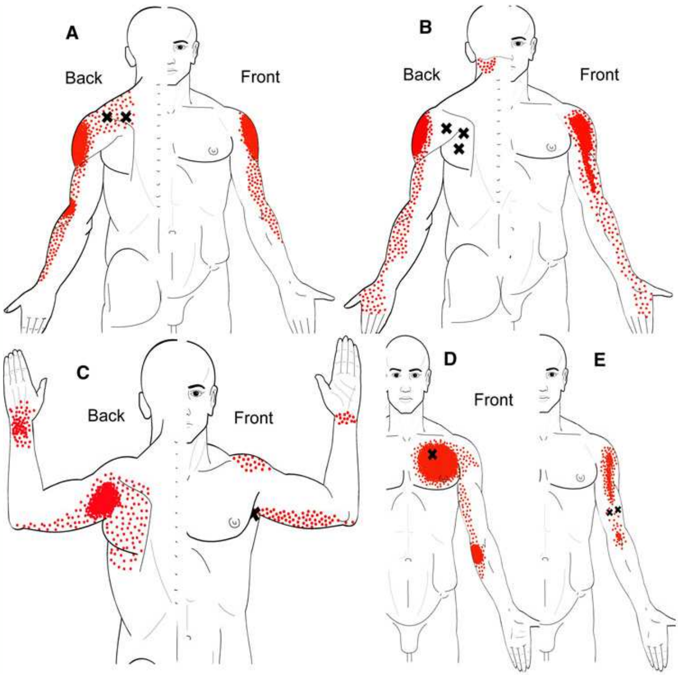

Fig. 1 Referred pain patterns from supraspinatus (**a**), infraspinatus (**b**) Subscapularis (**c**), pectoralis major (**d**), and biceps brachii (**e**) muscle TrPs as described by Simons et al. (1999)

pain). Please tell me whether the pain that you felt in the other area reproduced symptoms that you are suffering from". Participants had to indicate whether the pain elicited by palpation was located in the same area of their symptoms and reproduced the same pain sensation (active TrPs). If the elicited local or referred pain did not reproduce the same pain sensation than the patient suffered from, the TrP was considered latent.

Pressure pain threshold

Pressure pain threshold (PPT) is defined as the minimal amount of pressure where a sensation of pressure first changes to pain (Vanderweeen et al. 1996). A mechanical pressure algometer (Pain Diagnosis and Treatment Inc., Great Neck, NY) was used in this study. The device consists of a 1-cm^2 rubber disk attached to a pressure gauge, which displays values in kg/cm^2 (0–10 kg). The mean of 3 trials was calculated and used for the main analysis. A 30-s resting period was allowed between each trial. The reliability of pressure algometry has been found to be high the same day (ICC = 0.91 [95% CI 0.82–0.97]) (Chesterson et al. 2007) and between 4 separate days (ICC = 0.94–0.97) (Jones et al. 2007).

Study protocol

The study protocol was the same for shoulder patients and healthy controls. A 11-point numerical point rate scale (Jensen et al. 1999) (NPRS; 0 = no pain; 10 = maximum pain) was used to assess the intensity of current spontaneous pain and the pain experienced during arm elevation. Patients were asked to draw the distribution of their pain symptoms on an anatomic body map. None of the patients were taking any analgesic drug at the time the study was performed. Participants were asked to avoid any analgesic or muscle relaxant 72 h prior to the examination. Patients were examined when their rest pain intensity was less than 3 on a NPRS. All examinations were unilaterally conducted over the dominant-right arm, since all patients had the dominant-right shoulder affected.

PPT was first assessed over levator scapulae (2 cm superior to the superior angle of the scapula), supraspinatus (middle point over the fossa of the scapula), infraspinatus (muscle belly), pectoralis major (middle point under clavicle), major biceps (halfway between the coracoid process and the radial head), and tibialis anterior (halfway between the most superior attachment to the tibia and its tendon in the upper one-third of the muscle belly) muscles (Fig. 2). The order of point assessment was randomized between participants.

Secondly, myofascial TrPs in the levator scapulae, supraspinatus, infraspinatus, subscapularis, pectoralis major, and biceps brachii muscles were explored. The order of TrP evaluation was also randomized between participants. Both explorations were done by the same assessor who was blinded to the subjects' condition.

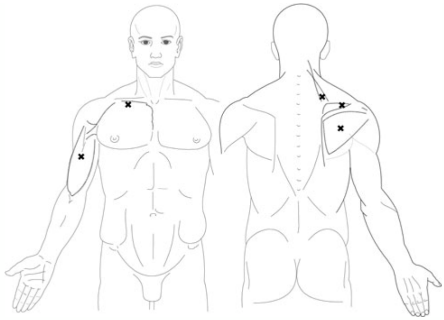

Fig. 2 Location of the points for pressure pain threshold assessment

Pressure pain threshold data management

In the current study, the magnitude of sensitization was investigated assessing the differences of absolute and relative PPT values between both groups. For relative values, we calculated a "PPT index" dividing PPT of each patient at each point by the mean of PPT score of the control group at the same point. A lower PPT Index (%) indicates greater degree of sensitization.

Statistical analysis

Data were analyzed with the SPSS statistical package (16.0 Version). Results are expressed as mean, standard deviation (SD), or 95% confidence interval (95% CI). The Kolmogorov–Smirnov test was used to analyze the normal distribution of the variables ($P > 0.05$). Quantitative data without a normal distribution (pain history, levels of pain, total number of muscle TrP, and number of latent or active TrPs) were analyzed with non-parametric tests and those data with a normal distribution (PPT) were analyzed with parametric tests. Differences in the number of myofascial TrPs (total, active, or latent TrPs) between groups were assessed with the non-parametric *U*-Man Whitney test. The chi square (χ^2) test was used to analyze the differences in the size of the distribution of muscle TrPs (active or latent) for each muscle within both study groups. Differences in PPT between both study groups were assessed with the unpaired Student's *t*-test. A one-way analysis of variance (ANOVA) test was used for assessing the differences in "PPT Index" between points. The non-parametric Kruskal–Wallis test was used to analyze the differences in the clinical pain variables between patients with non-TrPs, latent TrPs, or active TrPs within each analyzed muscle. A one-way ANOVA test was used to analyze the differences in PPT between patients with non-TrPs, latent TrPs, or active TrPs within each analyzed muscle. The Bonferroni test was used as post hoc analysis in all multiple comparisons. The Spearman's rho (r_s) test was used to analyze the association between the number of TrPs (total, active,and latent) with those variables relating to pain symptoms and with PPT levels. Finally, the Spearman's rho (r_s) was also used to investigate the association between clinical variables and PPT over each point. The statistical analysis was conducted at 95% confidence level, and a P value less than 0.05 was considered statistically significant.

Results

Demographic and clinical data of the patients

Twelve patients, 7 men and 5 women, aged 20–38 (mean: 25 ± 9 years) diagnosed with unilateral shoulder impingement, and 10 matched controls, 5 men and 5 women, aged 20–38 (mean: 26 ± 8 years) were included ($P = 0.497$). All patients reported pain located in the anterior and posterior parts of the shoulder region, and 5 patients also reported pain in the dorso-lateral aspect of the forearm.

The mean duration of shoulder pain history was 8.5 months (95% CI 5–12). The mean spontaneous resting level of shoulder pain was 3.5 (95% CI 2.5–4.2), whereas the level of pain experienced during arm active elevation was 7 (95% CI 5.5–8). No correlation was found between shoulder pain history and the pain intensity.

Muscle TrPs in patients with shoulder impingement and healthy controls

The mean ± SD number of TrPs for each shoulder impingement patient was 4.5 ± 1 of which 2.5 ± 1 were active TrPs, and the remaining 2 ± 1 were latent TrPs. Healthy controls only had latent TrPs (mean ± SD: 1 ± 1). Therefore, the number of TrP between both groups was significantly different for both active TrPs ($z = -4.207$; $P < 0.001$) and latent TrPs ($z = -3.042$; $P = 0.003$).

The distribution of myofascial TrPs between patients and healthy controls was significantly different for the levator scapulae ($\chi^2 = 18.471$, $P < 0.001$), supraspinatus ($\chi^2 = 10.831$, $P = 0.004$), infraspinatus ($\chi^2 = 15.278$, $P < 0.001$), pectoralis major ($\chi^2 = 7.374$, $P = 0.03$), and biceps brachii ($\chi^2 = 6.926$, $P = 0.03$), but not for the subscapularis ($\chi^2 = 5.683$, $P = 0.07$), muscles. Active TrPs within the supraspinatus ($n = 8$, 67%), infraspinatus ($n = 5$, 42%), and subscapularis ($n = 5$, 42%) muscles were the most prevalent within the patient group. Table 1 summarizes the distribution of muscle TrPs for all muscles in both patients and healthy controls, and Table 2 details the number of active and latent TrPs in each patient or healthy control.

Pressure pain thresholds in patients with unilateral shoulder impingement

Patients with shoulder impingement showed significant lower PPT levels in all muscles when compared to controls: levator scapulae ($t = -6.665$; $P < 0.001$), supraspinatus ($t = -6.243$; $P < 0.001$), infraspinatus ($t = -6.984$; $P < 0.001$), pectoralis major ($t = -8.400$; $P < 0.001$), biceps brachii ($t = -4.277$; $P < 0.001$), and tibialis anterior ($t = -6.198$; $P < 0.001$) muscles (Table 3).

The ANOVA revealed significant differences for PPT indices between sites ($F = 6.215$; $P < 0.001$). The post hoc analysis revealed a greater PPT index (lesser degree of sensitization) in the biceps brachii muscle when compared to those indices of the levator scapulae ($P = 0.008$), supraspinatus ($P = 0.045$) infraspinatus ($P = 0.01$), and pectoralis major ($P = 0.01$) muscles, but not when compared to the tibialis anterior ($P = 0.9$) (Fig. 3).

Trigger point activity, shoulder pain, and PPT levels

Within the patient group, a significant positive correlation was found between the total number of TrPs and spontaneous pain intensity ($r_s = 0.578$; $P = 0.045$): the greater the

Table 1 : Distribution of myofascial trigger points (TrPs) in subjects with shoulder impingement and healthy controls

	Levator scapulae	Supraspinatus	Infraspinatus	Subscapularis	Pectoralis major	Biceps brachii
Patients with unilateral shoulder impingement syndrome						
Active TrPs (n)	5	8	5	5	2	2
Latent TrPs (n)	7	0	5	3	6	8
Non-TrPs (n)	0	4	2	4	4	2
Healthy control subjects						
Active TrPs (n)	0	0	0	0	0	0
Latent TrPs (n)	1	1	0	3	1	3
Non-TrPs (n)	9	9	10	7	9	7

Table 2 Number of active and latent myofascial trigger points in each subject with shoulder impingement and healthy control

		Number of active TrPs	Number of latent TrPs
Patients with unilateral shoulder impingement syndrome			
Patient	1	2	3
	2	2	3
	3	3	2
	4	3	3
	5	2	4
	6	2	2
	7	2	1
	8	2	2
	9	1	2
	10	2	2
	11	3	2
	12	2	3
Healthy control subjects			
Control	1	0	2
	2	0	0
	3	0	0
	4	0	2
	5	0	2
	6	0	1
	7	0	0
	8	0	2
	9	0	0
	10	0	0

pain intensity, the greater the total number of muscle TrPs. No correlation was found between duration of pain symptoms and number of TrPs ($P > 0.8$).

Further, the Kruskal–Wallis test revealed that pain experienced during arm elevation was related to the presence of TrPs in the biceps brachii ($F = 6.817$; $P < 0.015$) and subscapularis ($F = 4.379$; $P = 0.045$): those patients with TrPs, either active or latent in these muscles, showed greater levels of pain experienced during arm elevation than those patients not diagnosed with TrPs in the same muscles.

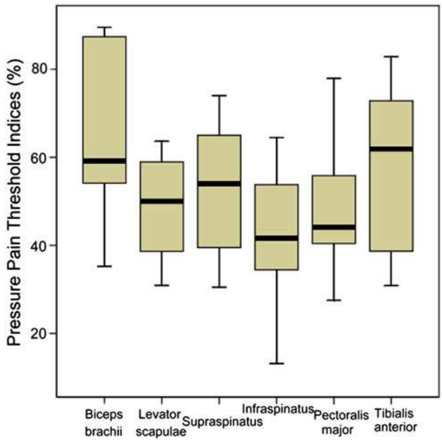

Fig. 3 Pressure pain threshold indices. The *boxes* represent the mean and the 25 and 75 percentile scores, and the *error bars* represent the standard deviation

In addition, spontaneous pain intensity was related to the presence of active TrPs in the supraspinatus ($t = -2.257$; $P = 0.045$) and infraspinatus ($F = 4.259$; $P = 0.045$) muscles. In such a way, patients with active TrPs in these muscles showed greater levels of pain experienced during arm elevation than those not diagnosed with TrP in the same muscles. Table 4 summarizes clinical pain variables depending on TrP activity on each examined muscle.

Additionally, significant negative correlations were found between the total number of TrPs and PPT levels over the biceps brachii ($r_s = -0.759$; $P = 0.004$) and the pectoralis major ($r_s = 0.771$; $P = 0.003$) muscles. Similar correlations were also found between the number of active TrPs and PPT over the biceps brachii: ($r_s = -0.645$;

Table 3 Pressure pain thresholds (PPT, kg/cm^2) in patients with shoulder impingement syndrome and healthy controls

	Patients with unilateral shoulder impingement#	Healthy control subjects
Levator scapulae muscle	1.6 ± 0.4 (95% CI 1.4–1.9)	3.3 ± 0.8 (95% CI 2.8–3.9)
Supraspinatus muscle	2.1 ± 0.6 (95% CI 1.7–2.5)	4.0 ± 0.8 (95% CI 3.4–4.6)
Infraspinatus muscle	1.9 ± 0.6 (95% CI 1.5–2.3)	4.5 ± 1.1 (95% CI 3.7–5.2)
Pectoralis major muscle	1.2 ± 0.4 (95% CI 1.0–1.4)	2.4 ± 0.3 (95% CI 2.2–2.6)
Biceps brachii muscle	1.5 ± 0.4 (95% CI 1.2–1.8)	2.4 ± 0.5 (95% CI 2.0–2.6)
Tibialis anterior muscle	3.4 ± 1.1 (95% CI 2.7–4.1)	6.0 ± 0.9 (95% CI 5.4–6.6)

Values are expressed as means ± standard deviation (95% confidence interval)

\# Significant lower PPT values when compared to healthy controls

Table 4 Shoulder pain characteristics depending on the presence of myofascial trigger points (trps) on each muscle within patients with shoulder impingement syndrome

		Spontaneous pain	Pain during shoulder movement
Levator scapulae muscle	Active TrPs ($n = 7$)	3.8 ± 1.3	7.8 ± 1.2
	Latent TrPs ($n = 5$)	2.4 ± 2.1	6.1 ± 3.7
	No TrPs ($n = 0$)	–	–
Supraspinatus muscle	Active TrPs ($n = 8$)	3.8 ± 1.9#	8.3 ± 1.3
	Latent TrPs ($n = 0$)	–	–
	No TrPs ($n = 4$)	1.5 ± 0.6	6.6 ± 2.9
Infraspinatus muscle	Active TrPs ($n = 5$)	4.0 ± 1#	8.6 ± 1.4
	Latent TrPs ($n = 5$)	4.0 ± 1.2	7.5 ± 1.2
	No TrPs ($n = 2$)	1.6 ± 0.5	5.5 ± 3.1
Subscapularis muscle	Active TrPs ($n = 5$)	4.0 ± 1.0	8.4 ± 1.2#
	Latent TrPs ($n = 3$)	3.4 ± 2.5	7.7 ± 1.4
	No TrPs ($n = 4$)	1.8 ± 1.0	4.3 ± 3.8
Pectoralis major muscle	Active TrPs ($n = 2$)	3.7 ± 2.5	8.1 ± 1.7
	Latent TrPs ($n = 6$)	3.0 ± 1.7	7.2 ± 0.2
	No TrPs ($n = 4$)	1.5 ± 0.7	5.6 ± 3.8
Biceps brachii muscle	Active TrPs ($n = 2$)	4.0 ± 1.4	8.5 ± 1.9#
	Latent TrPs ($n = 8$)	3.2 ± 2.0	7.9 ± 1.1
	No TrPs ($n = 2$)	1.0 ± 0.0	2.9 ± 4.0

Values are expressed as means ± standard deviation/NPRS = Numerical Pain Rate Scale (0–10)

Significantly different between both TrPs subgroups and non-TrP subgroup (ANOVA test, Bonferroni, $P < 0.01$)

$P = 0.025$) and the pectoralis major ($r_s = 0.690$; $P = 0.015$) muscles. In such a way, the greater the total number of TrPs, particularly active TrPs, the lower was the PPT over the biceps brachii and pectoralis major muscles.

Finally, significant differences in PPT levels were also found to be dependent on TrP activity: (a) levator scapulae TrPs were related to lower PPT over the levator scapulae ($t = 2.606$; $P = 0.025$) and biceps brachii ($t = 3.970$; $P = 0.003$) muscles; (b) TrPs in the supraspinatus muscle were related to lower PPT over the levator scapulae ($t = 3.716$; $P = 0.004$), supraspinatus ($t = 2.236$; $P = 0.045$), pectoralis major ($t = 3.571$; $P = 0.005$), and biceps brachii ($t = 2.503$; $P = 0.03$) muscles; (c) infraspinatus muscle TrPs were related to lower PPT level over the levator scapulae ($F = 6.898$; $P = 0.015$) muscle; (d) subscapularis muscle TrPs were related to lower PPT levels over the levator scapulae ($F = 8.246$; $P = 0.009$), supraspinatus ($F = 5.606$; $P = 0.025$), pectoralis major ($F = 7.249$; $P = 0.015$), and biceps brachii ($F = 9.505$; $P = 0.001$) muscles; and (e) biceps brachii TrPs were related to lower PPT over the biceps brachii ($F = 4.825$; $P = 0.04$) and pectoralis major ($F = 4.368$; $P = 0.04$) muscles. Table 5 shows PPT depending on TrP activity on each examined muscle.

Pressure pain sensitivity and clinical features in unilateral shoulder impingement

Within the patient group, significant negative correlations between spontaneous pain intensity and PPT over the levator scapulae ($r_s = -0.637$; $P = 0.025$), supraspinatus ($r_s = -0.577$; $P = 0.045$), and biceps brachii ($r_s = -0.680$; $P = 0.015$) muscles were found: the greater the pain intensity, the lower the PPT levels.

Discussion

The current study showed the existence of active TrPs in the shoulder muscles in patients with unilateral shoulder impingement. Both the local and the referred pain areas elicited by manual exploration of active TrPs reproduced the pain pattern in all patients. In addition, patients with unilateral shoulder impingement showed lower PPT levels when compared to healthy controls. A greater number of TrPs and lower PPT were related to greater pain intensity: the greater the pain intensity, the greater the number of TrPs and the lower the PPT. Finally, PPT levels were lower in some muscles in patients with active TrPs when compared to those patients without TrPs. The current results suggest both peripheral and central sensitization is present in patients with shoulder impingement syndrome.

Muscle TrPs in shoulder impingement syndrome

The rotator cuff is formed by the supraspinatus, the infraspinatus, the teres minor, and the subscapularis (Keating et al. 1993). Active muscle TrPs in the supraspinatus, infraspinatus, and subscapularis muscles elicited a referred pain that mimicked the patients' usual shoulder pain. Further, active TrPs in the levator scapulae, biceps brachii, and pectoralis major were also found. When active TrPs were explored, patients spontaneously reported: "Yes, this is exactly the pain that I usually feel either spontaneously, but particularly during arm elevation". These findings support the view that active TrPs in the neck–shoulder musculature are involved in the pathophysiology of shoulder impingement syndrome and that the referred pain sensations may contribute directly to shoulder pain complaint.

Active TrPs, by definition, were not found in healthy controls, since they did not suffer from any pain symptoms. In addition, shoulder impingement syndrome subjects also showed latent TrPs in the examined muscles in a greater proportion than healthy controls. Lucas et al. found that latent TrPs disturb normal pattern of motor recruitment and movement efficiency suggesting the clinical relevance of latent TrPs (Lucas et al. 2004). Further, it has been proposed that latent TrPs may become active under the

Table 5 Pressure pain thresholds (PPT) depending on the presence of myofascial trigger points (TrPs) on each muscle within patients with shoulder impingement syndrome

		Levator scapulae	Supraspinatus	Infraspinatus	Pectoralis major	Biceps brachii	Tibialis anterior
Levator scapulae	Active TrPs ($n = 7$)	1.3 ± 0.3*	1.9 ± 0.5	1.6 ± 0.7	1.0 ± 0.2	1.1 ± 0.2*	3.5 ± 1.1
	Latent TrPs ($n = 5$)	1.8 ± 0.3	2.2 ± 0.6	2.1 ± 0.5	1.4 ± 0.4	1.8 ± 0.3	3.4 ± 1.1
	No TrPs ($n = 0$)	–	–	–	–	–	–
Supraspinatus	Active TrPs ($n = 8$)	1.4 ± 0.3#	1.9 ± 0.5#	1.8 ± 0.7	1.0 ± 0.2#	1.3 ± 0.4#	3.3 ± 1.1
	Latent TrPs ($n = 0$)	–	–	–	–	–	–
	No TrPs ($n = 4$)	2.0 ± 0.1	2.6 ± 0.4	2.2 ± 0.4	1.5 ± 0.4	1.9 ± 0.3	3.7 ± 1.2
Infraspinatus	Active TrPs ($n = 5$)	1.4 ± 0.2#	1.4 ± 0.3	1.7 ± 0.7	1.0 ± 0.2	1.3 ± 0.5	3.3 ± 1.3
	Latent TrPs ($n = 5$)	1.4 ± 0.3	2.4 ± 0.5	2.0 ± 0.4	1.4 ± 0.4	1.7 ± 0.4	3.5 ± 1.0
	No TrPs ($n = 2$)	1.9 ± 0.2	2.0 ± 0.5	2.2 ± 0.9	1.0 ± 0.1	1.5 ± 0.3	3.3 ± 1.3
Subscapularis	Active TrPs ($n = 5$)	1.3 ± 0.3#	1.8 ± 0.4#	1.5 ± 0.9	1.0 ± 0.1#	1.1 ± 0.3#	2.8 ± 1.1
	Latent TrPs ($n = 3$)	1.5 ± 0.3	1.8 ± 0.6	1.9 ± 0.5	1.0 ± 0.3	1.4 ± 0.2	4.0 ± 1.0
	No TrPs ($n = 4$)	2.0 ± 0.1	2.7 ± 0.2	2.1 ± 0.4	1.6 ± 0.4	2.0 ± 0.1	3.7 ± 1.1
Pectoralis major	Active TrPs ($n = 2$)	1.4 ± 0.2	1.7 ± 0.6	1.6 ± 0.5	1.1 ± 0.1	1.3 ± 0.3	3.9 ± 1.1
	Latent TrPs ($n = 6$)	1.7 ± 0.4	2.1 ± 0.5	2.4 ± 0.1	1.1 ± 0.4	1.4 ± 0.5	3.7 ± 0.9
	No TrPs ($n = 4$)	2.0 ± 0.1	2.5 ± 0.2	2.1 ± 0.7	1.7 ± 0.3	2.0 ± 0.1	4.0 ± 0.1
Biceps brachii	Active TrPs ($n = 2$)	1.3 ± 0.4	2.0 ± 0.8	1.5 ± 1.3	0.9 ± 0.3#	1.1 ± 0.3#	3.1 ± 1.1
	Latent TrPs ($n = 8$)	1.6 ± 0.3	2.0 ± 0.6	2.0 ± 0.5	1.1 ± 0.3	1.5 ± 0.4	4.0 ± 1.3
	No TrPs ($n = 2$)	2.1 ± 0.1	2.7 ± 0.1	2.1 ± 0.6	1.7 ± 0.3	2.0 ± 0.1	4.1 ± 0.5

Values are expressed as means ± standard deviation

* Significant differences between active and latent TrP subgroups (Student *t*-test, $P < 0.03$)

Significant differences TrP and non-TrP subgroups (ANOVA test, Bonferroni, $P < 0.01$)

influence of several factors such as repetitive and sustained shoulder activities (Simons 2004). Therefore, it may be that the presence of muscle TrPs, either active or latent, may be implicated in the sensory-motor disturbances often observed in patients with shoulder impingement syndrome (Ludewig and Cook 2000; Tyler et al. 2005; Moraes et al. 2008). In such a way, we do not know if inactivation of muscle TrPs can prevent recurrence of symptoms, which are very common in this patient population (Mitchell et al. 2005). Our results underline the importance of inspection and inactivating TrPs in the shoulder muscles in patients with shoulder impingement syndrome as they may contribute to the overall picture of pain. Two randomized trials are in progress in order to elucidate the role of inactivation of TrPs in patients with shoulder impingement syndrome (Bron et al. 2007a; Perez-Palomares et al. 2009).

An interesting finding was that the number of TrPs was related to a greater pain intensity of the symptoms. Further, the presence of active TrPs in different muscles was related to a greater intensity of spontaneous pain (supraspinatus/infraspinatus) and pain during arm elevation (biceps brachii/subscapularis). These findings further support the role of active TrPs within the shoulder musculature in shoulder impingement syndrome. Further, a greater number of muscle TrPs suggest the presence of spatial summation of TrP pain activity in shoulder impingement related to the intensity of the pain symptoms. Spatial summation of TrP pain activity has been also suggested in chronic tension type headache (Fernández-de-las-Peñas et al. 2007e). In fact, we do not know if the presence of numerous TrPs is responsible for shoulder pain symptoms in these patients or that muscle TrPs are activated due to pain. Although future longitudinal studies are needed to answer this question, it is more conceivable that TrPs would be responsible for pain symptoms.

Multiple active TrPs in the same muscle (i.e. infraspinatus) have been previously described in patients with shoulder pain (Ge et al. 2008). The present study is the first to report the presence of TrPs in multiple and different shoulder muscles, particularly those forming the rotator cuff. Nevertheless, it may be possible that the muscles examined in this study also showed multiple active TrPs. Moreover, Ge et al. (2008) found latent TrPs within the infraspinatus muscle on the asymptomatic side in patients with unilateral shoulder pain. It is not know if patients with unilateral shoulder impingement syndrome have muscle TrPs in the shoulder musculature within the unaffected side. Future studies investigating bilaterally the presence of multiple muscle TrPs in patients with unilateral shoulder impingement syndrome are needed.

Mechanical pain hypersensitivity in shoulder impingement syndrome

In this study, PPT levels were significantly decreased over the levator scapulae, supraspinatus, infraspinatus, biceps brachii, and pectoralis major muscles in patients with unilateral shoulder impingement syndrome when compared to healthy controls, which suggests a sensitization of muscle tissues in this patient population. This is expected since all examined muscles are involved in arm motion. These findings suggest the presence of segmental sensitization mechanisms as the examined muscles received innervation from the same segments of the cervical spine (C4–C6 segments). Consistent with a significant decrease in PPT levels over the shoulder muscles, we also found lower PPT levels in the tibialis anterior muscle suggesting multi-segmental sensory sensitization or sensitization of the central nervous system in unilateral shoulder impingement. However, we should recognize that we only investigated PPT levels over the affected side.

Nevertheless, it seems that there is a greater sensitization degree in the shoulder musculature, which is supported by the fact that the magnitude of PPT changes was higher for the levator scapulae ($49 \pm 11\%$), supraspinatus ($52 \pm 14\%$), infraspinatus ($42 \pm 13\%$), and pectoralis major ($49 \pm 15\%$) muscles when compared to the magnitude of PPT changes over the tibialis anterior ($58 \pm 18\%$) muscle.

Finally, there is no consensus about the differences in PPT levels that are needed to consider real changes between groups (Sterling 2008). Different studies conducted over the cervical spine (Chesterson et al. 2007; Ylinen et al. 2007) have suggested that differences ranging from 123 to 200 kPa (1.2–2 kg) are needed to consider real PPT differences. In the current study, differences between symptomatic (1.0–2.1 kg) and non-symptomatic (2.6 kg) regions were placed within this interval, so differences between both groups can be considered as real. However, we should consider that these studies investigating PPT changes were conducted over the cervical spine, so extrapolation of their results to the shoulder region should be done with caution.

Sensitization mechanisms associated with muscle TrPs in shoulder impingement

Pressure pain thresholds (PPT) (Chesterton et al. 2003; Rolke et al. 2005) are extensively used for investigating mechanical pain hypersensitivity in different localized pain conditions, e.g. whiplash (Sterling et al. 2003), unilateral migraine (Fernández-de-las-Peñas et al. 2008), repetitive strain injury (Greening and Lynn 1998), lateral epicondylalgia (Fernández-Carnero et al. 2009), chronic tension type headache (Fernández-de-las-Peñas et al. 2007f), low back pain (O'Neill et al. 2007), knee osteoarthritis (Bajaj et al. 2001), and carpal tunnel syndrome (Fernández-de-las-Peñas et al. 2009). These studies have consistently showed lower PPT levels in both painful and distant pain-free areas, suggesting both segmental and extra-segmental spreading of hyperexcitability.

The results of the current study reflect the presence of peripheral and central sensitization mechanisms in patients with unilateral shoulder impingement syndrome. The presence of active TrPs in the shoulder musculature suggests sensitization of muscle nociceptors since high levels of algogenic substances (Shah et al. 2005, 2008) and lower pressure pain thresholds (Ge et al. 2008) has been found in active muscle TrPs. Additionally, a study has recently demonstrated the existence of both nociceptive and non-nociceptive hypersensitivity at muscle TrPs (Li et al. 2009). These studies support that active, and also latent, muscle TrPs constitute a focus of peripheral nociceptive sensitization of both nociceptive and non-nociceptive nerve endings, evidencing the relevance of muscle TrPs for sensitization mechanisms.

We found that pressure pain hypersensitivity was negatively related to the number of active TrPs: the greater the number of active TrPs, the lower the PPT levels. Further, the presence of muscle TrPs within the shoulder muscles was also related to lower PPT in different muscles. Our findings suggest that the higher hyperalgesia may come from spatial summation of TrP-related pain in the shoulder musculature. This may also indicate that multiple active TrPs spatially increase the mechanical pain sensitivity peripherally and centrally, since PPT were not measured directly on the TrP, but on fixed points over the muscles. We could not assess PPT at TrPs since we did not know the existence of TrPs at the beginning of the study. Then, PPT levels were assessed at fixed points at the belly of the muscles in which we looked for the presence of TrPs, except for the subscapularis muscle (for practical reasons) and the tibialis anterior (non-painful point). Finally, Ge et al. reported that the association of multiple active muscle TrPs and the heterogeneity of mechanical pain hypersensitivity distribution suggest a crucial role of peripheral sensitization in unilateral shoulder pain (Ge et al. 2008).

Nevertheless, we can not exclude a role of central sensitization mechanisms in the presence of muscle TrPs. In fact, the existence of sensitization mechanisms in local pain syndromes suggests that sustained peripheral noxious input to the central nervous system plays a role in the initiation and maintenance of sensitization processes (Mendell and Wall 1965) since central sensitization is considered as a dynamic condition influenced by multiple factors including the activity of peripheral nociceptive inputs (Herren-Gerber et al. 2004). In the current study, the decrease in PPT levels was associated with the intensity of pain symptoms, supporting a role of the peripheral nociceptive input as an important factor driving the development of spreading sensitization.

We found up to 3 active TrPs within each patient with shoulder impingement, supporting the assumption of spatial summation of TrP activity in these patients. Since active TrPs constitute a peripheral sensitization focus, the presence of multiple active TrPs may exert a spatial summation of nociceptive barrage to the dorsal horn neurons. Fernández-de-las-Peñas et al. formulated a pain model for patient with chronic tension type headache involving peripheral sensitization from active muscle TrPs and central sensitization mechanisms (Fernández-de-las-Peñas et al. 2007g). It is possible that similar sensitization mechanisms occur in shoulder impingement syndrome, although longitudinal studies are needed in order to further elucidate the role of muscle TrPs to the development of shoulder impingement syndrome.

Strengths and limitations of the study

Several methodological aspects of the current study should be mentioned. First, TrP examination was conducted by a blinded examiner ruling out of the chance of bias. Since manual palpation was done without any feedback of the participant about reproduction of pain symptoms, the examiner remained blinded until the end of the examination. This procedure has been used in previous studies (Fernández-Carnero et al. 2007; Fernández-de-las-Peñas et al. 2007a, b, c, d). Nevertheless, it is possible that a memory bias from any muscle can be present (Table 2). Second, we included a small sample size. Nevertheless, the results seem robust, which suggest that a greater sample size would not alter the direction of the results. Population-based epidemiological studies with greater sample sizes are now needed to permit a more generalized interpretation of these results. Finally, the third limitation of the current study was that we can not establish a cause-and-effect relationship between TrPs and shoulder impingement syndrome, because the design was not longitudinal and because the paper did not report the results of inactivating the active TrPs.

Conclusion

The current controlled study showed the existence of multiple active TrPs in the shoulder muscles in patients with unilateral shoulder impingement. Both local and referred pain elicited by manual exploration of active muscle TrPs reproduced the pain pattern in all patients. Patients showed pressure pain hyperalgesia in painful and non-painful distant areas, suggesting the presence of central sensitization. A greater number of TrPs and lower PPT levels were related to greater pain intensity: the greater the pain intensity, the greater the number of TrPs and the lower the PPT. Finally, active TrPs were related to lower PPT. Our results suggest both peripheral and central sensitization mechanisms in patients with shoulder impingement syndrome.

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Nadadores de élite con y sin síndrome subacromial: hiperalgesia mecánica y puntos gatillo en músculos del complejo hombro-cuello.

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Elite swimmers with and without unilateral shoulder pain: mechanical hyperalgesia and active/latent muscle trigger points in neck–shoulder muscles

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Our aim was to investigate the presence of mechanical hypersensitivity and active trigger points (TrPs) in the neck–shoulder muscles in elite swimmers with/without unilateral shoulder pain. Seventeen elite swimmers with shoulder pain; 18 swimmers without shoulder pain; and 15 elite athletes matched controls were recruited. Pressure pain thresholds (PPT) were assessed over the levator scapulae, sternocleidomastoid, upper trapezius, infraspinatus, scalene, subscapularis and tibialis anterior muscles. TrPs in the levator scapulae, upper trapezius, infraspinatus, scalene, sternocleidomastoid and subscapularis muscles were also explored. Swimmers with shoulder pain showed significant lower PPT in all muscles compared with controls ($P<0.01$). No differences in PPT were found between swimmers with and without shoulder pain, underlining widespread mechanical hypersensitivity. The mean number of TrPs for elite swimmer with and without shoulder pain was, respectively, 4.7 ± 1 (2.1 ± 1.5 active; 2.6 ± 1.4 latent) and 4.7 ± 1.3 (1.3 ± 1.3 active; 3.4 ± 1.5 latent), whereas healthy athletes only showed latent TrPs (2.4 ± 1.2). Elite swimmers with shoulder pain showed higher number of active TrPs than swimmers without pain, whereas it was the opposite for the number of latent muscle TrP ($P<0.05$). The reported mechanical hypersensitivity suggests that active TrPs play a role in the development of shoulder pain in elite swimmers.

Shoulder pain is highly important in elite swimmers as inherent biomechanics to swimming promote muscular imbalances that stress the capsule-ligamentous structures and contribute to shoulder instability (O'Donnell et al., 2005). The prevalence of shoulder pain in swimmers is slightly superior to the general population, ranging between 42% and 73% (McMaster & Troup, 1993; Allegrucci et al., 1994; Bak, 1996). Q1

The etiology of shoulder pain in elite swimmers is unclear. Jobe et al. (1989) proposed the "instability complex" hypothesis, which suggests that forceful overhead activity can cause gradual stretching of anterior-inferior capsule-ligamentous structures leading to laxity, instability and impingement; however, no study has confirmed this hypothesis yet (Sein et al., 2010). Shoulder pain in elite swimmers can be related to repetitive overhead shoulder movements engaged during swimming, which may increase joint laxity and supraspinatus tendinopathy (Soslowsky et al., 2000; Sein et al., 2010). Relatively inappropriate stroke technique (Yanai & Hay, 2000) and training load among swimmers are thought to lead to supraspinatus tendinopathy associated with shoulder pain (Soslowsky et al., 2000; Sein et al., 2010). Shoulder pain and increased mechanical pain sensitivity are common after extensive training (Nie et al., 2005; Binderup et al., 2010).

Although the etiology of shoulder pain is not completely understood, there is evidence showing the role of shoulder muscle imbalance as a potential-related factor to this condition (Rupp et al., 1995; Olivier et al., 2008). For instance, elite swimmers have demonstrated decreases in internal rotation after their training season, which could be associated to an increased neuromuscular control of the scapulae muscles (Thomas et al., 2009). Su et al. (2004) found a decrease in scapular upward rotation in swimmers with shoulder pain. Wadsworth and Bullock-Saxton (1997) showed a significant

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variability in the timing of activation of the serratus anterior and upper and lower trapezius muscle in swimmers with shoulder pain. Because of this imbalance in muscle activation, Simons et al. (1999) suggested that myofascial trigger points (TrPs) may play a relevant role in shoulder pain.

TrPs are defined as hypersensitive spots in a taut band of skeletal muscles that are painful on contraction, stretching or palpation and give rise to a referred pain distant from the spot (Simons et al., 1999). From a clinical point of view, TrPs may be active or latent. Active TrPs are those in which local and referred pains reproduce the patients' symptoms, whereas latent TrPs have the same clinical findings as active TrPs, but they are not responsible for symptoms (Simons et al., 1999). Clinical distinction between active and latent TrPs is substantiated by histochemical findings, i.e., higher levels of algogenic substances and chemical mediators (i.e., bradykinin, substance P or serotonin) have been found in active TrPs compared with latent TrPs and non-TrPs (Shah et al., 2005, 2008). For instance, Ge et al. (2008) found the presence of active TrPs in the infraspinatus muscle in patients with unilateral shoulder pain.

There is some evidence suggesting that TrPs may be implicated in the clinical picture of shoulder pain. Ingber (2000) described three patients presenting with shoulder pain successfully treated with subscapularis TrP injection. A recent clinical study has reported that local and referred pain elicited by active TrPs in the levator scapula, supraspinatus, infraspinatus, subscapularis, pectoralis major and biceps brachii muscles reproduce the pain pattern in patients with shoulder pain (Hidalgo-Lozano et al., 2010). These studies support the role of active TrP in shoulder pain development. However, to the best of our knowledge, there is to date no report on the presence of active TrPs in elite swimmers with/without shoulder pain.

In this study, we hypothesized that elite swimmers with shoulder pain would exhibit mechanical pain hyperalgesia and active muscle TrPs in the scapular region as compared with elite swimmers without shoulder pain and elite healthy athletes. For this purpose, we assessed pressure pain thresholds (PPT) as well as the presence and type of TrPs within the levator scapulae, sternocleidomastoid, upper trapezius, scalene, infraspinatus and subscapularis muscles in elite swimmers with and without unilateral shoulder pain and healthy elite athletes (controls).

Methods

Participants

Elite swimmers (Table 1) from four competitive swimming clubs of different countries (Spain, Portugal, Lithuania, Denmark and Brazil) located at High Altitude Sport Centre of Sierra Nevada (Granada, Spain) were screened for eligibility criteria. To be considered for the study, swimmers should fulfill the following criteria: 1, aged between 18 and 30 years; 2, have training for at least 2.5 years under coach supervision; and 3, swimming >6 h/week. Elite swimmers with and without unilateral shoulder pain were included. They were eligible if they had unilateral shoulder complaints (described as pain felt in the shoulder or arm) with duration of at least 3 months and an intensity of at least four on an 11-point numerical pain rating scale (NPRS) during arm elevation. They should report positive Neer (1983) and Hawkins (MacDonald et al., 2000) tests.

Exclusion criteria were 1, history of shoulder or neck surgery; 2, fracture of the shoulder region; 3, bilateral shoulder symptoms; 4, cervical radiculopathy; 5, previous interventions with steroid injections; or 6, any type of physical intervention in the neck–shoulder area within the last year.

In addition, age-matched right-handed elite athlete individuals (Table 1) were recruited as control subjects from volunteers training at the High Altitude Sport Centre of Sierra Nevada. They were excluded if they exhibited a history of neck, shoulder or arm pain, history of trauma or diagnosis of systemic diseases. Both Neer and Hawkin tests were negative. The study protocol was approved by the local Ethics Committee and conducted according to the Helsinki Declaration. All subjects signed an informed consent before their inclusion.

PPT

PPT is defined as the minimal amount of pressure where a sensation of pressure first changes to pain (Vanderweeen et al., 1996). A mechanical pressure algometer (Pain Diagnosis and Treatment Inc., Great Neck, New York, USA) was used in this study. The device consists of a 1-cm^2-rubber disk attached

Table 1. Anthropometric, sport and training characteristics of the participants

	Swimmers with shoulder pain ($n=17$)	Swimmers without shoulder pain ($n=18$)	Healthy athletes – control group ($n=15$)	P
Weight (kg)	73.7 ± 8.2	69.8 ± 12.1	64.5 ± 12.4	0.102
Height (cm)	179.9 ± 6.6	175.9 ± 9.2	172.8 ± 10.3	0.085
Body mass index (kg/m^2)	22.7 ± 1.5	22.6 ± 4.3	21.4 ± 2.1	0.408
Swimmer style (%)	2 (11.8%)	1 (5.6%)		
Breast stroke	10 (58.8%)	11 (72.0%)	–	
Freestyle	3 (17.6%)	2 (11.2%)		0.466
Butterfly	2 (11.8%)	2 (11.2%)		
Back stroke				
Begin training (years)	11.6 ± 3.4	8.9 ± 2.7	11.1 ± 5.5	0.107
Training hours (h/week)	26.8 ± 4.8	26.1 ± 5.5	23.4 ± 4.2	0.129

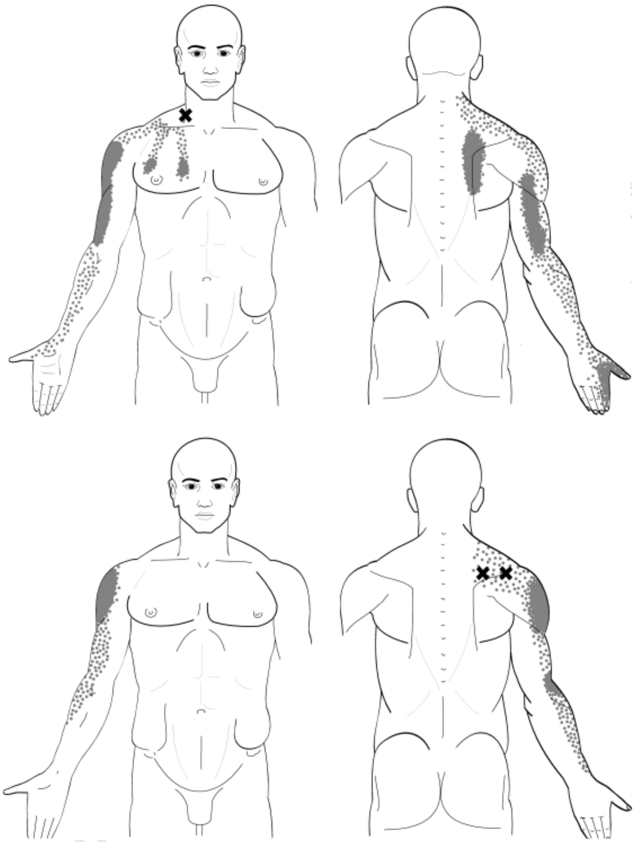

Fig. 1. Referred pain patterns from scalene (left) and infraspinatus (right) muscle trigger points as described by Simons et al. (1999).

to a strain gauge that displays values in kg/cm^2 (0–10 kg). The mean of three trials was calculated, converted to kPa (SI unit) and used for the analysis. A 30-s resting period was allowed between each recording. The reliability of pressure algometry has been found to be high the same day (ICC = 0.91, 95% confidence interval [95% CI 0.82–0.97]) (Chesterson et al., 2007) and between 4 separate days (ICC = 0.94–0.97) (Jones et al., 2007).

Muscle TrP examination

Muscle TrPs were explored in the levator scapulae, sternocleidomastoid, upper trapezius, scalene, infraspinatus and subscapularis muscles by an assessor with more than 8 years' of experience in muscle TrPs diagnosis and who was blinded to the subjects' condition. TrP diagnosis was performed following the criteria described by Simons et al. (1999) and by Gerwin et al. (1997): (1) palpable taut band in a skeletal muscle; (2) hypersensitive spot in the taut band; (3) local twitch response elicited by the snapping palpation of the taut band; and (4) presence of referred pain in response to compression. These criteria, when applied by an experienced assessor, have obtained a good inter-examiner reliability (κ) ranging from 0.84 to 0.88 (Gerwin et al., 1997).

TrPs were considered active when both the local and the referred pain evoked by stimulation reproduced the pain symptoms and when the subject recognized the pain as familiar pain. TrPs were considered latent when the local and the referred pain elicited by compression did not reproduce any familiar symptom (Simons et al., 1999). Figure 1 shows the referred pain evoked by TrPs in the scalene and infraspinatus muscles according to Simons et al. (1999).

TrP examination was performed in a blinded fashion. After TrP assessment on each muscle, the participant was asked: "When I pressed this muscle, did you feel any pain or discomfort locally, and in other areas (referred pain)? Please tell me whether the pain that you feel in the other area

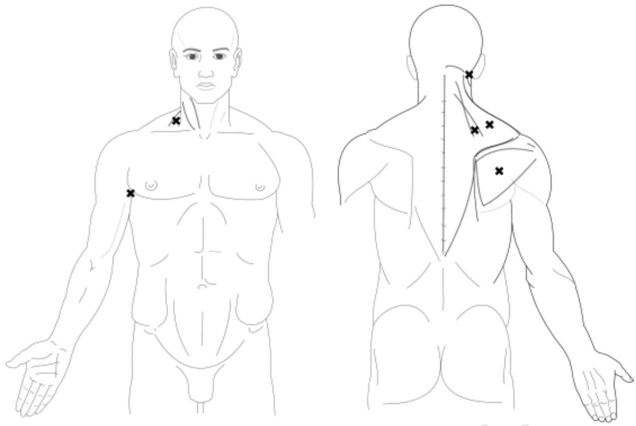

Fig. 2. Location of the points for pressure pain threshold assessment.

reproduced any symptoms that you suffered from?'' Participants had to indicate whether the pain elicited by palpation reproduced symptoms familiar to them or elicited different non-familiar pain.

Study protocol

The study protocol was the same all subjects. Each one completed a standardized survey training questionnaire administered by a sport medicine doctor, who requested the following information: number of years with coaching; hours per week in swimming training; level of competition (international, national, state, club); and weekly swimming distance. Each athlete was given a systematic clinical shoulder examination.

An 11-point NPRS (Jensen et al., 1999) (0: no pain; 10: maximum pain) was used to assess the intensity of current spontaneous pain at rest. Participants with pain were asked to draw the distribution of their pain symptoms on an anatomical body map. They were asked to avoid any analgesic or muscle relaxant 72 h before the examination, and they were examined when their pain intensity was <3 on the NPRS. All examinations were unilaterally conducted by an experienced assessor over the dominant arm.

PPTs were first assessed over levator scapulae (2 cm superior to the superior angle of the scapulae), sternocleidomastoid (on its insertion over the mastoid process), upper trapezius (halfway between C7 vertebra and acromion), scalene (2 cm superior to the clavicle over the anterior part of the transverse process of C6), infraspinatus (muscle belly mid point), subscapularis (muscle belly over the lateral border of the scapula) and tibialis anterior (halfway between the superior attachment to the tibia and its tendon in the upper one-third of the belly) muscles (Fig. 2). The order of assessment was randomized between participants.

Secondly, myofascial TrPs within levator scapulae, sternocleidomastoid, upper trapezius, scalene, infraspinatus and subscapularis muscles were explored. The order of TrP evaluation was also randomized between participants. Both explorations were carried out by the same assessor who was blinded to the subjects' condition.

Statistical analysis

Data were analyzed with the R-software (Auckland, New Zeland, 2.3.1 version). Results are expressed as mean, standard deviations (SD) and 95% CI. The Kolmogorov–Smirnov test was used to analyze the normal distribution of the data ($P>0.05$). Quantitative data without a normal distribution (training hours per week, pain history, pain intensity, total number of muscle TrP and number of latent or active TrPs) were analyzed with non-parametric tests and those data with a normal distribution (PPT) were analyzed with parametric tests. A one-way analysis of variance (ANOVA) test was used to analyze the differences in PPT levels within each point. The Bonferroni test was used as *post hoc* analysis in multiple comparisons. Differences in the number of muscle TrPs (total, active or latent TrPs) among groups were assessed with the non-parametric Krukal–Wallis test. A logistic regression was used to calculate the differences within the distribution of active and latent TrPs among groups. The χ^2-test was applied as *post hoc* analysis to investigate the differences in the distribution of muscle TrPs for each muscle between each study group in multiple comparisons. The Spearman rho (r_s) test was used to analyze the association between the numbers of TrPs (total, active, latent) with those variables relating to symptoms. The statistical analysis was conducted at 95% confidence level and a P-value <0.05 was considered statistically significant.

Results

Anthropometric and clinical data of the participants

Seventeen elite swimmers, nine men and eight women, aged 18–28 years (age: 21 ± 3 years) with unilateral shoulder pain; 18 elite swimmers, none men and nine women, aged 18–26 years (age: 20 ± 3 years) without shoulder pain; and 15 elite sports people (eight athletics/seven skiers)-matched controls, seven men and eight women, aged 16–28 years (mean: 23 ± 4 years) participated (age, $P = 0.137$). Among the swimmers with shoulder pain, 11 (65%) exhibited pain before and during training, and also six after training. Demographic, training and sport specialty are detailed in Table 1. The mean duration of shoulder pain history was 3 years (95% CI 2–3.4), and the spontaneous level of shoulder pain at rest was 4.8 (95% CI 4.0–5.7). No correlation among shoulder pain intensity, training hours per week or training years was found.

PPT

The ANOVA revealed significant differences among groups for PPT levels over scalene ($F = 4.4$; $P = 0.017$), sternocleidomastoid ($F = 5.2$; $P = 0.009$), upper trapezius ($F = 5.5$; $P = 0.007$), levator scapulae ($F = 3.6$; $P = 0.034$), infraspinatus ($F = 4.4$; $P = 0.018$), subscapularis ($F = 8.3$; $P = 0.001$) and tibialis anterior ($F = 5.8$; $P = 0.005$) muscles. *Post hoc* analysis showed reduced PPT levels in swimmers with shoulder pain as compared with elite athletes over all muscles ($P < 0.01$). Further, swimmers without pain also exhibited lower PPT over the upper trapezius, subscapularis and tibialis anterior as compared with elite athlete controls ($P < 0.01$). No significant differences between elite swimmers with and without shoulder pain were found. Table 2 details PPT levels over each point in the three groups.

Muscle TrPs

The mean $\pm$ SD number of TrPs for elite swimmers with shoulder pain was 4.7 ± 1, of which 2.1 ± 1.5 were active TrPs and the remaining 2.6 ± 1.4 were latent. The mean $\pm$ SD number of TrPs for each elite swimmer without shoulder pain was 4.7 ± 1.3, of which 1.3 ± 1.3 were active TrPs and the remaining 3.4 ± 1.5 were latent TrPs. Healthy athletes only showed latent TrPs (mean $\pm$ SD: 2.4 ± 1.2). Swimmers with and without shoulder pain had a higher number of TrPs compared with elite healthy controls ($P < 0.001$), but without significant differences between swimmers. The *post hoc* analysis revealed that swimmers with shoulder pain exhibited a higher number of active TrPs ($P = 0.045$) than swimmers without pain; and that swimmers without pain showed a higher a number of latent TrPs ($P = 0.041$) than those with pain.

Within elite swimmers with shoulder pain, no significant correlations between the total number of TrPs, the number of active TrPs or the number of latent muscle TrPs and the intensity of shoulder pain at rest were found.

Muscle TrPs in swimmers with shoulder pain and athletes (controls)

The distribution of TrP between elite swimmers with shoulder pain and elite athlete controls was significantly different for the levator scapulae, infraspinatus, subscapularis, upper trapezius and sternocleidomastoid ($P < 0.05$) muscles, but not for the scalene. Active TrPs in the upper trapezius ($n = 10$, 58.8%), levator scapulae ($n = 10$, 59%) and infraspinatus ($n = 6$, 35%) muscles were the most prevalent in elite swimmers with shoulder pain. Table 3 summarizes the distribution of TrPs for all muscles.

Muscle TrPs in elite swimmers with and without shoulder pain

The distribution of muscle TrPs between swimmers with and without shoulder pain was significantly different for sternoicleidomastoid ($P < 0.05$), but not for levator scapulae, scalene, upper trapezius, infraspinatus and subscapularis muscles. Active TrPs in the upper trapezius ($n = 10$, 55.6%) and infraspinatus ($n = 6$, 33.3%) muscles were the most prevalent in swimmers without pain (Table 3).

Muscle TrPs in swimmers without shoulder pain and athletes (Controls)

The distribution of muscle TrPs between swimmers without shoulder pain and elite athlete controls was significantly different for the upper trapezius, levator

Table 2. Pressure pain thresholds (PPT, kPa) in swimmers with/without shoulder pain and healthy athletes

	Swimmers with shoulder pain	Swimmers without shoulder pain	Healthy athletes
Upper trapezius muscle#*	166.7 ± 68.6 (95% CI 127.5–205.9)	191.2 ± 68.6 (95% CI 147.1–225.6)	274.6 ± 147.1 (95% CI 196.1–353)
Levator scapulae muscle#	205.9 ± 98 (95% CI 156.9–255)	245.2 ± 78.5 (95% CI 205.9–294.2)	310.2 ± 137.3 (95% CI 235.4–382.5)
Scalene muscle#	98 ± 39.2 (95% CI 78.5–117.7)	107.9 ± 39.2 (95% CI 88.3–127.5)	137.3 ± 3924 (95% CI (117.7–156.9)
Infraspinatus muscle#	225.6 ± 98 (95% CI 176.5–274.6)	255 ± 98 (95% CI 206–04)	343.2 ± 156.9 (95% CI 264.9–431.5)
Subscapularis muscle#*	117.7 ± 0.4 (95% CI 98–137.3)	147 ± 39.2 (95% CI 127.5–166.7)	205.9 ± 88.3 (95% CI 166.7–245.2)
Sternocleidomastoid muscle#	78.5 ± 39.2 (95% CI 58.8–107.9)	98.1 ± 39.2 (95% CI 78.5–117.7)	132.3 ± 29.4 (95% CI 107.9–147.1)
Tibialis anterior muscle#*	284.4 ± 166.7 (95% CI 215.7–372.7)	343 ± 137.3 (95% CI 284.4–421.7)	490.3 ± 186.3 (95% CI 292.3–588.4)

Values are expressed as means ± standard deviation (95% CI).
#Significant differences between swimmers with shoulder pain and healthy athletes.
*Significant differences between swimmers without shoulder pain and healthy athletes.
CI, confidence interval

Table 3. Distribution of myofascial trigger points (TrPS) in swimmers with/without shoulder pain and healthy athletes

	Levator scapulae	Scalene	Infraspinatus	Subscapularis	Sternocleidomastoid	Upper trapezius
Swimmers with shoulder pain ($n = 17$)						
Active TrPs	10	3	6	2	4	10
Latent TrPs	6	7	3	12	13	5
Non-TrPs	1	7	8	3	0	2
Swimmers without shoulder pain ($n = 18$)						
Active TrPs	6	1	5	1	0	10
Latent TrPs	10	10	9	9	17	8
Non-TrPs	2	7	4	8	1	0
Healthy athletes ($n = 15$)						
Active TrPs	1	0	0	0	0	2
Latent TrPs	7	5	2	4	11	8
Non-TrPs	7	10	13	11	4	5

scapulae and infraspinatus ($P < 0.05$), but not for scalene, sternocleidomastoid or subscapularis muscles (Table 3).

Discussion

The current study showed that elite swimmers with and without shoulder pain exhibited lower PPT levels as compared with controls. In addition, active TrPs in the shoulder muscles were also found in elite swimmers with unilateral shoulder pain. The local and the referred pain areas elicited by active TrPs reproduced the pain symptoms. Further, elite swimmers without shoulder pain showed latent TrP. The current results suggest a role of active TrPs in the development of shoulder pain in elite swimmers and the presence of mechanical hyperalgesia among elite swimmers.

Mechanical pain hypersensitivity in elite swimmers

Shoulder pain in swimmers is common and can be debilitating (Pollard & Croker, 1999). Pain is usually caused by swimming-specific demands such as increased shoulder range of motion, increased internal rotation/adduction strength and prolonged training causing fatigue (Weldon & Richardson, 2001). In this study, we hypothesized that mechanical hyperalgesia would characterized elite swimmers with shoulder pain but not elite swimmers without shoulder pain, or controls. Surprisingly, PPT was actually decreased in both elite swimmers with and without shoulder pain as compared with healthy athletes suggesting a general mechanical sensitization of neck and shoulder girdle tissues among elite swimmers. These findings suggest the presence of segmental sensitization mechanisms as the examined muscles received innervation from the neck region. Consistent with the significant decrease in PPT over the neck and shoulder muscles, we also found lower PPT levels in the tibialis anterior muscle suggesting multi-segmental sensory sensitization or central sensitization in elite swimmers with/without unilateral shoulder pain (Graven-Nielsen, 2006).

The presence of sensitization mechanisms is in agreement with several studies, which have reported widespread pressure hypersensitivity in different localized pain conditions, such as lateral epicondylalgia (Fernández-Carnero et al., 2009), tension-type headache (Fernández-de-las-Peñas et al., 2007), low back pain (O'Neill et al., 2007) or knee osteoarthritis (Bajaj et al., 2001). In fact, our results reveal the presence of both peripheral and central sensitization mechanisms in elite swimmers with and without shoulder pain.

Active TrPs in the neck and shoulder musculature in elite swimmers

In the current study, active TrPs were found in elite swimmers with shoulder pain in line with our hypothesis. Further, active TrPs in the infraspinatus and subscapularis muscles elicited a referred pain that mimicked the elite swimmer shoulder pain. When active TrPs were explored, swimmers spontaneously reported "Yes, this is exactly the pain that I usually feel spontaneously, or during training." Additionally, active TrPs in the upper trapezius, levator scapulae or scalene muscles were also found. Our findings support the view that active TrPs in the neck/shoulder muscles contribute directly to shoulder pain complaint in elite swimmers.

Active TrPs were not found as expected in elite athlete controls, since they did not suffer from pain in the shoulder region. In addition, we included elite athletes as controls to have subjects with similar levels of physical activity. Interestingly, elite swimmers without diagnosis of shoulder pain also exhibited active TrPs, but to a lower extent than swimmers with pain. When exploring active TrPs, elite swimmers without shoulder pain reported that the referred pain elicited by active muscles TrPs was similar to previous pain episodes. The presence of active TrPs may actually be related to the relative high prevalence of shoulder pain among elite swimmers (McMaster & Troup, 1993; Allegrucci et al., 1994; Bak, 1996).

Another relevant finding was the striking presence of a greater proportion of latent TrPs in elite swimmers with and without shoulder pain compared with controls. More important, swimmers without shoulder pain exhibited a greater number of latent TrPs than swimmers with shoulder pain. Lucas et al. (2004) found that latent TrPs disturb normal pattern of motor recruitment and movement efficiency suggesting the clinical relevance of latent TrPs. Moreover, the presence of latent TrPs in this population may be related to the lower PPT levels found in elite swimmers without shoulder pain. Thus, the presence of latent TrPs may be hence implicated in sensory-motor disturbances observed in individuals with shoulder pain (Rupp et al., 1995; Su et al., 2004; Olivier et al., 2008; Thomas et al., 2009). Latent TrPs may become active under the influence of several factors such as repetitive and sustained shoulder activities (Simons, 2004). Thus, our results underline the importance of inspection and inactivation of TrPs in the shoulder muscles among elite swimmers with shoulder pain. As such, TrPs may contribute to the overall picture of the pain. Our results are potentiated by a recent study suggesting that the presence of latent TrPs on the contralateral side could be the pathological basis of pain spreading observed in chronic myofascial pain syndromes (Ge et al., 2008). Further, the presence of latent TrPs in elite swimmer without shoulder pain may be related to previous pain episodes or to a possible predisposition for developing active and latent TrPs within the shoulder muscles in these athletes. At present, we could suggest that the inactivation of TrPs can prevent recurrence of symptoms, which are very common in this population (Mitchell et al., 2005).

Clinical applications for sport practice

The results of the current study have potential clinical applications for elite sport practice. First, elite swimmers may be evaluated and treated as a separate clinical entity, aimed toward underlying pathology and dysfunction of the neck–shoulder musculature. In fact, the presence of muscle TrPs implies the application of multimodal approaches targeted to the neck and shoulder muscles. For instance, manual therapies aimed at inactivating active TrPs, balanced strength training of the rotator cuff, improvement of core stability as well as correction of scapular dysfunction should be the main rehabilitation strategies. Maladapted swimming techniques and training are some of the main causes leading to shoulder pain in elite swimmers (Bak, 2010), underlining the importance of intervention targeting this issue. Finally, treatment of latent TrPs may be applied as a prevention strategy for reducing recurrences of shoulder pain in elite swimmers.

Limitations of the study

Finally, some methodological aspects of the current study should be mentioned. First, we included swimmers from four different countries, which increase external validity; however, they were recruited from the same place. In addition, the population size was small. Future studies with larger samples are needed to further confirm the current results. Moreover, we cannot establish a cause-and-effect relationship between PPT, muscle TrPs and shoulder pain, because the design was not longitudinal. Longitudinal studies are now needed to determine the role of mechanical sensitization and active TrPs in the development of shoulder pain in elite swimmers.

Perspectives

Our results suggest a role of active muscle TrPs in the development of shoulder pain in elite swimmers and the presence of sensitization mechanisms in swimmers. The presence of TrPs implies the application of multimodal approaches targeted to the neck and shoulder muscles in this elite sport population. Finally, the similar PPT levels found in elite swimmers with and without shoulder pain suggest that elite swimmers may be predisposed to develop a degree of mechanical sensitization related to the swimming-specific physical demands.

Key words: swimmers, shoulder pain, trigger points, pressure pain threshold.

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Nadadores de élite con dolor unilateral de hombro exhiben una mayor actividad muscular cervical durante un test functional de miembro superior.

(Artículo IV)

TITLE PAGE

TITLE

ELITE SWIMMERS WITH UNILATERAL SHOULDER PAIN EXHIBIT BILATERAL HIGHER CERVICAL MUSCLE ACTIVITY DURING A FUNCTIONAL UPPER LIMB TASK

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Abstract

It is suggested that muscle imbalance and motor control impairments can play a role in the development of shoulder pain in elite swimmers. Our aim was to investigate the differences in cervical muscle activation level between elite swimmers with and without shoulder pain during a low-load functional upper limb task. For that purpose, surface electromyography (SEMG) from sternocleidomastoid (SCM), upper trapezius (UT), and anterior scalene (SCL) muscles was recorded bilaterally in 17 elite swimmers (9 men, 8 women; age: 21±3 years) with unilateral shoulder pain, and 17 elite age- and gender- matched swimmers without pain. Root mean square was calculated to assess the level of activity before (5s) during (at 120s and 150s into the task), and after (10s post-task) an upper limb task. The repeated measure model revealed significant differences between groups for RMS of right SCL (F=3.146; P=0.041) and left SCL (F=3.465; P=0.040), but not for SCM (left: F=1.101, P=0.365; right: F=0.839, P=0.483) or UT (left: F=1.303, P=0.292; right: F=0.032, P=0.991) muscles. Swimmers with shoulder pain exhibited greater EMG amplitude of both SCL at 120s, 150s (P<0.001) and 10s post-task (P<0.05) as compared with those without pain. Our results showed that elite swimmers with shoulder pain exhibited greater activation of both SCL muscles during a repetitive low-load upper limb task and a decreased ability to relax the SCL muscles on completion of the task compared with elite swimmers without shoulder pain. These findings can have implications in relation to incidence and recurrence of shoulder pain in elite swimmers.

Key words: neck muscle, surface electromyography, activation pattern, shoulder pain

ELITE SWIMMERS WITH UNILATERAL SHOULDER PAIN EXHIBIT BILATERAL HIGHER CERVICAL MUSCLE ACTIVITY DURING A FUNCTIONAL UPPER LIMB TASK

INTRODUCTION

Shoulder injuries accounting with pain are very frequent in relation to physical activity in sports with overhead or repetitive arm movements such as swimming (Hume et al., 2006; Weldon &, Richardson 2001; Bak, 2010). Shoulder pain is one of the most common causes of physical disability in elite swimmers as inherent biomechanics to swimming promote muscular imbalances stressing the neck and shoulder complex (O'Donnell et al., 2005). The prevalence of shoulder pain in swimmers is higher than in the general population, ranging between 42% and 73% (McMaster & Troup, 1993; Allegrucci et al., 1994), and similar to volley ball players (Lo et al., 1990).

The etiology of shoulder pain in elite swimmers remains still unclear and several hypotheses have been proposed. A relative inappropriate stroke technique (Yanai & Hay, 2000) and training load among swimmers are thought to lead to shoulder pain (Soslowsky et al., 2000). In fact, shoulder pain and mechanical hypersensitivity are common after extensive training (Binderup et al., 2010). Additionally, there is evidence showing the role of shoulder muscles imbalance as a potential related factor associated to shoulder pain (Rupp et al., 1995; Olivier et a., 2008; Escamilla et al., 2009). Su et al (2004) reported a decrease in scapular upward rotation in swimmers with shoulder pain, whereas Wadsworth and Bullock-Saxton (1997) showed a significantly decreases in the timing of activation of serratus anterior, upper and lower trapezius muscles in swimmers with shoulder pain. On the contrary, Santos et al (2007) did not find differences in kinematics, latencies and recruitment order of shoulder muscles during shoulder elevation in the scapular plane among swimmers with shoulder impingement syndrome. Thomas et al (2009) found that elite swimmers reported decreases in internal rotation after their training seasons, which could be associated to an increased activity of the shoulder musculature. Additionally, a recent study has reported high prevalence of abnormal scapular kinesis during a normal training session in pain-free swimmers (Madsen et al., 2011).

These findings suggest that motor control impairments are most likely related to shoulder pain in elite swimmers. As the shoulder complex operates with finely tuned balance between the shoulder and the cervical spine, it is possible that impairments in motor control of the cervical muscles could be involved in the development of shoulder pain in elite swimmers. In fact, evidence of motor control impairments in the cervical musculature has been documented in patients with mechanical neck pain and whiplash associated neck pain during prescribed motor tasks (Fredin et al., 1997; Falla et al., 2004a). Some authors have investigated neck-shoulder muscles pattern activation in individuals with whiplash associated neck pain (Nederhand et al., 2000; 2002) or chronic neck pain (Madeleine et al, 1999; Falla et al, 2004b) during low-load, functional upper limb tasks. They reported that subjects with neck pain exhibited increased activity of sternocleidomastoid and upper trapezius muscles compared to healthy volunteers, underlining the presence of altered pattern of muscle activation in these pain conditions.

To the best of the authors' knowledge, no studies have previously investigated whether elite swimmers exhibit altered cervical muscle activation in the upper trapezius, sternocleidomastoid or scalene muscles during a low load functional task. Therefore, the aim of the current study was to analyse the differences in cervical muscle behaviour between elite swimmers shoulder pain and those without pain during a functional upper limb task. In this study, we hypothesized that elite swimmers with shoulder pain would exhibit increased muscle activity in the neck-shoulder region as compared with elite swimmers without shoulder pain.

METHODS

Participants

Elite swimmers (**Table 1**) from five competitive swimming clubs of different countries (Spain, Portugal, Lithuania, Denmark and Brazil) located at the High Altitude Sport Centre of Sierra Nevada (Granada, Spain) were screened for eligible criteria. To be included, swimmers should fulfil the following criteria: 1, aged between 18 and 30 years; 2, have training for at least 2.5 years under coach supervision; and 3, swimming 6 hours/week. In the current study, elite swimmers with and without unilateral shoulder pain were included. Within the shoulder pain group, swimmers should present unilateral shoulder complaints (described as pain felt in the neck-shoulder or arm) during at least 3 months of duration and with intensity > 4 points on an 11-point numerical pain rating scale. Additionally, age- and sex- matched elite swimmers without shoulder pain within the last year were included as control group.

Exclusion criteria for both groups were: 1, history of neck-shoulder surgery; 2, fracture of the shoulder area; 3, bilateral shoulder symptoms; 4, cervical radiculopathy; 5, previous interventions with steroid injections; or 6, any type of physical intervention in the neck-shoulder area within the previous year.

The study protocol was approved by the local Ethics Committee and conducted according to the Helsinki Declaration. All subjects signed an informed consent prior to their inclusion.

Surface Electromyography

SEMG signals were acquired bilaterally from upper trapezius (UT), sternal head of the sternocleidomastoid (SCM), and anterior scalene (SCL) muscles using adhesive Ag/AgCl surface electrodes (Ambu Inc®, Spain) following careful skin preparation and according to previous guidelines for electrode placement (Falla et al., 2002). The SEMG procedure was adapted from previous published studies (Nederhand et al., 2000; 2002; Falla et al., 2004a; 2004b). Ground reference was placed on the lateral epicondyle of the unaffected elbow. Signals were amplified, band-pass filtered [10–500 Hz], and sampled at 1000 Hz (12 bits A/D converter).

Procedure

Before the experimental trials, SEMG data were collected for 10s during standardized manoeuvres for normalization of the SEMG amplitude. To normalize SCM and SCL, a combined movement of cranio-cervical and cervical flexion in supine position for 10s was used. This movement consists of lifting the head, so that it just cleared the bed. This position is maintained isometrically. For the UT muscle, subjects performed 90° bilateral arm abduction sustained for 10s in a standing position. Each contraction was repeated 3 times with a 30s rest period between each repetition in line with previous studies (Nederhand et al., 2000; 2002; Falla et al., 2004a; 2004b).

For the experimental task, participants sat at a desk in a height adjustable office chair with their feet flat on the ground. Participants were asked to do pencil marks in 3 circles in an anticlockwise direction with their affected hand. This task was performed to the beat of a metronome set at 88 beats/minute. The other forearm rested motionless on the table. The 70 mm diameter circles were positioned in an equilateral triangle with a distance of 23 cm between each centre. The task was conducted for a total of 150s. SEMG signals were recorded in epochs of 5s prior to (baseline), 120s, 150s into performance of the task and 10- after the experimental task (post task).

Data Processing

To obtain a measure of the amplitude of the SEMG signal, the root mean square (RMS) values were calculated over 1-s epoch without overlapping for both the reference contractions and the experimental task using MegaWin 2.0 software (Mega Electronics, Kuopio, Finland). The maximum RMS value was extracted for each 10 or 5s window. For normalization, RMS values were expressed with respect to RMS values obtained during the reference voluntary contractions.

Statistical Analysis

The independent variables for this study were the two groups (between-groups factor), the experimental condition (4 measurements, within-groups factors), and the muscles examined (6 muscles, within-subject factor). The dependent variables were the normalized RMS values obtained for each muscle in elite swimmers with and without shoulder pain. As necessary, SEMG data were log-transformed to achieve homogeneity of variance. Each normalized RMS value was entered into a repeated measures general linear model to identify the overall differences between elite swimmers with and without shoulder pain. Means and 95% confidence intervals were calculated to analyse between group differences for each muscle in each group. Covariate analyses using the logged data were carried out for the factors of unilateral shoulder pain using a backward elimination technique in the repeated measures general linear model. Data were analyzed with the SPSS package version 16.0 (SPSS Inc, Chicago, IL).

RESULTS

Seventeen elite swimmers, 9 men and 8 women, aged 18 to 28 years old (age: 21 ± 3 years) with unilateral shoulder pain, and 17 elite swimmers, 9 men and 8 women, aged 18 to 26 years (age: 21±3 years) without shoulder pain participated (age, P=0.937). Among elite swimmers with shoulder pain, 11 (65%) exhibited pain before and during training, and also 6 after training. Demographic, training and swimming specialty are detailed in **Table 1**.

The mean duration of shoulder pain history was 2.9 years (95%CI 2.1 - 3.2), and the spontaneous level of shoulder pain at rest was 4.6 (95% CI 4.1 - 5.8). No correlation among shoulder pain intensity, training hours per week or training years was found.

The repeated measure general lineal model revealed significant main differences between both groups for SEMG amplitude of right SCL (F = 3.146; P=0.041) and left SCL (F = 3.465; P = 0.040), but not for both EMC (left: F = 1.101, P = 0.365; right: F = 0.839, P = 0.483) or UT (left: F = 1.303, P = 0.292; right: F = 0.032, P = 0.991) muscles **(Table 2).** Swimmers with shoulder pain exhibited significant greater EMG amplitude for both SCL at 120sec (right: P < 0.001; left: P < 0.001), 150 sec (right: P < 0.001; left: P < 0.001) and 10sec post-task (right: P = 0.004; left: P = 0.011) as compared to those without shoulder pain **(Fig 1).**

Finally, the inclusion of shoulder pain as covariate did not influence the SEMG amplitude for the affected (F=0.124; P=0.994) and the non-affected (F=1.807; P = 0.184) SCL muscles.

DISCUSSION

In accordance with our hypothesis, we found higher level of muscular activation in elite swimmers with shoulder pain compared with pain-free elite swimmers. The SCM muscles (both the affected and the unaffected side) showed higher SEMG activity during the functional task and 10s post-task. This finding underlines an altered pattern of activation of the superficial neck flexor muscles during functional motor tasks in elite swimmers with shoulder pain.

The existence of motor imbalance within the shoulder muscles in swimmers with shoulder pain has already been reported at several occasions as demonstrated by the altered kinematics of the shoulder complex (Rupp et al., 1995; Wadsworth & Bullock-Saxton, 1997; Su et al., 2004; Olivier et al, 2008; Escamilla et al., 2009). Ruwe et al (1994) have reported both an increase in the internal rotators and a decrease in the teres minor, supraspinatus, and the upper trapezius muscles activation level among swimmers with painful shoulders. The present study substantiates these findings and provides new key information on altered pattern of cervical muscle activation in elite swimmers with shoulder pain. Previous studies have also reported a delay in the timing of activation and increase level of activity of the UT muscle in swimmers or patients with shoulder pain (Ludewig & Cook, 2000; Thomas et al., 2009); however, we did not find differences in UT muscle activation during the functional task. This can be due to the low activation level of the upper trapezius muscle in the investigated functional task or to variance induced by the normalization procedure of the SEMG (Jackson et al., 2009).

The altered pattern of muscle activation was characterized by bilateral increased EMG activity for the SCL muscle throughout performance of the functional activity. In addition, elite swimmers with shoulder pain also demonstrated a decreased ability to relax the SCL muscles on completion of the task. Current results were similar to those previously found in chronic neck pain where patients also exhibited increased EMG activity of SCL muscles during and at completion of the same task (Falla et al., 2004b).

Undoubtedly, the increased activation level of the SCL muscles may alter the kinematics of the cervical-shoulder complex contributing to muscle imbalance (higher strengthening of the anterior neck muscles compared to the posterior muscles) (Becker, 1986) in elite swimmers with shoulder pain. Guth et al (1995) reported that swimmers exhibited greater cervical rotation (5º in average) on their breathing side as compared to non-swimmer. Considering that swimmers average 8000-12000 m/day, practice 2x/day, 5-7 days/week, cumulating on average 9900 strokes per week per shoulder, enormous demands are placed on the shoulder complex (Richardson, 1986). Our study provides evidence of possible relevance of SCL muscle over-activity in shoulder pain in elite swimmers as these muscles influence the neck, the shoulder and the rib regions.

Altered muscle activation patterns could be the physical manifestation of either pain pathways or may contribute/perpetuate pain. The neuro-physiological mechanisms of altered motor control are beyond the scope of this study and a few hypotheses specific to elite swimmers can be proposed. It has been suggested that increased muscle activation patterns represents an altered motor strategy of the central nervous system to minimize activation of painful muscles or compensate for inhibited muscles. In fact, pain influences motor control through complex mechanisms (Madeleine, 2010) and may explain the altered muscle activity of SCL muscles and changes in shoulder kinematics reported in elite swimmers (Rupp et al., 1995; Wadsworth & Bullock-Saxton, 1997; Su et al., 2004; Olivier et al, 2008; Escamilla et al., 2009). Additionally, higher EMG amplitude from superficial neck flexor muscles may compensate lower deep cervical flexors activation (Madsen et al., 2011). Future studies should investigate further muscle activation profiles to elucidate the role of motor control impairments in the cervical spine in elite swimmers.

The results of the current study have potential clinical applications for elite sport practice. Bilateral greater activation of superficial neck flexors such as the SCL muscles may result in excessive compressive loads on the cervical facet joints, a more superficial respiratory pattern, and impairment of the shoulder kinematics, subsequently promoting overload of these structures and the spreading of pain to the contra-lateral side via spinal mechanisms (Madeleine et al., 1999; Weldon &, Richardson 2001). Nevertheless, our cross-sectional study cannot answer whether the altered pattern of muscle activation identified precedes the onset of shoulder pain, or is the consequence of the pain. Undoubtedly, the presence of an altered pattern of muscle activation prior or after the onset of shoulder pain most likely constitutes a perpetuating factor. Our results suggest that elite swimmers should be evaluated and treated as a separate clinical entity. Maladapted swimming techniques and excessive training are the main cause of shoulder pain in swimmers (Bak, 2010). In fact, exercise protocols focusing on motor control training have been advocated for effective management of different pain disorders (Jull et al, 2002; Van Ettekoven & Lucas, 2006; Bak, 2010). A recent study has demonstrated that an 8-week exercise training program decreased forward head and rounded shoulder postures in elite swimmers (Lynch et al., 2010). Therefore, it would be interesting to investigate the use of motor control techniques targeted to the altered pattern of muscle activation identified in this study, and assess the incidence and recurrence of shoulder pain in elite swimmers.

Finally, some methodological aspects of the current study should be mentioned. First, we included swimmers from 4 different countries which increase external validity; however, they were recruited from the same sport elite place. The main limitation of the present cross-sectional design concerns the impossibility to establish a cause-and-effect relationship between increased activation of SCL muscles and shoulder pain. Longitudinal studies are needed to further determine the role of motor control impairment of the neck and shoulder muscles in the development of shoulder pain in elite swimmers.

CONCLUSIONS

Elite swimmers with shoulder pain exhibited greater EMG activation of anterior scalene muscles during a repetitive upper limb functional task as compared to elite swimmers without shoulder pain. Our finding supports the evidence of altered patterns of superficial cervical flexor musculature activation during a low-load functional motor task of the upper limb in elite swimmers with shoulder pain. These findings provide new key information about muscle action and shoulder pain with potential implications about the incidence and the recurrence of symptoms in elite swimmers.

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Conflict of interest: None conflict of interest are declared.

FIGURE LEGEND

Figure 1: Normalized RMS values for the bilateral, upper trapezius (UT), sternocleidomastoid (SCM) and scalene (SCL) muscles in elite swimmers with and without shoulder pain.

* Statistical significant differences between swimmers with and without pain $P < 0.05$

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Table 1: Descriptive statistics mean (SD) for anthropometric, swimming style and training data of the participants

	Healthy Swimmers (n=17)	Shoulder Pain Swimmers (n=17)	P
Weight (Kg)	69.8 (9.1)	70.3 (8.2)	0.482
Height (cm)	178.9 (6.2)	179.6 (5.6)	0.661
BMI ($Kg*m^{-2}$)	21.8 (4.3)	22.2 (3.5)	0.940
Swimmer style (%)			
Breast stroke	2 (12%)	2 (12%)	
Freestyle	10 (59%)	10 (59%)	0.916
Butterfly	2 (12%)	3 (17%)	
Back stroke	3 (17%)	2 (12%)	
Begin training (years)	10.9 (3.7)	10.6 (3.4)	0.448
Training hours (hours/week)	24.3 (3.5)	24.8 (3.8)	0.731

Elite Swimmers with shoulder pain (n = 17)						
	Scalene		Sternocleidomastoid		Upper Trapezius	
Normalized EMG	Affected side	Non-affected side	Affected side	Non-affected side	Affected side	Non affected side
Baseline	0.27 ± 0.14 (0.14-0.39)	0.14 ± 0.07 (0.10-0.18)	0.05 ± 0.02 (0.03-0.06)	0.04 ± 0.02 (0.03-0.05)	0.19 ± 0.15 (0.08-0.28)	0.08 ± 0.06 (0.03-0.12)
120 s	0.70 ± 0.41* (0.49-0.93)	0.25 ± 0.14* (0.18-.0.33)	0.14 ± 0.07 (0.10-0.17)	0.08 ± 0.04 (0.06-0.11)	0.68 ± 0.36 (0.49-0.85)	0.21 ± 0.18 (0.11-0.31)
150 s	0.72 ± 0.42* (0.47-0.92)	0.27 ± 0.16* (0.18-0.35)	0.14 ± 0.07 (0.10-0.17)	0.09 ± 0.04 (0.07-0.11)	0.67 ± 0.46 (0.43-0.90)	0.21 ± 0.18 (0.11-0.47)
10 s post-task	0.42 ± 0.22* (0.30-0.54)	0.20 ± 0.14* (0.12-0.27)	0.11 ± 0.06 (0.07 ± 0.14)	0.06 ± 0.02 (0.05-0.08)	0.28 ±0.18 (0.20-0.38)	0.15 ± 0.11 (0.07-0.24)
Elite Swimmers without shoulder pain (n = 17)						
Normalized EMG	Right side	Left side	Right side	Left side	Right side	Left side
Baseline	0.19 ± 0.07 (0.10-0.18)	0.16 ± 0.10 (0.11-0.21)	0.07 ± 0.04 (0.03-0.10)	0.05 ± 0.02 (0.03-0.05)	0.20 ± 0.13 (0.12-0.26)	0.07 ± 0.06 (0.04-012)
120 s	0.34 ± 0.15 (0.25-0.42)	0.19 ± 0.09 (0.15-0.24)	0.12 ± 0.09 (0.07-0.17)	0.07 ± 0.04 (0.05-0.09)	0.65 ± 0.38 (0.47-0.75)	0.16 ± 0.14 (0.09-0.23)
150 s	0.33 ± 0.17 (0.25-0.44)	0.18 ± 0.09 (0.14-0.23)	0.13 ± 0.08 (0.08-0.17)	0.07 ± 0.04 (0.05-0.09)	0.64 ± 0.34 (0.48-0.81)	0.14 ± 0.10 (0.09-0.19)
10 s post-task	0.22 ± 0.12 (0.16-0.29)	0.19 ± 0.11 (0.13-0.24)	0.10 ±0.07 (0.06-0.15)	0.06 ± 0.03 (0.04-0.07)	0.26 ± 0.20 (0.15-0.32)	0.16 ±0.12 (0.06-0.22)

Table 2: Normalized RMS values for upper trapezius, sternocleidomastoid and scalene muscles in elite swimmers with and without shoulder pain. * Statistical significant differences between groups ($P < 0.05$)

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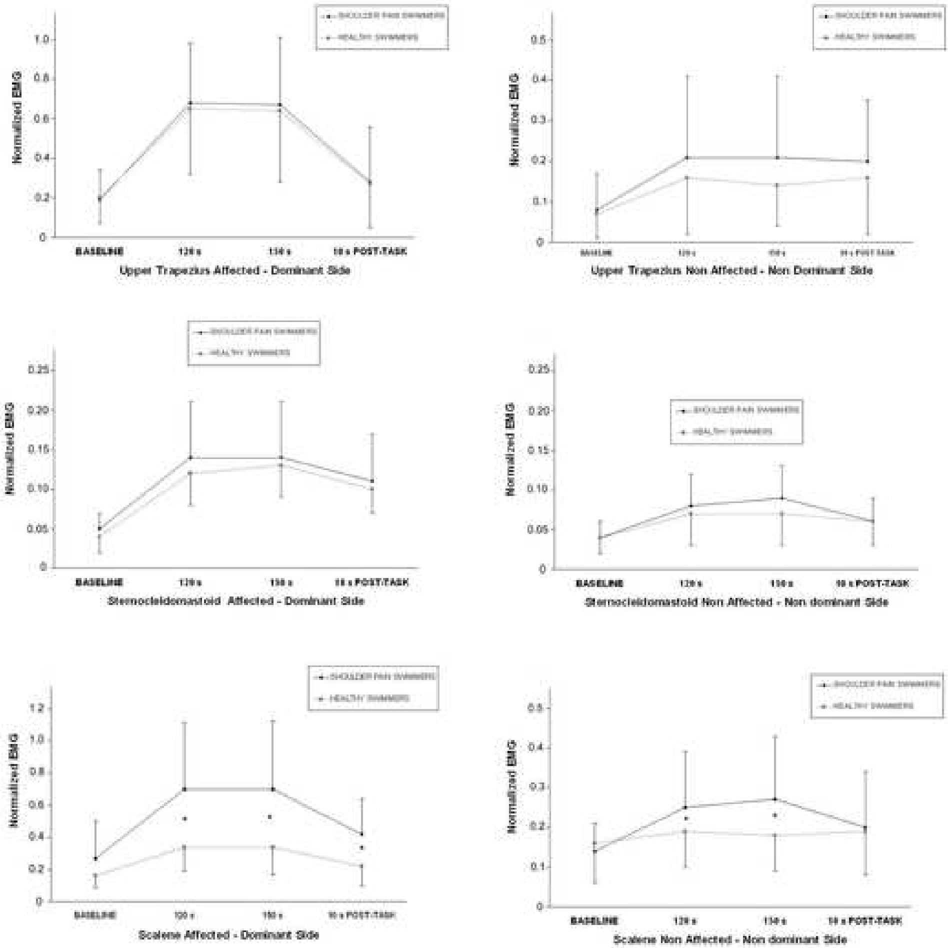

CONCLUSIONES

I. Los nadadores de élite con y sin síndrome subacromial exhiben valores más bajos de umbral doloroso a la presión, comparados con los controles; por lo que pueden tener predisposición a desarrollar un grado de sensibilización mecánica relacionada con las demandas físicas específicas de la natación.

II. Los puntos gatillo activos en los músculos del hombro fueron encontrados en nadadores de élite con síndrome subacromial unilateral y los puntos gatillo latentes en nadadores de élite sin síndrome subacromial.

III. Los puntos gatillo activos fueron relacionados con el umbral más bajo de dolor a la presión.

IV. Existe una mayor proporción de puntos gatillo latentes en los nadadores de élite con y sin síndrome subacromial comparados con los controles.

V. La exploración manual de los puntos gatillo activos de la musculatura, provocó tanto dolor local como dolor referido, reproduciendo el patrón de dolor de todos los pacientes.

VI. En nadadores de élite, con y sin síndrome subacromial, existen mecanismos de sensibilización central y periférica. Los pacientes con síndrome subacromial mostraron hiperalgesia a la presión tanto en áreas dolorosas como no dolorosas.

VII. Los puntos gatillo modifican el patrón normal de reclutamiento motor y de la eficiencia de movimiento sugiriendo la relevancia clínica de los puntos gatillo latentes.

VIII. Un mayor número de puntos gatillo y niveles de umbral doloroso a la presión más bajos fueron relacionados con una mayor intensidad del dolor.

IX. Los nadadores de élite con dolor de hombro exhibieron una mayor activación de los músculos escalenos anteriores durante un test funcional repetitivo del miembro superior, en comparación con los nadadores de élite sin dolor de hombro.

X. El tratamiento manual de los puntos gatillo puede descender la hipersensibilidad del dolor a la presión; tiene un efecto generalizado antinociceptivo.

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Salud.

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