Journal of the American Chiropractic Association – April 1999 - PP. 32-37, 57-58

The Symptomatic Upper Extremity: An Algorithmic Approach to Diagnosis, Part 1

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Numbness in an arm or hand is a common presenting or secondary patient complaint. Carpal tunnel syndrome poses a familiar mechanism and is frequently cited as the cause of the patient's symptoms. While it may be the most common upper-extremity peripheral nerve entrapment neuropathy, a myriad of other nerve entrapment sites and pathologies are capable of producing upper-limb symptoms. In this article, an algorithmic approach by topographical region is utilized to narrow or expand the diagnostic focus.

Algorithms have been looked upon as a "cookbook" approach to diagnosis. To the specialist, who has formulated multi-dimensional pathways, which reside solely in his hippocampus, this may be particularly true. But for the rest of us who see and treat a broad variety of disorders, the challenge is to expand our focus and broaden the list of differential diagnoses. Algorithms area unique instrument that serves this very purpose. Algorithms can bring to the forefront other considerations – for example, maybe that hand numbness is due to median nerve entrapment beneath the arch of the flexor digitorum superficialis, and not carpal tunnel syndrome after all. By no means does our assumed end point of a linear flow chart point us to a definitive diagnosis. Rather, it broadens our awareness and reminds us of what may be, and nudges us to perform new and unfamiliar tests that bring more data for synthesis.

The body of the text will supplement the two algorithms found in this article (four more will come in the upcoming issues of the *JACA*). The discussion will be brief, and limited primarily to diagnostic concerns.

Figure 1 generates a list of conditions that can produce symptoms in a limb that do not follow a typical neuroanatomical pattern (i.e. nerve root, peripheral nerve).

Thoracic Outlet Syndrome

Thoracic outlet syndrome (TOS) refers to the clinical presentation that occurs when neurovascular structures entering the arm are compromised. Thoracic outlet syndrome has been categorized pathologically as the result of compression upon the lower trunk of the brachial plexus or subclavian artery or vein. While the latter was the prevailing view in early literature, technological 'advancements in diagnosis have refuted this assumption. Today, neurogenic causation appears in the literature as a favored mechanism. Interestingly, some of the controversy surrounding the discussion of TOS is whether or not it even exists. Despite questions, however, it will not disappear from the diagnostic armamentarium any time soon. Rather, it will remain a diagnosis that is entertained when a patient presents with diffuse, non-dermatomal pain or paresthesia of a single limb, particularly if the complaint is more prominent in the medial brachium, antebrachium, and ulnar aspect of the forearm and hand. Vigorous action with the involved arm, especially repetitive or sustained tasks done overhead, is likely to exacerbate the complaint. Occasionally, autonomic disturbance like cool skin, blanching, and trophic changes may accompany limb pain and paresthesia. The autonomic findings occur because autonomic fibers that supply the upper limb travel almost exclusively through the lower trunk of the brachial plexus. Patients with long-standing disease may show weakness and

wasting of intrinsic hand muscles, the muscles of the hypothenar eminence, and thenar eminence.[1]

As with most conditions, a search for a treatable cause of TOS is warranted. Pathologies with a benign influence account for the majority of TOS and include: fibrotic bands or adhesions, an anomalous first rib, or hypertrophied scalene muscles. Rarely is the underlying pathology serious, but if an apical lung tumor or aneurysm of the subclavian artery is identified, a surgical consultation is necessary. The diagnostic work-up should include a thorough neurological exam, peripheral vascular testing including auscultation of the neck vessels, orthopedic tests such as Adson's, Wright's, Roos', and costoclavicular tests, and plain film evaluation. Rarely is EMG necessary but is usually performed when uncertainty exists about the diagnosis (e.g., differentiating ulnar neuropathy and CB radiculopathy) or when the patient has failed to respond after a trial of conservative care. Somatosensory Evoked Potentials have been regarded favorably, and thermography has been reported as a sensitive and non-invasive procedure that may help establish the diagnosis.[2]

Non-surgical, conservative interventions have been used successfully and include myofascial release techniques, stretching, and postural training (i.e., awareness of drooping shoulders or slouched positions).[2] Manipulation of the lower cervical motion units and first rib and use of modalities (e.g., ultrasound) may prove beneficial. A shoulder orthosis can counterbalance downward traction on the brachial plexus and reduce the tension on it, thereby relieving symptoms.[3] In a study looking at response to conservative treatment by Novak, Collins, and Mackinnon, poor overall outcome was related to obesity, workers' compensation, and associated carpal or cubital tunnel syndrome.[4]

When the diagnosis of thoracic outlet syndrome is made, it is important to investigate whether a treatable pathology is present. A conservative approach can be safely and effectively utilized once a surgical lesion has been determined not to be present.

Classic Migraine Headache

The aural phenomenon of classic migraine headache takes place in the cortical gray matter of the brain. Topographically, the thumb and tongue occupy disproportionate areas of the cortex. This is why patients with classic migraine often experience paresthesia, during the aural stage, that is first noticed in the thumb. Like seizure, as the wave of migraine spreads across the cortex, there will be a march or migration of the paresthesia from the point of origin to the remainder of the limb or body-limited by the sagittal plane. The diagnosis can be established with confidence ii' the paresthesia is accompanied by visual disturbances that develop gradually and have a progressive but relatively short life (10 to 60 minutes). The limb paresthesia and visual disturbances develop early, but then fade as the typical hemicranial headache intensifies. During the headache, which may last between four and 84 hours, many migraineurs will suffer photophobia, phonophobia, nausea, or vomiting concomitant to the headache.[5]

Simple Partial Sensory Seizure
Spencer, et al., made the observation that simple partial (focal) seizure, alone or with complex partial or tonic-clonic seizure, is nearly always associated with neoplasms, especially in an adult with no previous history of seizure.[6] While the presence of movement disorders unequivocally warrants investigation, the presence of episodic paresthesia in a limb is more easily overlooked because of' the myriad of benign conditions that come to mind (e.g., carpal tunnel syndrome). Clues to simple partial sensory seizure are migration or spread of the paresthesia, involvement of the face, stereotypical occurrence, and an obvious post-ictal period of nausea or feeling "out of' sorts." Unlike migraine, the ictus of simple partial sensory seizure is shorter (usually minutes) and is not followed by headache.

Because simple partial sensory seizure may be a harbinger of intracranial pathology, referral for

electroencephalogram (EEG) and advanced imaging such as CT or MRI is indispensable to the diagnosis. In the absence of a surgical lesion, simple partial sensory seizure can be controlled with medication.

Transient Ischemic Attack

Transient ischemic attacks (TIA) occur when there is a temporary interruption of blood to the brain. The ischemia is usually reversed within two to 20 minutes from onset, and neurological sequellae do not survive beyond the event. Unlike migraine and seizure, the effect of a transient ischemic attack is usually subcortical. As a result, anesthesia and weakness are more characteristic than paresthesia and movement disorders. The onset and resolution of transient ischemic attack are very distinct, something that further distinguishes them from migraine or seizure.

Most of the time, transient ischemic attacks occur following thromboembolism due to atheromatous lesions,[7] but they may also occur with neoplasm, arteriovenous malformation, occlusion of the proximal subclavian artery, and cerebral vasospastic disorders.[8] In younger patients with TIA-like events, a search for intracranial pathology with advanced imaging procedures should be undertaken. For the older patient, prompt attention should be given since the occurrence of TIA may warn of impending stroke. Evaluation of the great vessels and a thorough cardiac examination are necessary. A routine physical should include auscultation of the heart and carotid arteries. Duplex imaging of the carotid arteries, trans-esophageal echocardiogram, chest x-ray, and EKG can be appropriately performed in this clinical situation.

If an intracranial lesion is identified, a neurosurgical consultation is required. Atherothrombotic occlusion or cardiac emboli should prompt a referral to a vascular specialist.

Ischemic Stroke - Emboli or Atherothrombosis?

Primary ischemic strokes account for about 80 percent of all strokes. Atherothrombosis is responsible in 50 percent and emboli in the remaining 30 percent.[7]

Infarction secondary to an embolic process frequently occurs without warning. The onset of neurological loss may be sudden, rapid, and maximal. Isolated aphasias without hemiparesis are classic and occur when an embolus lodges in a terminal branch of the left middle cerebral artery. Rapid recovery may occur, but this may be followed by worsening as the bland infarction suddenly hemorrhages. Although headache may be present, it is generally not of the severity that typifies a hemorrhagic stroke. A history of heart disease (e.g., atrial fibrillation, bacterial endocarditis, myxoma, mitral valve prolapse, myocardial infarction) may be the sole identifying risk factor for emboli, and 75 percent of these cardiogenic emboli lodge in the brain.[9] Patients may survive and recover, but are likely to have another event occur in a different vascular distribution. Thus, multiple events occurring in different vascular distributions are a diagnostic clue that suggests embolic disease.

Cerebral vessel thrombosis, unlike embolic disease, is usually preceded by transient ischemic attacks. If infarction occurs, a stuttering progression of neurological loss over a period of 48 to 96 hours further distinguishes this process from embolic disease. Many patients, however, who suffer an atherothrombotic infarction will awake from sleep with a paralytic deficit. Because atherothrombosis has its effect at more proximal sites in the vessel, it involves larger territories and subsequently results in more complex neurological loss than embolic precipitated events.

The evaluation of infarction hopes not only to establish whether the process is atherothrombotic or embolic, but also to identify underlying conditions that require treatment. It further recognizes

that patients who suffer acute stroke are at risk for subsequent sequellae (e.g., secondary emboli, hemorrhagic conversion). Magnetic resonance imaging (MRI) is preferable to computerized tomography (CT) for the evaluation of intracerebral infarction. Echocardiogram or transesophogeal echocardiogram may be employed to evaluate cardiac sources of emboli, and carotid duplex imaging is useful to screen for carotid stenosis.

Hemorrhagic Stroke

Hemorrhagic stroke may be either intracranial or subarachnoid in location. Hypertension accounts for the majority of the former and rupture of an aneurysm or arteriovenous malformation occurs in the latter. Cranial hemorrhage is considered when a patient presents with a sudden onset of neurological deficit accompanied by an explosive and unusually severe headache. These neurological effects are global and involve large regions of the body. Weakness and paralysis are usually limited by the sagittal plane (i.e., affecting only one side of the body), but, unlike an entrapment neuropathy, are not confined to the distribution of a single nerve root or peripheral nerve. Elevated intracranial pressure may produce pathological reflexes (e.g., Babinski, Hoffman), papilledema and cranial nerve palsies (e.g., external ophthalmoplegia, lower facial weakness). CT visualizes the acute hemorrhagic stroke well. MRI approaches the sensitivity of CT between six and 24 hours from onset and is superior to CT after 24 hours.[10-12] Deterioration can be rapid, requiring immediate stabilization in an acute-care center.

Brachial Neuritis

The diagnosis of brachial neuritis or neuralgic amyotrophy is reserved for a characteristic presentation involving the upper limb. It is marked clinically by a rapid onset of weakness and intense pain localized to one or both shoulders and occasionally involving the entire extremity. The limb pain decreases over a period of three to 10 days, during which time atrophy becomes apparent. Neck pain is typically absent, coughing doesn't aggravate, and the deficit extends beyond the distribution of a single nerve root-findings that would suggest cervical disc herniation. ~ is helpful to rule out disc disease, whereas electromyography (EMG) helps determine the extent of involvement and can provide information regarding disease progress. Although its natural course can be lengthy, most patients will eventually improve spontaneously. Efforts to prevent joint contractures and maintain motion to minimize functional loss must be emphasized.[13]

Combined Plexus Lesion

The antecedent history in brachial neuritis may be unremarkable or reveal only a recent upper-respiratory-tract infection, injection, or surgical procedure. A combined plexus lesion, however, is likely to produce a history of significant trauma and subsequent whole-limb weakness and sensory loss below the shoulder. The upper trunk of the brachial plexus is vulnerable to traction injuries (i.e., an increase in the angle between the head and neck), and the lower trunk is damaged during avulsive forces (i.e., hyperabduction of the arm overhead). In the absence of trauma, a neoplastic process must be ruled out, whether or not a Homer's syndrome is concomitant. In a patient who has received radiation treatments of the lung, breast, head, neck, or thyroid, differentiating a radiation-induced plexopathy from recurrent tumor plexopathy is necessary. An electrodiagnostic consultation can be helpful at this point.

Figure 2 generates a list of conditions that predominantly involve the lateral brachium (the outside of the upper arm).

Next Month: The second and final portion of this article.

Reference:

1. *Katirji B, Hardy RWJi Classic neurogenic thoracic cutlet syndrome in a competitive swimmer: a true scalenus ant cus syndrome. Muscle Nerve (United States), Feb 1995, 18(2) p229-35*
2. *JAm Oseopath Assoc. 90(8) L990*
3. *Naktztsuchi Y; Saitob S; Hosaka /W,vWatsuda S. Conservative treatment of thoracic outlet syndrome using an orthosis, JHand Swg [Br] (Scotland), Feb 1995,20(1) p34-9*
4. *Novak CB, Collins ED, Mackinnon SE. Outcome following conservative management of thoracic outlet syndrome. J Hand Sury [Am] (Un/ted States), Jul1995, 20(4) p542-8*
5. *Headache classification committee of the International Headache Society. Classification and diagnostic criteria for headache disorders, cranial neuralgias and facial pain. Cephalgia 1988, 8(suppl. 7): 1-96*
6. *Spencer D, et aL Intracerebral masses in patients with intractable part/a/epilepsy. Neurology 1984; 34:432-6*
7 *Kelly R. Berger J TM and major stroke. Postgraduate Medicine 1992; 91(6): 197-202 & Donnan G. Investigation of patients with stroke and transient ischemic attacks. Lancet 1992; 339:475-77*
9. *Jaigobin CS, Perry JR, Carotid artery stenosis. Selecting candidates for surgical management. PGM 1994; 96(5): 61-72*
10. *Hect-L&witt C, Gomori JM, Grossman RI et aL High-field MRI of hemorrhagic cortithi infarction, AJNR 1986; 7:581 -585*
11. *Goinori JM, Grossman RI, Goldberg HI, et aL High field MRI of intracranial hematomas, Radiology 1985; 1:87-93*
12. *Edebnan RR, Johnson K, Buxton R, et aL MR of hemorrhage: a new approach. AJNR 1986; 7: 751-756*
15. *Plezbert J, Nicholson C., Dorsal scapular nerve entrapment neuropathy: A unique clinical syndrome. JNMS 1994;2(4) 206-211*

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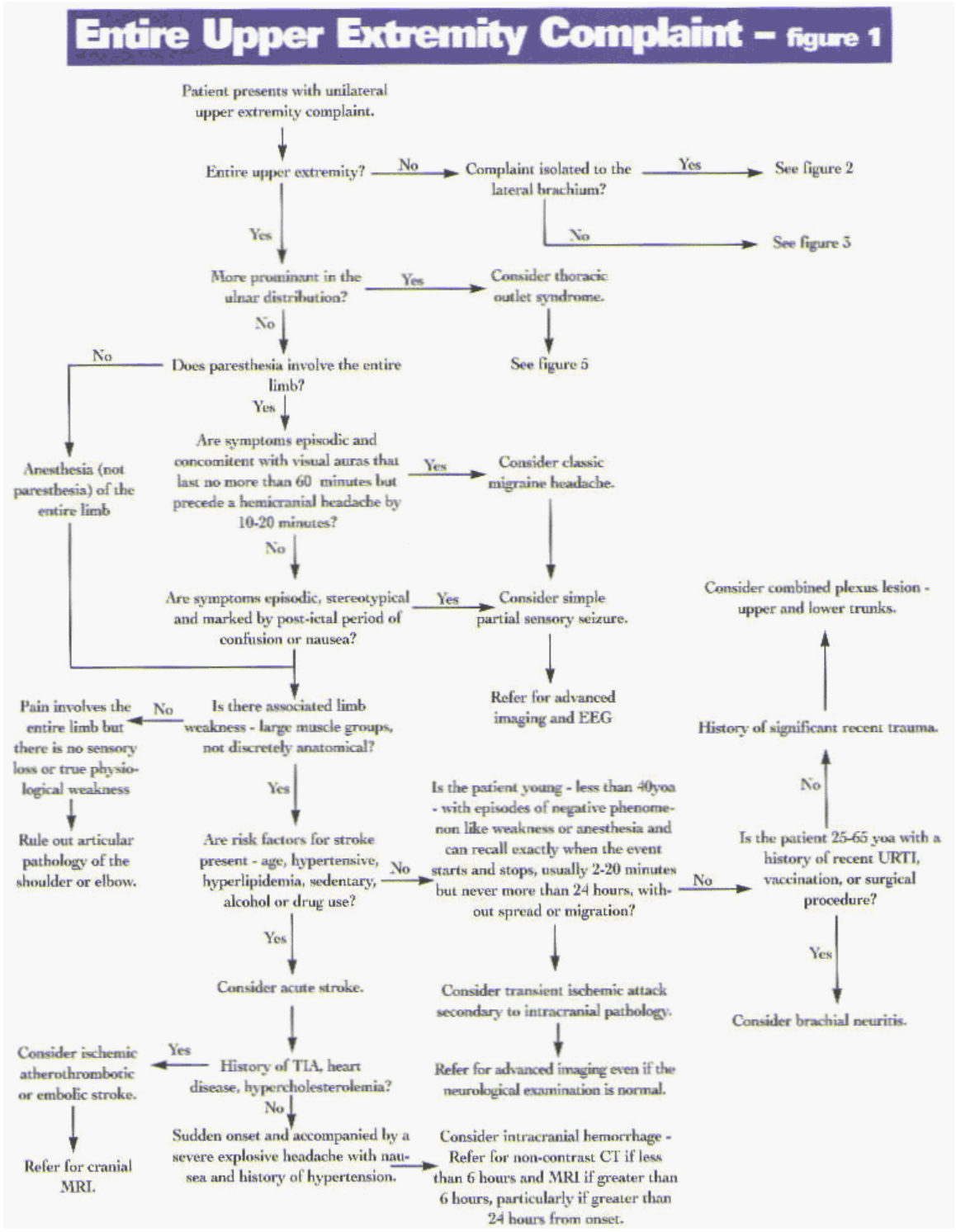
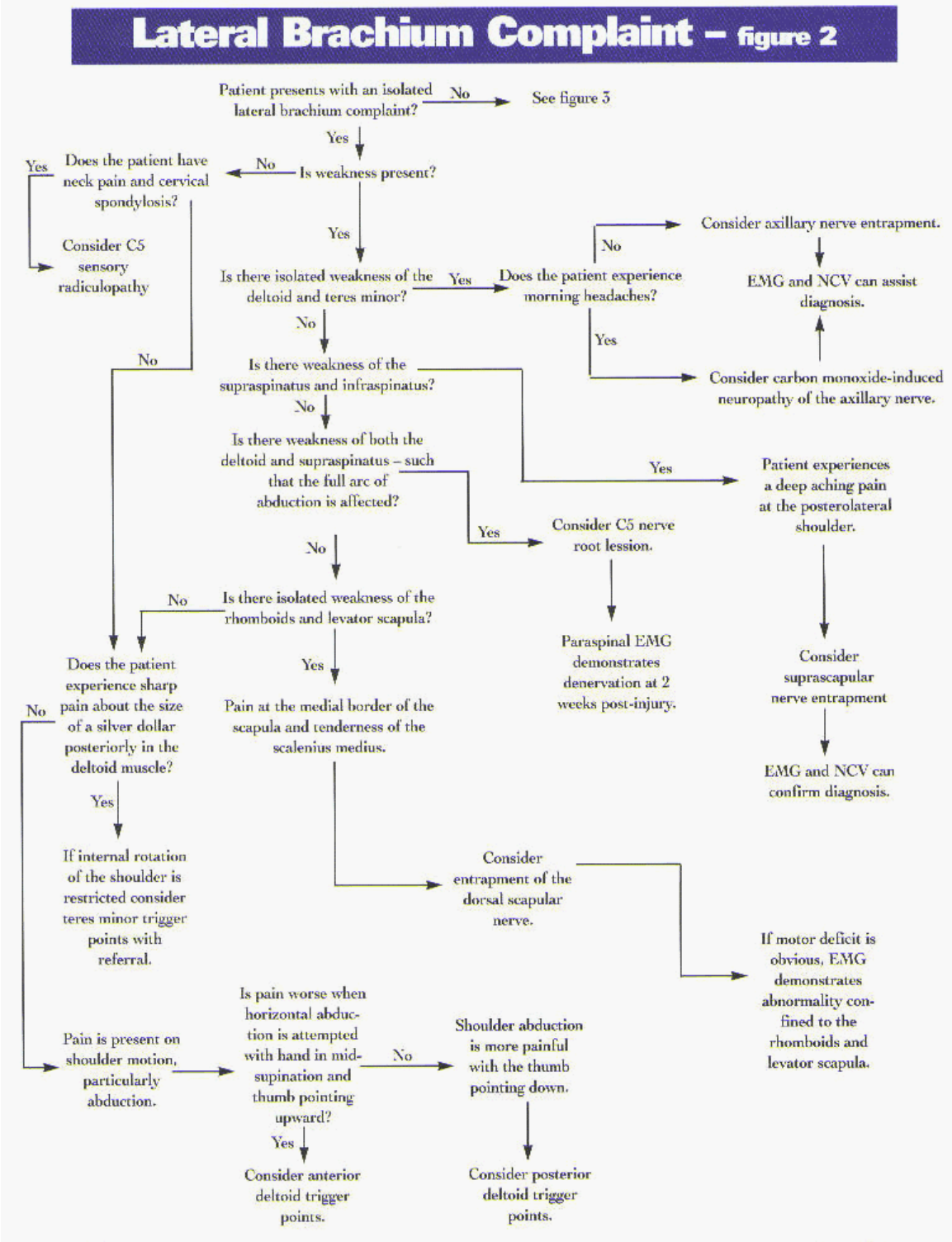
Entire Upper Extremity Complaint – figure 1



Lateral Brachium Complaint – figure 2



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