

Pattern of muscle involvement according to needle electromyography findings in clinically unaffected extremities of polio survivors with lower extremity weaknesses

Alt ekstremitelerde güçsüzlüğü olan polio hastalarının klinik olarak etkilenmeyen ekstremitelerinde iğne elektromiyografi bulgularına göre kas tutulum paterni

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Abstract

Aim: Late neuromuscular deterioration may be seen in patients with a history of paralytic poliomyelitis. One of these problems is the development of a new weakness in clinically unaffected muscles. We aimed to determine needle electromyography (EMG) findings in these clinically unaffected limb muscles and to contribute to the physiotherapy strategies of poliomyelitis.

Methods: Patients with sequelae of poliomyelitis were included in this retrospective cohort study. Needle EMG findings of the patients were reviewed. If there were neurogenic needle EMG findings in the limb or muscle with no weakness, this muscle or limb was considered to be a subclinically affected muscle or limb.

Results: Eighteen patients were included in the study. Needle EMG findings of 190 muscles were analyzed. In the lower extremities, 18 (72%) of 25 clinically unaffected muscles had neurogenic needle EMG findings, and 14 (35%) of 40 upper extremity muscles had subclinical involvement. In the lower extremity muscles, this subclinical involvement was significantly higher than in the upper extremity muscles ($P=0.004$). In clinically unaffected upper and lower extremity muscles, the most prominent neurogenic needle EMG findings were in the deltoideus and vastus lateralis muscles, respectively ($P=0.022$ and $P=0.028$, respectively).

Conclusion: Subclinical involvement was more prominent in the lower extremity than in the upper extremity in polio survivors with weakness of lower extremity. The most prominent subclinical muscle involvement in the lower and upper extremities was the vastus lateralis and deltoideus muscles, respectively. We think that physical therapy strategies considering these findings will be beneficial for polio survivors.

Keywords: Needle electromyography, Poliomyelitis, Subclinical involvement

Öz

Amaç: Paralytik poliomyelit öyküsü olan hastalarda geç dönem nöromusküler kötüleşme görülebilir. Bu sorunlardan birisi, klinik olarak etkilenmemiş kaslarda yeni bir güçsüzlüğün gelişimidir. Bu klinik olarak etkilenmemiş ekstremitelerdeki iğne elektromiyografi (EMG) bulgularını saptamayı ve poliomyelitinin fizik tedavi stratejilerine katkıda bulunmayı amaçladık.

Yöntemler: Poliomyelit sekeli olan hastalar bu retrospektif kohort çalışmasına çalışmaya dahil edildi. Hastaların iğne EMG bulguları gözden geçirildi. Güçsüzlüğü olmayan ekstremitelerde veya kasta nörojenik iğne EMG bulguları varsa, bu kas veya ekstremitelerde subklinik olarak etkilenmiş olarak kabul edildi.

Bulgular: Onsekiz hasta çalışmaya dahil edildi. Yüzdoksan kasın iğne EMG bulguları analiz edildi. Alt ekstremitelerde, klinik olarak etkilenmeyen 25 kasın 18'inde (%72) nörojenik iğne EMG bulguları vardı, 40 üst ekstremitelerde kasın 14'ünde (%35) subklinik tutulum mevcuttu. Alt ekstremitelerde kaslardaki bu subklinik tutulum, üst ekstremitelerde kaslarına göre anlamlı olarak daha fazla orandaydı ($P=0,004$). Klinik olarak etkilenmemiş üst ve alt ekstremitelerde kasları içinde, en belirgin nörojenik iğne EMG bulguları sırasıyla deltoideus ve vastus lateralis kaslarında idi (sırasıyla; $P=0,022$ ve $P=0,028$).

Sonuç: Alt ekstremitelerde güçsüzlüğü olan polio hastalarında subklinik tutulum alt ekstremitelerde üst ekstremitelere daha belirgindi. Alt ve üst ekstremitelerde en belirgin subklinik kas tutulumu sırasıyla vastus lateralis ve deltoideus kaslarındaydı. Bu bulguları dikkate alan fizik tedavi stratejilerinin polio hastaları için faydalı olacağını düşünüyoruz.

Anahtar kelimeler: Elektromiyografi, Poliomyelit, Subklinik tutulum

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Ethics Committee Approval: Ethics committee approval was received from Adana City Training and Research Hospital Ethics Committee (number 37/514).

Etik Kurul Onayı: Etik kurul onayı Adana Şehir Eğitim ve Araştırma Hastanesi Etik Kurulu'ndan alınmıştır (no: 37/514).

Conflict of Interest: No conflict of interest was declared by the authors.

Çıkar Çatışması: Yazarlar çıkar çatışması bildirmemişlerdir.

Financial Disclosure: The authors declared that this study has received no financial support.

Finansal Destek: Yazarlar bu çalışma için finansal destek almadıklarını beyan etmişlerdir.

Published: 9/3/2019
Yayın Tarihi: 03.09.2019

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Introduction

Poliomyelitis was a disease that caused deaths and disabilities in children and young adults in between the 1940s and 1950s. It is caused by the RNA enterovirus affecting brain and anterior horn cells of spinal cord and brainstem. Limb weakness due to death of motor neurons is observed in 1-2% of patients infected by this agent [1,2]. Due to the development of vaccines, it is rarely seen in developing countries today [1]. However, late neuromuscular deterioration and musculoskeletal problems in patients with a history of poliomyelitis continue to be a problem. Although some patients with a history of non-paralytic poliomyelitis do not have weakness, it is known that motor neurons are damaged in these patients [1,3]. In addition, it has been shown that the motor units of the limb muscles with no weakness are affected in patients with a history of paralytic poliomyelitis [4,5] because poliovirus affects most of the motor neurons during the acute phase of the disease [1,6,7]. Approximately 50% of these affected motor neurons are destroyed [7]. After the acute phase, the stable disease may worsen as a result of damage to motor neurons. The causes of this neuromuscular deterioration are controversial [1,8]. Furthermore, studies have shown that the death of motor neurons in polio survivors is faster than normal aging. [5,9]. Many factors such as chronic poliovirus infection or excessive exercise can accelerate the death of motor neurons. [1,8]. At this point, it is important to protect the motor neurons and axons of clinically unaffected muscles. According to clinical and histopathological findings, studies on muscle involvement pattern are available in the literature [10,11]. In this study, we wanted to discuss needle electromyography (EMG) findings in clinically unaffected extremities and muscles in polio survivors with lower extremity weaknesses. Thus, we wanted to contribute to the literature and to the physiotherapy strategies of poliomyelitis.

Materials and methods

Subjects

Patients older than 18 years with sequelae of poliomyelitis referred to the EMG Laboratory of Adana City Training and Research Hospital (ACTRH) between July 2018 and July 2019 were included in this retrospective cohort study. EMG laboratory records were reviewed retrospectively. Patients were included in the study if they had a definite history of poliomyelitis and weakness in at least one limb. The ages of the patients, ages of patients at the time of poliomyelitis, neurological examination findings, nerve conduction study and needle EMG findings were included in the analysis. Patients were excluded from the study if they had one of the following: a disease that can cause polyneuropathy such as diabetes mellitus, a surgical history of cervical or lumbosacral region, a major surgical history of extremities and findings suggestive of polyneuropathy in nerve conduction studies. In addition, patients who met the diagnostic criteria of post-polio syndrome were excluded from the study [12]. In neurological examination, the limb with weakness was considered a clinically affected limb, and otherwise, it was considered a clinically unaffected limb. Ethics committee approval was obtained from the ethics committee of ACTRH (number 37/514).

Electrodiagnostic tests

Nerve conduction studies and needle EMG were performed with Cadwell Sierra Summit EMG unit (Cadwell laboratories, Kennewick, Washington, USA). In the nerve conduction studies, surface electrodes were used for recording and stimulation. Nerve conduction studies and needle EMG were performed if the extremities were above 32°C; otherwise, the extremities were heated with a hair dryer. Band-pass filters for sensory and motor nerve conduction studies and needle EMG were set at 20 Hz to 2 kHz, 20Hz to 10kHz and 10Hz to 10kHz, respectively. Nerves were stimulated supramaximally. Sensitivity was 2 mV/division and 10 μ V/division in motor and sensory nerve conduction studies, respectively. Sweep speed was 5 ms/division and 1 ms/division in motor and sensory nerve conduction studies, respectively. Compound muscle action potential (CMAP) and sensory nerve action potential (SNAP) amplitudes were measured from peak to peak. Sensory nerve conduction velocity was calculated using peak latency. Median and ulnar sensory nerve conduction studies were performed orthodromically by stimulating the 1st, 2nd, 3rd and 5th fingers and the palm. Sural sensory nerve conduction study was performed antidromically. Median, ulnar, posterior tibial and peroneal nerve minimum F-wave latencies were determined by evaluating at least 10 responses. We used the reference values for nerve conduction studies recommended by American Association of Neuromuscular and Electrodiagnostic Medicine [13]. According to these suggested values, lower limits of normal median, ulnar, posterior tibial and peroneal CMAP amplitudes were considered to be 4.1, 7.9, 4.4 and 2.6 mV, respectively. Needle EMG was performed visually using a concentric EMG needle electrode (length=50mm, diameter=0.46mm, Bionen medical devices, Florence, Italy). Sensitivity was 50 μ V/division for the analyses of spontaneous activity. It was 200-1000 μ V/division for motor unit potential (MUP) and interference pattern evaluation. Sweep speed was 10 ms/division for the analyses of spontaneous activity and MUPs, and it was 100 ms/division for the analyses of interference patterns. Positive sharp waves (PSW) and fibrillations were carefully evaluated. According to tolerability of the patients, at least 10-20 MUPs were recorded during mild muscle contraction. Duration, amplitude, phase number and interference pattern of MUPs were recorded. MUP was considered neurogenic if: MUP peak to peak amplitude was \geq 4mV and/or MUP duration \geq 15ms or MUP could not be obtained. In our EMG laboratory, as a protocol for poliomyelitis, needle EMG is applied to the muscles of the extremity or extremities with weakness, the muscles of the contralateral extremity with weakness and the muscles of another upper or lower extremity. According to tolerability of the patient, needle EMG was performed to the tibialis anterior, medial gastrocnemius, peroneus longus, vastus lateralis, iliopsoas, first dorsal interosseous, biceps brachii, deltoideus and trapezius muscles. If needle EMG findings in muscle or limb with no weakness showed neurogenic features, it was accepted that muscle or extremity was affected subclinically.

Statistical analysis

The Shapiro-Wilk test was used to determine the distribution of the data. Mean (standard deviation) of numeric data were calculated for descriptive statistics. Pearson's and

Fisher's Chi-squared tests were used to analyze categorical variables. Kruskal-Wallis and Mann-Whitney U tests were used in the analysis of quantitative data. A p value less than 0.05 was considered significant. Statistical Package for the Social Sciences (SPSS IBM Corp; Armonk, NY, USA) 22.0 was used to perform the statistical analysis.

Results

Twenty-nine patients with a history of poliomyelitis were referred to our EMG laboratory. One patient was not included in the study due to diabetes mellitus. In addition, two patients had a suspicious history of poliomyelitis, and seven patients met the criteria of post-polio syndrome, so these patients were also excluded from the study. Eighteen patients were included in the study. The mean age of the patients was 46.5 (8.3) years. Six patients were female. None of them met the post-polio diagnostic criteria. The patients had no pain or paresthesia or newly occurring muscle weakness. Table 1 shows the clinical and demographic characteristics of the patients. Asymmetric clinical features were clearly present in 16 patients. Neurological examination did not reveal any evidence of bulbar involvement. Median and ulnar motor nerve conduction studies were normal in all patients. In 11 patients, the peroneal or posterior tibial nerve CMAP amplitude was reduced, or CMAP could not be obtained. Two patients had mild carpal tunnel syndrome. Median sensory nerve conduction studies of 16 patients were normal. Ulnar and sural sensory nerve conduction studies of all patients were normal.

Muscles of 56 extremities and the trapezius muscle of 16 patients were examined by needle EMG. Three extremities (two lower and one upper extremities) per patient in 16 patients and 4 extremities per patient in 2 patients were included in the study. One of the upper extremities of 16 patients was not included in the study because needle EMG was not performed on these extremities. Needle EMG was performed in 190 muscles. The mean number of muscles examined by EMG per patient was 10.6 (1.8) (min 8, max 16). In 138 (73%) of the 190 muscles, needle EMG findings showed neurogenic features. The needle EMG findings of the all examined muscles are shown in Table 2. Thirty-four (42%) of 81 clinically unaffected muscles had neurogenic needle EMG findings. Two of the 16 trapezius muscles were subclinically affected. Needle EMG findings in clinically unaffected muscles are shown in table 3. Among clinically unaffected lower extremity muscles, vastus lateralis (86%) had the highest rate of subclinical, whereas deltoideus (75%) had the highest rate of subclinical involvement among the clinically unaffected upper extremity muscles. The MUP duration of the vastus lateralis muscle was significantly longer than that of the medial gastrocnemius and tibialis anterior muscles ($P=0.028$, Table 3). The MUP duration and amplitude of the deltoideus muscle were significantly higher than the MUP duration and amplitude of the first dorsal interosseous and biceps brachii muscles ($P=0.046$, $P=0.022$, Table 3). In 18 (72%) of 25 clinically unaffected muscles examined in the lower extremities, needle EMG findings showed neurogenic features, and 35% of the upper extremity muscles were subclinically affected. This subclinical involvement was significantly higher in the lower extremity muscles than in the upper extremity muscles.

($P=0.004$, Table 4). Fifteen of the 188 muscles had PSW or fibrillation or fasciculation potentials. Positive sharp wave or fibrillation potentials were observed in the tibialis anterior, medial gastrocnemius, peroneus longus, vastus lateralis or iliopsoas muscles of 5 patients. Fasciculation was seen in the tibialis anterior muscles of 2 patients.

Clinically, 28 of 36 lower extremities had weakness, and at least one muscle of these lower extremities had neurogenic needle EMG findings. When needle EMG findings were taken into consideration, abnormal needle EMG findings in at least one muscle of extremity were present in 35 of 36 lower extremities. Two patients had weakness in one upper extremity. Ten upper extremities were affected subclinically. Two patients with clinically affected upper extremities had neurogenic needle EMG findings in other upper extremity muscles with no weakness. There were no weaknesses in 28 extremities (20 upper extremities, 8 lower extremities). In 15 (54%) of 28 clinically unaffected extremities (8 upper extremities, 7 lower extremities), neurogenic needle EMG findings were present in at least one muscle of the extremity. Subclinical involvement in the lower extremities was significantly higher than in the upper extremities ($P=0.029$, Table 4). The affected body regions according to the clinical findings of the patients and the needle EMG findings of the muscles are shown in figure 1.

Table 1: Clinical features of patients

Parameter	Value
Age mean (SD) (min-max) (year)	46.5 (8.3) (31-59)
Age at acute poliomyelitis mean (SD) (min-max) (year)	2 (1.3) (0.4-5)
The interval between age at the acute poliomyelitis and age mean (SD) (min-max) (year)	46 (8.5) (29-58)
Sex Female / Male	6 / 12
Clinically affected limbs of patients	
Number of patients with weakness in one lower extremity	7 patients (6 with weaknesses in the left lower extremity, 1 with weakness in right lower extremity)
Number of patients with weakness in two lower extremities	9 patients (4 with weaknesses in left-right lower extremities, 3 with weakness in right-left lower extremities)
Number of patients with weakness in upper and lower extremities	2 patients (1 with weaknesses in the left upper extremity + right-left lower extremities, 1 with weakness in the left upper extremity + 1 right lower extremity)
Weakness of bulbar muscles	None
Number of patients with asymmetric clinical features	16 patients

SD: Standard deviation

Table 2: Needle EMG findings of the all examined muscles

Muscle	Right	Left	Total (%)
Tibialis anterior	15 / 18	15 / 16	30 / 34 (88)
Medial gastrocnemius	16 / 17	13 / 15	29 / 32 (91)
Peroneus longus	2 / 2	2 / 2	4 / 4 (100)
Vastus lateralis	16 / 17	11 / 12	27 / 29 (93)
Iliopsoas	13 / 14	15 / 17	28 / 31 (90)
First dorsal interosseous	7 / 16	3 / 4	10 / 20 (50)
Biceps brachii	3 / 16	1 / 3	4 / 19 (21)
Deltoideus	3 / 3	1 / 2	4 / 5 (80)
Trapezius	1 / 8	1 / 8	2 / 16 (13)
Upper extremities muscles			18 / 44 (40.9)
Lower extremities muscles			118 / 130 (91)
Upper extremities, lower extremities and bulbar muscles			138 / 190 (73)

Number of muscles with neurogenic needle EMG findings; total number of muscles examined with needle EMG, EMG: Electromyography

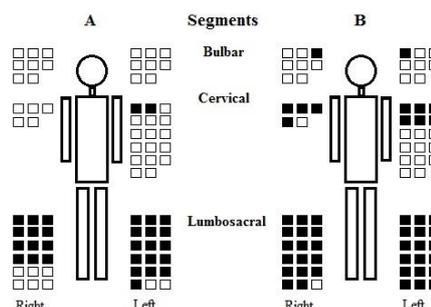


Figure 1: Affected body regions according to clinical features and needle EMG findings. Each box shows an extremity or a trapezius muscle. 1A was made using the clinical features of the patients. In case of clinical weaknesses in the examined extremity or bulbar segment, the box was stained black, and in regions with clinically unaffected muscles, the box was stained white. 1B was made using needle EMG findings of the muscles examined. If neurogenic needle EMG findings were present in at least one muscle of the extremity

or bulbar segment, the box was stained black; otherwise, the box was stained white.
Table 3: Needle EMG findings of clinically unaffected muscles

	Number of muscles of unaffected extremities with neurogenic needle EMG findings / total number of unaffected muscles examined with needle EMG (%)	MUP amplitude of unaffected muscles (mV) Mean (SD)	MUP duration (ms) of unaffected muscles Mean (SD)
Lower extremity muscles			
Tibialis anterior	5 / 7 (71%)	5.1 (2.9)	16.6 (4.4)
Medial Gastrocnemius	4 / 7 (57%)	4.4 (3.5)	14.3 (6.0)
Vastus lateralis	6 / 7 (86%)	8.4 (5.1)	23.0 (3.8)
Iliopsoas	3 / 4 (75%)	5.5 (2.3)	18.0 (4.3)
<i>P</i> -value		0.299	0.028*
Upper extremity muscles			
First dorsal interosseous	8 / 18 (44%)	3.5 (2.9)	13.3 (5.2)
Biceps brachii	3 / 18 (17%)	2.5 (2.6)	14.9 (4.1)
Deltoides	3 / 4 (75%)	7.9 (4.4)	23.5 (8.1)
<i>P</i> -value		0.046**	0.022**
Bulbar muscles			
Trapezius	2 / 16 (13%)	1.9 (2.2)	12.1 (2.9)
Lower extremity muscles	18 / 25 (72%)	5.9 (3.9)	17.9 (5.6)
Upper extremity muscles	14 / 40 (35%)	3.5 (3.3)	15.1 (5.8)
Total	34 / 81 (42%)		

EMG: Electromyography, MUP: Motor unit action potential, *: The MUP duration of the vastus lateralis muscle was significantly longer than that of the medial gastrocnemius and tibialis anterior muscles, **: Both the MUP amplitude and duration of the deltoides muscle were significantly higher than the MUP amplitude and duration of the other two upper extremity muscles, Kruskal-Wallis test was used, and a *P*-value less than 0.05 was considered significant.

Table 4: Comparison of subclinical involvement of upper and lower extremities

	Lower extremity	Upper extremity	<i>P</i> -value
Number of clinically unaffected muscles with neurogenic needle EMG findings / total number of unaffected muscles examined with needle EMG (%)	18/25 (72%)	14/40 (35%)	0.004
Number of clinically unaffected extremities with neurogenic needle EMG findings in at least one muscle / total number of clinically unaffected extremities (%)	7/8 (88%)	8/20 (40%)	0.029
Mean MUP amplitude of clinically unaffected muscles (SD) (mV)	5.9 (3.9) (n=25)	3.4 (3.2) (n=40)	0.007
Mean MUP duration of clinically unaffected muscles (SD) (ms)	17.8 (5.7) (n=25)	15.1 (5.8) (n=40)	0.026

EMG: Electromyography, MUP: Motor unit action potential, n: number of muscles. Pearson's and Fisher's Chi-squared test were used. A *P*-value less than 0.05 was considered significant.

Discussion

Although poliomyelitis is rarely seen in developing countries due to the use of vaccines, the problems of polio survivors continue to be a health problem. In some patients, the disease is stable for many years after acute poliomyelitis, and later neuromuscular and musculoskeletal deteriorations may develop [1]. One of these conditions is post-polio syndrome. The causes of these disorders are controversial. Chronic poliovirus infection, excess weight gain, overuse weakness, changes in muscle fiber metabolism and immune-mediated mechanisms are some of the reasons responsible for these neuromuscular deteriorations [1,8]. Regardless of the cause, the death of motor neurons is faster than normal aging [5,9]. As motor neuron death increases, muscle weakness increases [5,6]. It is known that the motor units of unaffected limb muscles are affected [4,5]. Excessive effort may exacerbate the death of motor neurons [1,8,14]. For these reasons, protection of motor neurons and axons of the unaffected extremity muscles is important. Our study showed that 42% of clinically unaffected limb muscles and 54% of clinically unaffected limbs had neurogenic needle EMG findings, and this finding was consistent with the literature [5]. In our study, considering the clinically unaffected limb muscles and limbs, neurogenic needle EMG findings were found to be more prevalent in the lower extremity muscles than in the upper extremity muscles. It is known that lower extremity muscles are more clinically affected than the upper extremity muscles in

poliomyelitis [15], and we thought that it was important to demonstrate a similar situation in clinically unaffected extremities in our study. We think that physical therapy strategies considering these findings will be beneficial for patients with sequelae of poliomyelitis. Patients with a history of poliomyelitis should stay away from activities that will over-exercise their muscles, especially the lower extremity muscles. However, this finding should not mean over-exercising the upper extremity muscles because poliovirus affects most of the motor neurons [1,6,7]. Note that two patients with upper limb weaknesses had subclinical involvement on the other upper extremities in our study. It may also be important to treat each patient separately. Treatment can be planned considering needle EMG findings because muscles with no weakness may be severely neurogenic in needle EMG examination. In our study, PSW, fibrillation or fasciculation potentials were observed in 5 of (28%) 18 patients and in 15 (8%) of 190 muscles. These findings were consistent with the literature [5]. Presence of active denervation findings in EMG can be seen in patients with poliomyelitis, and it is known that detection of active denervation is not used to differentiate patients with post-polio syndrome from those without [5,15,16].

There have been studies on the pattern of muscle involvement in the upper and lower extremities in poliomyelitis [10,11]. However, these studies were performed according to clinical and histopathological findings rather than needle EMG findings. We found that the most affected subclinical muscle in the upper extremity was the deltoid muscle. The most commonly paralyzed muscle has been shown to be the deltoides muscle [10]. It has been reported in the literature that the muscles innervated by the upper lumbar spinal segments were more affected than the muscles innervated by the sacral segments [11]. In our study, we detected more neurogenic effects in the vastus lateralis muscle innervated by the upper lumbar spinal segments than in the medial gastrocnemius and tibialis anterior muscles innervated by the lower lumbar spinal and sacral segments. These findings may suggest that there are similar patterns of muscle involvement in clinically affected and unaffected extremities.

Limitations

There were some limitations in our study. Firstly, the number of patients and the number of unaffected limbs were low. Secondly, needle EMG was performed to three different muscles in the upper extremity, and more accurate results would be obtained by evaluating the muscles such as triceps or abductor pollicis brevis with needle EMG. Finally, the retrospective nature of the study can be considered as a limitation.

Conclusion

This study showed that clinically unaffected limb muscles could have neurogenic needle EMG findings and that this was more pronounced in the lower extremity muscles in polio survivors with lower extremity weaknesses. Subclinical involvement was most prominent in the deltoides muscle among the examined upper extremity muscles and vastus lateralis among the examined lower extremity muscles. These findings should be taken into consideration, and physical therapy strategies should be planned to protect motor neurons and axons in polio survivors.

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Suggested citation: Patrias K. Citing medicine: the NLM style guide for authors, editors, and publishers [Internet]. 2nd ed. Wendling DL, technical editor. Bethesda (MD): National Library of Medicine (US); 2007-[updated 2015 Oct 2; cited Year Month Day]. Available from: <http://www.nlm.nih.gov/citingmedicine>