No benefit of arthroscopy in subacromial shoulder pain

Since its introduction nearly 100 years ago,1 arthroscopy of the knee has revolutionised the care of patients with meniscal lesions, ruptures of the anterior cruciate ligament, and cartilage damage. Although knee arthroscopy has proved to be an asset of patients with meniscal lesions, ruptures of the anterior cruciate ligament and a bursectomy. An arthroscopic version had no effect, and this finding was confirmed in another study.2 Knee arthroscopy of the knee has revolutionised the care of osteoarthritis: in a trial using a sham surgery,2 knee arthroscopy in osteoarthritic knees.4 No benefit of arthroscopy in subacromial shoulder pain

To overcome this impingement problem, in 1972 Neer5 proposed open decompression of this area, removing osseous spurs from the caudal side of the acromion, often combined with release of the coracoacromial ligament and a bursectomy. An arthroscopic version of this popular procedure was developed and is now frequently undertaken, with an increase of nearly ten times in England from 2500 surgeries in 2000 to 21 000 in 2010.6

However, as correctly pointed out in The Lancet by David Beard and colleagues7 for the Can Shoulder Arthroscopy Work (CSAW) study group, the scientific evidence-based surgery in daily surgical practice is clearly defined. Two areas that are possibly indicated are the glenohumeral joint for intra-articular lesions and the subacromial area that is extra-articular. An impingement syndrome of the subacromial area is responsible for up to 70% of all shoulder problems.7 This impingement arises due to the restricted space between the acromion and the glenohumeral head, where rotator cuff tendons slice back and forth in abduction and ante version, facilitated by a subacromial bursa. An impingement of these rotator cuff tendons occurs frequently in patients older than 40 years, with typical complaints such as a painful arc.8

References:


clinical evidence in favour of this type of shoulder surgery is scarce. In Beard and colleagues’ multicentre, pragmatic, parallel group, placebo-controlled, three-group, randomised surgical trial, 106 patients were assigned to decompression surgery, 103 to arthroscopy only, and 104 to no treatment. The primary outcome was the Oxford Shoulder Score at 6 months, analysed by intention to treat.3

The study group should be commended for undertaking this difficult trial. Two factors that made an assessment of effect difficult were the rather high level of non-compliance in the groups (24 [23%], 43 [42%], and 12 [12%] of the decompression, arthroscopy only, and no-treatment groups, respectively, did not receive their assigned treatment, making the groups more similar and thus differences harder to detect) and the long waiting list, with a waiting time of up to 4 months considered to be acceptable. Thus, patients who were only 2 months post surgery were compared with patients who were 6 months into the no-treatment option. However, the outcome was still remarkable. There was no difference in the primary outcome between the arthroscopic decompression and arthroscopy only groups (decompression mean 32·7 points [SD 11·6] vs arthroscopy mean 34·2 points [9·2]; mean difference –1·3 points [95% CI –3·9 to 1·3], p=0·3141). This is an intriguing finding, showing that subacromial decompression does not affect the clinical outcome.

Additionally, although patients in the surgical groups had statistically better outcomes than the no-treatment group, the differences were small and not clinically important. Although the sizes of the differences are difficult to interpret because of the high non-compliance levels and long waiting list, the differences themselves might be attributed to the placebo effect after surgery. Another explanation, but also a criticism of the study design, is that the no-treatment group was left alone (one reassessment appointment with a specialist shoulder clinician 3 months after study entry but no intervention) in this study with no offered rehabilitation programme, unlike the surgical groups.

The findings send a strong message that the burden of proof now rests on those who wish to defend the standpoint that shoulder arthroscopy is more effective than non-surgical interventions. Hopefully, these findings from a well respected shoulder research group will change daily practice. The costs of surgery are high, and although the low occurrence of complications might suggest that the surgery is benign,10 there is no indication for surgery without possible gain. Therefore, the focus on the cure for these patients should be on developing effective conservative treatment programmes based on exercise and probably combined with tape, manual therapy, extracorporeal shockwave lithotripsy, or laser treatment.11

The emerging pattern that arthroscopic interventions might, for some indications, not be more beneficial than non-surgical options should also be taken into consideration when the decision for hip arthroscopy is made. There is a worrisome trend to do arthroscopy of the hip, including in elderly patients with degenerative hips.12 The orthopaedic field is in urgent need of well designed studies that assess the effectiveness of this kind of surgery in osteoarthritic hips.13

Arthroscopy is a useful procedure that benefits many patients. However, there are serious concerns that a substantial number of these procedures are done in patients with mostly degenerative diseases, and therefore will not be curative. Unfortunately, it will be demanding to change daily orthopaedic practice as both patients and surgeons believe sincerely that the problems will be reduced after arthroscopic surgery. Evidence such as that reported by the CSAW study group should help to change such views and improve practice.

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Walking to a pathway for cardiovascular effects of air pollution

There is a well documented association between human exposure to fine particulate matter air pollution (PM$_{2.5}$) and an increased risk of cardiovascular disease and death. 1,2 Indeed, the Global Burden of Disease (GBD) study 1 recently estimated that exposure to PM$_{2.5}$ contributed to 4.2 million deaths in 2015, representing the fifth-ranked risk factor for global deaths; of these, mortality from cardiovascular disease (CVD; ie, ischaemic heart disease and cerebrovascular disease) accounted for most deaths attributed to ambient PM$_{2.5}$ air pollution. However, despite these strong epidemiological associations and the documented widespread adverse health effects, the exact biological mechanisms and the types of particles that are most responsible for the PM$_{2.5}$–CVD associations are not well defined.

In The Lancet, Rudy Sinharay and colleagues 4 use a simple but elegant randomised crossover design to gain insight into the type of pollution that can lead to the air pollution–CVD associations that have been reported in population-based epidemiological studies, as well as to identify specific cardiovascular changes consistent with the causality of those associations. The researchers studied the effects of traffic pollution exposure in adult participants aged 60 years and older during a 2 h walk along a busy commercial street in London, England (Oxford Street) compared with a similar walk in a nearby London park (Hyde Park), which has much lower air pollution. 40 healthy volunteers, 40 participants with chronic obstructive pulmonary disease, and 39 participants with ischaemic heart disease took part. In all 119 participants, irrespective of disease status, walking in Hyde Park led to an increase in lung function and a decrease in arterial stiffness, measured as pulse wave velocity and augmentation index, following the walk. By contrast, these beneficial responses were significantly diminished after walking along the more polluted Oxford Street. Specifically, among healthy volunteers the investigators reported a roughly 5% (95% CI –10·40 to –0·27) decrease in pulse wave velocity from 2 to 26 h after the Hyde Park walk, an exercise benefit that was not only negated but even reversed 26 h after the Oxford Street walk (7% increase in pulse wave velocity, 95% CI 2·16 to 12·20). Thus, the multifactorial benefits of low-to-moderate intensity physical activity, such as walking, for the primary and secondary prevention of CVD 5 were offset by the presence of air pollution. Reductions in measures of arterial stiffness have been recorded with the use of guideline-directed medical therapy,6 however, until this study, evidence has been scarce on the adverse effects of air pollution exposure on vascular function during physical activity.7

Important to the interpretation of this study is the finding that air pollution causes phospholipid oxidation 8 and oxidative stress (eg, by transition metals in fossil fuel combustion particles). 9 These pathways accelerate atherogenesis and increase arterial stiffness, itself a strong predictor of cardiovascular events and all-cause mortality. 10 However, one limitation of such panel studies is their size; as such, generalisability can be an issue. In view of this limitation, more and larger practical real-world exposure studies like the one done by Sinharay and colleagues 4 that also assess novel in-vivo biomarkers of oxidative stress and phospholipid oxidation might further clarify the mechanistic pathways and clinical implications of air pollution exposure, and broaden their known applicability. Furthermore, additional evidence on the temporal relationships and longer-term cumulative effects of chronic air pollution on arterial stiffness is also needed. Overall, however, data from Sinharay and colleagues provide significant new evidence of an