EXTENDED REPORT

Idiopathic osteoarthritis and contracture: causal implications

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Objective: To use the known association of idiopathic osteoarthritis with contracture as a means of searching for its cause. There are currently two theories concerning this association, one assuming that the contracture is a consequence of the osteoarthritis and the other that it precedes and causes the osteoarthritis. This study tested both theories.

Methods: Flexion ranges in the 12 finger joints were obtained by goniometric measurement in two samples of normal female subjects, one group with a mean age of 22 years (25 subjects) and one with a mean age of 45 years (50 subjects). The results were compared with the known regional prevalence of osteoarthritis in the finger joints of women.

Results: The older group showed evidence of reduced flexion range consistent with development of contracture in the extensor mechanism of the fingers. The distribution of the contracture showed a strong negative correlation with the regional prevalence of osteoarthritis.

Conclusions: An early dorsal contracture develops in the fingers of normal subjects, but it is neither a consequence of nor the cause of digital osteoarthritis. The most parsimonious explanation for the association is that both contracture and idiopathic osteoarthritis are independent consequences of failure to use the full movement range. If this hypothesis is correct, the disease could be preventable.

It is a necessary prediction from the unused arc theory of idiopathic osteoarthritis^{1–3} that the disease should be accompanied or preceded by an independent contracture of those muscles and tendons that have been habitually underextended. While contracture—defined as a persisting loss of movement range resulting from structural change⁴—has long been recognised as one of the features regularly associated with osteoarthritis,^{5–10} it has not normally been regarded as independent. Conventional theory assumes that it is a complication of the osteoarthritis resulting from osteophytes or from synovial deposition of irritant cartilaginous debris.^{6–8–11} Recently Smythe, investigating finger osteoarthritis, has reported that the contracture occurs early, preceding the osteoarthritis, and has raised the contrary suggestion that it may play a causal role in its development.^{12–13} Our present investigation tests both theories.

The objective of the study was to determine if there is any evidence of contracture development in the fingers before the onset of osteoarthritis, and if such contractures are identified, to determine whether the distribution of the contractures does or does not correlate with the known distribution of osteoarthritis in the hands.

METHODS

The age of onset of contracture was investigated by a cross sectional study of passive flexion range in the 12 finger joints in two samples of normal subjects taken from two different age groups. The mean ages were 22 years (range 18 to 28) in the first group and 45 years (35 to 55) in the second group. To maximise relevance and reduce confounding by secondary osteoarthritis the investigation was confined to women.

All subjects were clinically examined, and those accepted for the study were free from finger deformity, joint swelling or tenderness, or other evidence of digital osteoarthritis. In each subject the passive flexion range of the finger joints was measured in both hands using the zero axis technique recommended by the American Academy of Orthopaedic Surgeons.¹⁴ To avoid interobserver error all measurements were made by the same metrologist (NL), using a standard digital goniometer. Precision was determined by repeated

blind measurements of the same subject on a distal interphalangeal (DIP), a proximal interphalangeal (PIP), and a metacarpophalangeal (MCP) joint of a single subject, and was calculated for each joint as the standard deviation $\times 100$ divided by the mean. Mean flexion angles were calculated for each joint in each hand in both age groups. Absolute contractures were calculated from the difference in mean movement range in the two age groups, and relative contracture expressed the absolute contractures as a percentage of the normal range in the younger group. The distribution of both contractures was compared with the distribution of digital osteoarthritis, derived from the data of Acheson *et al.*¹⁵

Statistical methods

To investigate the relation of contracture with age and to determine if the relation differed for different fingers and joints, the passive flexion movement ranges were analysed using a mixed linear model. This allowed for the correlation structure of the repeat measurements from each individual, namely from the two hands each with four fingers and from the three different joints in each finger. The three way interaction of age group with joint and finger was investigated to see if the difference in the effect of age on flexion movement was the same in all fingers. This was followed by an investigation of the joint by age group interaction to investigate whether the effect of age on flexion could be shown to be different in the different joints. To investigate the correlation of the distribution of contracture with that of osteoarthritis, Spearman's correlation was calculated between the observed contractures and the regional prevalence of osteoarthritis. The results are shown in table 1.

RESULTS

The difference in the effect of age on amount of movement in different joints could not be shown to differ in different fingers ($p = 0.23$). This three way interaction was therefore

Abbreviations: DIP, distal interphalangeal joint; MCP, metacarpophalangeal joint; PIP, proximal interphalangeal joint

Table 1

Least square means of passive flexion movement ranges in normal subjects

Joint	Osteoarthritis prevalence* (%)	Digit	Sample A (n = 50 hands, mean age 22 years) Angle (SE)	Sample B (n = 100 hands, mean age 45 years) Angle (SE)	Contracture	
					Absolute	Relative
DIP	49	Index	77.5 (1.5)	76.2 (1.1)	1.3	1.7
	43	Middle	78.8 (1.5)	79.7 (1.1)	-0.9	1.1
	36	Little	80.7 (1.5)	81.5 (1.1)	-0.8	1.0
	32	Ring	77.1 (1.5)	76.6 (1.0)	0.5	0.6
PIP	21	Middle	107.0 (1.1)	105.0 (0.8)	2.0	1.9
	17	Index	104.9 (1.2)	103.9 (0.8)	1.0	0.9
	15	Ring	108.8 (1.1)	105.3 (0.8)	3.5	3.2
	14	Little	106.7 (1.2)	103.4 (0.8)	3.3	3.1
MCP	13	Index	92.5 (1.1)	89.4 (0.8)	3.1	3.4
	10	Middle	94.9 (1.1)	91.1 (0.8)	3.8	4.0
	4	Ring	96.6 (1.1)	92.2 (0.8)	4.4	4.6
	3	Little	96.3 (1.1)	93.8 (0.8)	2.5	2.6

Values shown are mean ranges of movement in degrees.

*Joints ranked in order of prevalence of osteoarthritis in females from the data of Acheson *et al.*¹⁵

DIP, distal interphalangeal joint; MCP, metacarpo-phalangeal joint; PIP, proximal interphalangeal joint.

removed from the analysis. No difference in the effect of age group on flexion movement in the three joints could be demonstrated ($p = 0.2$). However, there was evidence of an overall decrease in flexion movement in the older age group compared with the younger ($p = 0.02$). The least square means and standard errors for each finger and joint are given in table 1.

There was a strong negative correlation between the expected prevalence of osteoarthritis in a joint and the degree of contracture ($r = -0.8$, $p = 0.005$). Recalculation using only one hand from each subject did not affect the observed difference in ranking between osteoarthritis prevalence and contracture. The precision for this metrologist was 3% in the DIP joint, 2.1% at the PIP joint, and 1.7% in the MCP joint

DISCUSSION

The age related contracture found in these normal joints is comparable with that found in the spine and in other joints in previous studies.¹⁶⁻¹⁹ The data confirm Smythe's¹² contention that a contracture develops in the extensor mechanism of the fingers before there is any overt evidence of osteoarthritis. This does not rule out the possibility that a second capsular contracture could develop later as a complication once osteoarthritis is established, but this early contracture cannot reasonably be explained on this basis. However, the strong negative correlation between the distribution of this contracture and the regional prevalence of osteoarthritis makes it equally unlikely that it plays a causal role in its development. It appears that the two conditions are independent.

While no direct conclusions concerning cause can be drawn from a cross sectional study, the results have nonetheless some indirect implications. If two conditions are regularly associated and neither causes the other, it is likely—although not inevitable—that they arise from the same cause. Finding the cause of this early contracture could thus provide a potential lead to the cause of idiopathic osteoarthritis.

The most obvious candidate for this common cause, age, is not satisfactory in either case. Age relatedness is a necessary concomitant of any disease that develops slowly over a long period of time, but the flexor tendons in these subjects were the same age as the extensors and showed no evidence of contracture. Similarly, in the case of idiopathic osteoarthritis, the demonstration that many joints remain normal into advanced old age²⁰⁻²³ has led to a consensus that age can be removed from the list of possible causes.²⁴⁻²⁷ It remains then

to find some other cause for contracture which can be tested for a possible role in the development of idiopathic osteoarthritis.

The only biological mechanism for shortening of collagen and development of contracture so far identified is habitual underextension. It is now well established that contracture will develop in any condition in which the design range of a tension structure is not used, apparently as a negative feedback response to reduction in peak tension.²⁸ It occurs in a range of clinical conditions that have the common denominator of restricted movement²⁹⁻³⁵ and is readily produced in experimental animals by partial immobilisation.³⁶⁻⁴⁰ The mechanism has been identified as a slow structural change in the collagen fibrils, initially reversible but eventually locked in by development of new cross links.^{28, 35} It follows then that contracture in the finger extensors most probably reflects an appropriate biological response to a habitual failure to reach the peak tension levels operating when the fingers are fully flexed. As the only factor involved in extending these elements is contraction of the opposing flexors, development of extensor contracture is an indication of habitual underuse of interphalangeal flexion, an underuse that has already been observed in human behavioural studies of finger movement.⁴¹ Underuse of movement range thus emerges as a contender for a causal role in the two independent variables, contracture of the extensor mechanisms and idiopathic osteoarthritis.³

This underuse hypothesis was in fact proposed 50 years ago by Harrison *et al*¹ and later by Bullough and Goodfellow.² It received further support from behavioural studies on hip osteoarthritis,⁴² but it has not in general been regarded as a serious contender for the role of cause, perhaps in the main from the lack of an obvious mechanism. Recent studies on joint physiology may remove this obstacle. It has been shown that the dominant factor controlling synovial clearance—intra-articular pressure⁴³—reaches maximum value only at the extremes of the movement range.⁴⁴ Accumulation of normal enzymes and growth factors as a result of synovial stasis could explain both the catabolic and the unexplained anabolic components of the osteoarthritic process. The demonstration by Langenskiöld and others that partial immobilisation of animal joints leads not only to contracture but also to osteoarthritis^{37, 45} suggests that the hypothesis is worth exploring. If it is correct, idiopathic osteoarthritis could be preventable, and possibly, in its early stages, reversible.

In summary, the study confirms that an independent contracture does develop with age in the extensor

mechanisms of the fingers in normal subjects. This is indicative of a longstanding underuse of the flexor component of the movement arc, and provides supporting evidence for the unused arc hypothesis in the causation of osteoarthritis.

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