Curr Neurol Neurosci Rep (2012) 12:386–391
DOI 10.1007/s11910-012-0280-9

MOVEMENT DISORDERS (SA FACTOR, SECTION EDITOR)

Anterocollis and Camptocormia in Parkinsonism: A Current Assessment

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Published online: 26 May 2012

Abstract Postural deformities in parkinsonian syndromes are well recognized, but poorly understood and largely refractory to available therapies. In recent times a number of hypotheses have been proposed to explain the underlying etiology of anterocollis and camptocormia, but currently there is no consensus. As these conditions are more precisely characterized we begin to uncover that this is a heterogeneous and evolving phenomenon. These conditions bring to light the inadequacies of our current tools to study biomechanics of posture, neuromuscular disorders, and dystonic muscular contractions. The development of objective, accurate tools to directly study and measure the severity of these postural disorders will allow for further understanding of the pathophysiology, the development of novel therapeutics, and adequate clinical trial design.

Keywords Anterocollis · Head drop · Dystonia · Myopathy · Camptocormia · Parkinsonism

Introduction

Postural changes have been part of the clinical description of Parkinson's disease (PD) since its original characterization by James Parkinson in 1817. In his essay he described both anterocollis as "the chin is now almost immovably bent down upon the sternum" and truncal deformities in the context of festination: "a propensity to bend the trunk forward." Major advances have been made in our understanding of PD in general, and these have been translated into effective symptomatic therapies; however, postural deformities have remained largely refractory to these available therapies. Improved understanding of the mechanisms underlying these postural phenomena and better characterization of this heterogeneous group of disorders is necessary for effective treatment strategies to emerge. This review presents the current state of knowledge regarding the clinical presentation, underlying etiology, and treatment of anterocollis and camptocormia associated with parkinsonism.

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Anterocollis

Anterocollis was reported in parkinsonian syndromes sporadically throughout the 20th century, particularly in cases of multiple system atrophy (MSA) [1–3]. As early as 1926 reports of "severe antecollis" in suspected cases of olivopontocerebellar atrophy were reported. In 1989 the term "disproportionate antecollis" was used to imply that antecollis was out of proportion to truncal postural changes and this was a sign suggestive of MSA [4]. Anterocollis has since been reported in multiple series of idiopathic PD patients [5], but remains more common in MSA. Although no formal epidemiological studies have been performed to establish prevalence, reported rates range from 5 % to 6 % in PD [5–7] and as high as 42 % in MSA [6].

In later years, the "dropped head syndrome," in neuromuscular diseases and in isolation, was characterized, and the term "isolated neck extensor myopathy" (INEM) was born [8, 9]. Following these reports investigators were able to document myopathic changes in patients with parkinsonism and anterocollis, concluding the underlying etiology of this condition was myopathy of the cervical extensors [10, 11]. Since then alternative hypotheses arose implicating

unbalanced tone, rigidity, and dystonia [7, 12, 13] as the primary culprits. More recently, a definition for anterocollis has been put forth describing the phenomenon as marked flexion of the neck (>45°), out of proportion to truncal flexion, and which may be overcome voluntarily (in early stages). Under this definition, patients are unable to fully extend the head against gravity when sitting or standing, but are able to exert force against the examiner's hand [6, 14•]. Furthermore, our group and others have begun to characterize this condition by identifying clinical subtypes of anterocollis, acknowledging the heterogeneity in presentation and etiology of this condition [15, 16•]. These distinctions will be crucial in future clinical trial design.

Camptocormia

The term camptocormia, literally translated from Greek to mean bent trunk or bent spine, was originally described in 1818 as an orthopedic deformity [6]. In the early 20th century the term resurfaced to describe a psychogenic disorder observed in soldiers following the first World War [14•]. It was not until 1999 that the term was first used to describe this posture in patients with PD [17]. Today camptocormia is defined as marked flexion of the trunk (>45°) in the sagittal plane, which originates in the thoracolumbar spine and resolves (almost completely) upon laying supine [6, 17]. Reported prevalence rates for camptocormia range from 3 % to 17 % in PD. More recently studies have focused on the hypothesis of myopathy of the paraspinal muscles, performing detailed electromyographic studies, biopsies, and imaging of the musculature to gather evidence of myopathy [18–20]. No consensus has been reached thus far, in either condition, regarding underlying etiology, or treatment, but multiple clinical descriptions, and case reports describing successful therapies have been reported [21•].

Clinical Presentation

Anterocollis

When anterocollis is seen in parkinsonian patients it can present subacutely or insidiously. There have been reports of onset following the administration of dopamine receptor blocking agents [22] as well as dopamine agonists [7, 23–25]. Patients complain of pain (usually early) in the cervical extensor or suboccipital muscles, but pain can also take on a radicular pattern [26]. The condition usually progresses and becomes pronounced with the chin approximating the chest in most cases. There is flexion of the neck accompanied by a sagittal shift in most. Some patients also exhibit various degrees of laterocollis and torticollis [15, 26]. Pseudohypertrophy of the extensor musculature is common and thought to be related to edema. Some patients exhibit atrophy of the superficial flexors (sternocleidomastoids [SCMs]), while others show hypertrophy and spasm of these muscles. Weakness of the extensors is not universal, but when it is present, these patients are more likely to have myopathy of the extensor musculature [15]. Range of motion is not typically limited early, but usually does diminish as the condition progresses and as the duration of anterocollis increases [15].

Certainly not all anterocollis patients fit into one clinical description. It is important to note not only that this condition is heterogeneous, but that it evolves in some patients with time. A parkinsonian patient may present with cervical flexor spasm and hypertrophy resulting in moderate anterocollis with a saggital shift, and mild torticollis, but no rigidity and a full range of motion. This presentation suggests a dystonic etiology. However, the same patient may present 2 years later, with or without extensor pseudohypertrophy, less marked flexor hypertrophy, and some extensor weakness, which may prompt the clinician to consider an extensor myopathy. Finally, the same patient may present 5 years later, now with a clear diagnosis of MSA, with a fixed chin to chest posture. At this point, orthopedic etiologies may be considered. This is merely a hypothetical example, and not all patients progress this way, but it is important to keep in mind that clinical findings may evolve over time.

Extensor weakness is the cardinal feature of the myopathic subtype of anterocollis. If present this should warrant neuromuscular evaluation with electromyography (EMG) and possible biopsy [15]. This subgroup tends not to have flexor hypertrophy or spasm, but may have atrophy of this group, with extensor pseudohypertrophy and no significant reduction in cervical range of motion, at least early [15]. The dystonic subtype tends to have clear hypertrophy and spasm of the superficial flexors, may have superimposed laterocollis or torticollis [26], and extensor pseudohypertrophy [16•]. The levator scapulae is often observed to be in spasm, and this is thought to be compensatory, but often a source of pain.

As a consequence of anterocollis patients experience a number of non-motor features including dysphagia, sialorrhea, and pain [14•, 15, 26]. An important consequence is worsening balance attributed to the change in center of gravity [14•]. In addition, when some patients lay supine their posture normalizes, while others remain with a fixed posture even when laying supine; the so-called "psychic pillow" [15].

Camptocormia

Camptocormia has also been described as presenting subacutely or insidiously over days or months. There are reports

of back pain preceding the abnormal posture, and it is a common problem throughout the course of the disease. There have been reports of camptocormia occurring following surgical interventions, and this has been suggested as a possible risk factor [17, 27]. Patients with camptocormia (as opposed to patients with orthopedic truncal deformities) often complain of postural deterioration when they walk [14•]. Some patients do report tightening of their abdominal musculature as well as a subjective feeling of being pulled down [28]. Most patients, particularly early in the disease process, can overcome the abnormal posture by standing next to a wall, or laying supine, which is another important distinguishing feature [14•]. The occurrence of camptocormia correlates with PD severity, and longer disease duration [14•]. On examination, axial or truncal rigidity is often present, palpation of the paraspinals reveals hardening, and the rectus abdomini are often in spasm [14•, 27]. Superimposed lateral flexion deformities or Pisa syndrome can also be found [14•]. Truncal extensor weakness has not been reported. Non-motor features of camptocormia include fatigue and back pain [17, 21•, 29]. Balance is also affected.

Competing Hypotheses

Potential etiologies of anterocollis and camptocormia have been suggested and are addressed here. They include dystonia, myopathy, and disproportionate rigidity. It is possible that all three contribute and that these disorders are heterogeneous in nature.

Dystonia

Clinical findings reminiscent of cervical and/or truncal dystonia have led some clinicians to suspect a dystonic etiology. These include subjective sensation of being pulled down, reports of a geste antagoniste [7], examination findings of cervical flexor (SCM) spasm and hypertrophy, superimposed laterocollis and torticollis [15, 26], reports of increased spontaneous activity on EMG of the cervical flexors [26], and response to botulinum toxin and deep brain stimulation (DBS), although beneficial responses are not universal [14•, 15]. In general, this subgroup of patients is not weak on extension, and is less likely to have EMG findings consistent with myopathy of the cervical extensors [15]. Extensor pseudohypertrophy may be interpreted as a result of compensatory activity of this muscle group.

Myopathy

Clinical findings of extensor weakness and flexor atrophy reminiscent of INEM or "dropped head" seen in other neuromuscular diseases led clinicians to investigate for myopathy of the extensors. Multiple studies have been able to document "myopathic features" on EMG and biopsy [18–20, 30]. However, findings are inconsistent and there are conflicting reports. Some studies report definite myopathy on 100 % of their cohort [10, 18] while others found only nonspecific changes (Table 1) [7, 26]. These findings may represent the fact that the former excluded patients without extensor weakness and the latter excluded patients with extensor weakness. It is possible that subtypes of this condition exist which would explain these conflicting findings. Importantly, it is difficult to draw meaningful conclusions, particularly with respect to a causal relationship, without knowing what the EMG or biopsy findings of the cervical extensors are in advanced parkinsonian patients without anterocollis. Although recent biopsy studies have used age- and gender-matched controls, these controls did not have advanced PD [18, 19].

Rigidity

Disproportionate axial rigidity in this patient population, as well as reports of response to dopaminergic therapy in some patients, has led some investigators to hypothesize that this is the primary underlying etiology. One group set out to prove this hypothesis by using surface EMG of the SCMs, and demonstrating an increase in activity with passive extension of the neck, with a relative inactivity at rest. This increase in activity resolved with lidocaine injections to this muscle group. Based on these findings the authors concluded the primary etiology may be anterior neck muscle rigidity [13]. Axial rigidity has also been studied in the context of truncal postural changes like camptocormia. In camptocormia, axial rigidity in PD can be thought of as a compensatory measure for the abnormal posture [31] or it can be caused by "balance correcting responses" [32].

Therapeutic Interventions

No randomized controlled studies have been performed in patients with anterocollis or camptocormia to establish safety, tolerability, or efficacy of any treatment modality. Case reports, series, and retrospective studies have reported their experience with the following treatments. A suggested treatment algorithm based on these therapies, and the author's clinical experience, is shown in Fig. 1.

Conservative Therapies

Most patients should be offered conservative therapy either as an adjunct to other therapies or as the primary approach. Conservative therapies include physical therapy to improve posture, and range of motion to avoid contracture. This may

Table 1 EMG findings in parkinsonian anterocollis

Study	Subtype	EMG findings, (*n*)	Biopsy findings, (*n*)
Askmark et al. [10]	Myopathic	Myopathic in all (7)	Myopathic in all (5)
Gdynia et al. [18]	Myopathic	Mixed (19)	Myopathic in all (19)
van de Warrenburg et al. [26]	Dystonic	Nonspecific extensors Overactive flexors (16)	Fibrosis (2)
Yoshiyama et al. [13]	Myopathic	Normal (2)	n/a

be of particular benefit following some therapeutic interventions (eg, botulinum toxin injections). Cervical collars of multiple modalities have been reported to be of benefit (soft or orthopedic types). Corsets are often used in the treatment of camptocormia with short-term reported benefit [19, 33]. There are isolated reports of more ingenious devices like backpacks to improve posture in camptocormia [34] or baseball cap orthoses for anterocollis [35], and patients may manufacture their own, achieving some relief.

Dopaminergic Medications

Severity of anterocollis has been reported to improve with dopaminergic therapies in multiple reports [16•]. However, peak-dose dystonic anterocollis can also occur as a result of dopaminergic therapy. There are a few reports of anterocollis and camptocormia occurring soon after initial therapy with dopamine agonists, and improvement or resolution once they are discontinued [5, 7, 23–25]. Other studies have also found there to be an association with the onset of anterocollis, particularly of the myopathic subtype, and dopamine agonists [15]. If a close temporal association is found with relation to dopamine agonist use or any other drug (eg, neuroleptics), a trial off of this drug class is warranted.

Botulinum Toxin

There are two groups of muscles that, if activated, can be responsible for anterior flexion of the head and neck: the superficial cervical flexors (SCMs) and the deep cervical flexors (longus colli and capitis). In idiopathic cervical dystonia with predominant anterocollis, treatment with botulinum toxin is often difficult, patients can be refractory to injections of the superficial flexors, and the deep flexors may need to be targeted. In dystonic anterocollis, targeting the deep flexors using fluoroscopy [36], CT guidance [37], fluorodeoxyglucose (^{18}F) (FDG)-positron emission tomography (PET)/CT guidance [38, 39], and more recently, using EMG guidance alone [40], has been reported to be effective. Experience with injection of superficial cervical flexors in parkinsonian anterocollis has demonstrated modest results at best, with multiple reports of lack of efficacy [16•, 26]. There are no reports of botulinum toxin injections of the deep flexors in parkinsonian anterocollis; however, one FDG-PET study showed lack of hypermetabolism of the deep flexors, in contrast to reports of deep flexor hypermetabolism in dystonic anterocollis [41]. For the treatment of camptocormia, injections to the rectus abdomini have also been performed, with similar mixed results [21•]. General guidance for the use of botulinum toxin in parkinsonian anterocollis or camptocormia is to reserve its use for patients

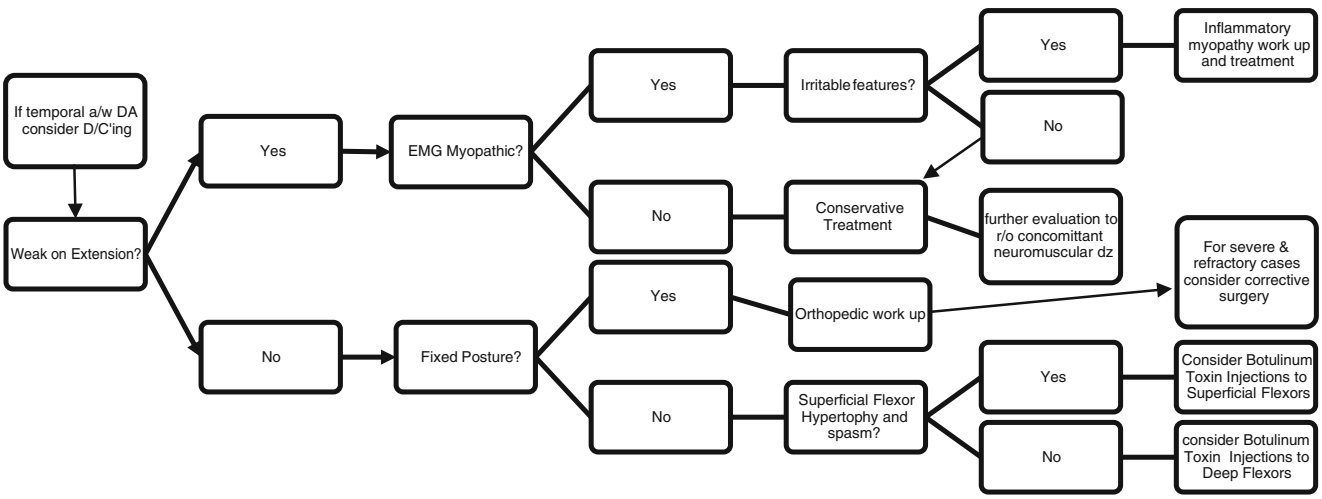

Fig. 1 A suggested treatment algorithm for parkinsonian anterocollis. *DA* dopamine agonist; *a/w* association with; *EMG* electromyography; discontinuing; *dz* disease; *r/o* rule out

with overactive, hypertrophied cervical or abdominal flexors, and take every precaution to avoid dysphagia in the anterocollis group. If myopathy is thought to be the primary etiology then botulinum toxin should be avoided.

Therapies of Myopathy

Multiple groups have reported inflammatory EMG and biopsy findings in their cohorts of parkinsonian anterocollis. These patients have largely been treated with immunosuppressive agents and many of these have improved significantly, although not all achieved full recovery [16•, 18, 20, 42]. Patients with anterocollis, particularly those with neck extensor weakness, should have a neuromuscular evaluation including EMG. If EMG is consistent with an inflammatory myopathy, further workup including biopsy is warranted, and treatment with immunosuppressive agents should be considered. Some have suggested creatine therapy but there is no data to support its use.

Deep Brain Stimulation

Very little evidence exists to support the use of DBS for the treatment of anterocollis. There have been some reports of improvement [15, 43], no effect [5], and others of worsening [21•]. Both the subthalamic nucleus and globus pallidus internus have been targeted [5]. There is no indication to pursue DBS for this patient population unless they meet prespecified criteria to treat other medically refractory motor symptoms.

Orthopedic Surgery

It is important to evaluate for orthopedic causes of abnormal postures, including degenerative changes and vertebral compression fractures with secondary radiculopathy and myelopathy. MRI of the cervical and thoracic spine should be performed if radicular or myelopathic features arise, and referral for surgical evaluation made if abnormal findings requiring surgical repair are identified. Bone density should also be monitored, and abnormal findings should be treated. A small number of patients have undergone elective surgery to correct the abnormal posture, also with mixed results [44].

Conclusions

The phenomena of anterocollis and camptocormia remain poorly understood entities. Patients with these conditions are functionally disabled, and there are no available therapies with proven efficacy. Currently, clinicians have very little guidance from the literature regarding evaluation and treatment of postural deformities in parkinsonian patients. This is largely due to the heterogeneous presentation and clinical features. It is now clear that multiple subtypes of anterocollis exist and, henceforth, each subtype may respond differently to individual treatments. Less is known regarding the heterogeneity of camptocormia, and although these postural abnormalities are often discussed jointly (since dystonic and myopathic causes are considered for both) they are probably not one in the same. Future studies attempting to establish clinical efficacy of a particular therapy should take into consideration the heterogeneity of these disorders when devising inclusion and exclusion criteria. Clinical trialists should also take into consideration that these are evolving conditions by documenting stage of progression. Ideally, a prospective trial would be able to differentiate stage and subgroup a priori, and determine efficacy for each group.

Another confounding factor in both our understanding of these conditions and in the design of effective clinical trials is the lack of objective tools to study and measure conditions like dystonia. Further research studies should focus on the development of novel tools to determine the underlying pathophysiology of these conditions, as well as developing clinical outcome measures, and biomarkers that would aid in determining the efficacy of treatment modalities being investigated.

Disclosure Conflicts of interest: G.J. Revuelta: has received grant support from Biotie, Phytopharm, and NIH; and has received payment for lectures including service on speakers' bureaus from Teva and Lundbeck.

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