УДК 616.134.6/.9-007.271-07
https://doi.org/10.20538/1682-0363-2021-2-210-215

The problem of overdiagnosis of vertebral artery compression syndrome

Shvedov A.N.[1], Ivchenko A.O.[1], Fedorova E.P.[2], Ivchenko O.A.[1]

[1] Siberian State Medical University (SSMU)
2, Moscow Trakt, Tomsk, 634050, Russian Federation

[2] Medical Association ‘’Zdorovie’’
19, Kotovskogo Str., Tomsk, 634034, Russian Federation

ABSTRACT

Vertebral artery compression is a syndrome that occurs as a result of hemodynamically significant partial or complete obstruction of vertebral arteries by extravascular structures. In clinical practice, this condition is most often called vertebral artery syndrome. Any vertebral segments can be compressed, but most often the lesion is determined at the level of C1–C2. Russian authors consider vertebral artery compression to be a common cause of a wide range of patient complaints, including dizziness, headaches, and subjective tinnitus. In some studies, it is reported that vertebral artery syndrome develops in 50% of patients with degenerative changes in the cervical spine.

In the world literature, vertebral artery compression syndrome which is often referred to as “bow hunter’s syndrome” is called a rare pathology. Such a pronounced difference in the frequency of detection of vertebral artery compression in Russian and world literature may be associated with a lack of common diagnostic criteria, low awareness of alternative diagnoses, and incorrect interpretation of patient complaints. It is obvious that these factors need to be corrected in order to reduce the likelihood of overdiagnosis of vertebral artery compression syndrome and improve the quality of medical care.

Key words: vertebral artery syndrome, compression, ultrasound imaging, criteria, overdiagnosis.

Conflict of interest. The authors declare the absence of obvious and potential conflicts of interest related to the publication of this article.

Source of financing. The authors state that they received no funding for the study.

For citation: Shvedov A.N., Ivchenko A.O., Fedorova E.P., Ivchenko O.A. The problem of overdiagnosis of vertebral artery compression syndrome. *Bulletin of Siberian Medicine.* 2021; 20 (2): 210–215. https://doi.org/10.20538/1682-0363-2021-2-210-215.

Проблема гипердиагностики синдрома экстравазальной компрессии позвоночных артерий

Шведов А.Н.[1], Ивченко А.О.[1], Федорова Е.П.[2], Ивченко О.А.[1]

[1] Сибирский государственный медицинский университет (СибГМУ)
Россия, 634050, г. Томск, Московский тракт, 2

2 Медицинское объединение «Здоровье»
Россия, 634034, г. Томск, Котовского, 19

✉ *Shvedov Andrei N.,* e-mail: doc.shvedov@gmail.com.

РЕЗЮМЕ

Синдромом экстравазальной компрессии позвоночных артерий называют симптомокомплекс, возникающий в результате гемодинамически значимой частичной или полной обструкции позвоночных артерий экстравазальными структурами. В клинической практике данное состояние чаще всего называют синдромом позвоночной артерии. Компрессии может быть подвержен любой сегмент, но наиболее часто поражение определяют на уровне С1–С2. Отечественные авторы считают компрессию позвоночных артерий распространенной причиной широкого спектра жалоб пациента, включая головокружение, головные боли и субъективный шум в голове. В отдельных работах сообщают, что синдром позвоночной артерии развивается у 50% пациентов с дегенеративными изменениями шейного отдела позвоночника.

В мировой литературе синдром компрессии позвоночных артерий, который чаще именуется «синдром лучника» (bow hunter's syndrome), называют редкой патологией. Чем обусловлена столь выраженная разница частоты выявления компрессии позвоночных артерий в отечественной и мировой литературе? К возможным причинам указанного феномена могут быть отнесены: отсутствие единых диагностических критериев, низкая осведомленность об альтернативных диагнозах и неверная интерпретация жалоб пациента. Очевидным является необходимость коррекции указанных факторов с целью снижения вероятности гипердиагностики синдрома позвоночной артерии и повышения качества оказания медицинской помощи.

Ключевые слова: синдром позвоночной артерии, компрессия, ультразвуковая диагностика, критерии, гипердиагностика.

Конфликт интересов. Авторы декларируют отсутствие явных и потенциальных конфликтов интересов, связанных с публикацией настоящей статьи.

Для цитирования: Шведов А.Н., Ивченко А.О., Федорова Е.П., Ивченко О.А. Проблема гипердиагностики синдрома экстравазальной компрессии позвоночных артерий. *Бюллетень сибирской медицины.* 2021; 20 (2): 210–215. https://doi.org/10.20538/1682-0363-2021-2-210-215.

INTRODUCTION

Extravascular compression of the vertebral arteries (VA) is a syndrome arising as a result of hemodynamically significant partial or complete obstruction of the VA by extravascular structures. In clinical practice, this condition is most often referred to as vertebral artery compression syndrome (VACS). Any vertebral segments can be compressed (Fig. 1), but most often the lesion is determined at the level of C1–C2 [1].

Russian authors consider VA compression to be a common cause of a wide range of patient complaints, including dizziness, headaches, and subjective tinnitus [2]. There are publications in which VA compression was detected in 45% of patients with dizziness [3]. Also, some studies reported that VACS developed in 50% of patients with degenerative changes in the cervical spine [4].

At the same time, in the world literature, VACS, which is often referred to as “bow hunter’s syndrome”, is called a rare pathology [5, 6]. This is perfectly demonstrated by V. Rastogi et al. (2015) who had founded only 153 cases of VACS from 1966 and 2013 [7]. Such a pronounced difference in the frequency of VACS detection in Russian and world literature may be associated with a lack of common diagnostic criteria, low awareness of alternative diagnoses, and incorrect interpretation of patient complaints.

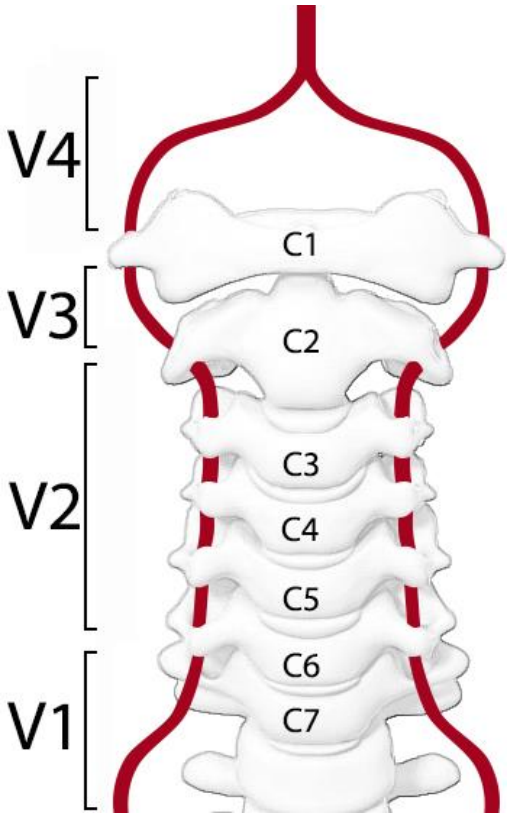

Fig. 1. Vertebral artery segments: V1 – origin to the transverse foramen of C6; V2 – VA ascends through the transverse foramina of the cervical vertebrae; V3 – from the C2 transverse foramen until it enters the skull; V4 – intracranial segment

LACK OF COMMON DIAGNOSTIC CRITERIA FOR VACS

Nowadays, there are no clinical guidelines that propose recommendations for the diagnosis of VACS [8, 9], as a result, different authors use different criteria and methods of patient examination. Computed tomography (CT) and magnetic resonance imaging (MRI) contribute to the diagnosis of VACS, however, obligatory head rotation (maintaining this position for 2 minutes) to demonstrate compression carries a potential risk of neurological complications [10], therefore, researchers use conventional angiography for diagnosis [5, 11]. Most often, the diagnostic criteria for VACS are considered to be intraluminal filling defects in the VA during head rotation with the onset of clinical manifestations. Due to a high cost and potential complications of the procedure, angiography is commonly performed after ultrasound examination [12].

It should be noted that the ultrasound criteria for VACS vary from author to author. For example, in a duplex study, T.V. Zakhmatova et al. (2014) evaluated the effect of extravascular structures on VA by calculating the ratios of blood flow indicators (V3 / V1 maximum blood flow rate; V3 / V1 time-averaged maximum blood flow rate). With the V3 / V1 ratio of more than 1.0, the blood flow was regarded as compensated, and with the ratio being less than 0.7 – as decompensated [13]. M.L. Dicheskul and V.P. Kulikov (2011) demonstrated that the peak systolic velocity (PSV) in the V4 segment in patients with cervical spine pathology during head rotation was significantly decreased compared to healthy volunteers.

The authors suggested a decrease in PSV ≥ 30% as a diagnostic threshold [14]. The same group of authors studied blood flow parameters in the VA during head rotation in patients with vertebrobasilar insufficiency (VBI). Duplex scanning of the suboccipital and intracranial segments of the VA was performed in 28 healthy volunteers and 70 patients with stage I–II VBI according to the classification by A.B. Sitel [15]. The authors concluded that a reduction of PSV ≥ 30% in the V4 segment of the VA during head rotation was a highly specific indicator of extravascular compression – 98% (CI 94.8−99.8%) with a predictive value of 94.6% (CI 81.8−99.2%); the PSV was significantly lower in patients with VBI ($p < 0.05$). It was also noted that the absence of changes in the PSV during head rotation did not guarantee the absence of extravascular compression of VA [16]. The main methodological defect of the study was the use of VBI classification proposed by A.B. Sitel (Table 1).

Table 1

VBI classification proposed by A.B. Sitel (2008)

Stage	Clinical features
I	It is characterized by the predominance of subjective symptoms over objectively detectable disturbances of movement and sensitivity, there is an autonomic dysfunction, mild short-term dizziness, episodes of blurred vision. Functional blockages are detected in the craniocervical junction, the region of the cervicothoracic junction, and the lower back. This stage is also characterized by the inferior oblique muscle syndrome, contracture of the muscles of the neck, anterior chest wall syndrome, and interscapular pain syndrome
II	It is characterized by more intense and longer episodes of dizziness and headache attacks usually starting with pain in the neck or behind the ear region. There are periods of hearing impairment in the form of hearing loss, the appearance of tinnitus and imbalance; visual disturbances in the form of floaters and cloudy vision; pain in the region of the heart, not related to stress or premature ventricular contractions. Functional blockages, in contrast to the first stage, are detected in both the middle and upper cervical regions, the lumbar lordosis is straightened
III	It is characterized by more pronounced clinical presentation: severe headache attacks, analgesics and non-steroidal anti-inflammatory drugs do not bring relief, dizziness with nausea and vomiting, and drop attacks. Attacks are triggered by throwing the head back or turning to the side and using the rolling stairs or transport. Functional blockages are detected in the motion segments of the cervical and lumbar spine
IV	Stage IV clinically coincides with stage III discirculatory encephalopathy, since patients have signs of prior strokes in the carotid arteries and brain stem and / or persistent neurologic deficit

Clinical signs of stages I and II of VBI classification by A.B. Sitel are non-specific and can be manifestations of a wide range of pathological or physiological conditions. Unfortunately, this fact challenges the conclusions of M.L. Dicheskul and V.P. Kulikov, since the study was conducted on a heterogeneous group of patients whose complaints were most likely caused by various diseases (primary headaches [17], benign paroxysmal positional vertigo (BPPV) [18] and others [19]).

The absence of common diagnostic ultrasound criteria for VACS is observed not only in the Russian Federation, but in other countries as well. For example, in Japan. M. Kamouchi et al. (2003) evaluated the qualitative parameters of blood flow in the VA (reduction of the PSV in the V2 segment and disappearance

of the diastolic component during rotation) in patients with suspected VACS [20]. Y. Iguchi et al. (2006) assessed the qualitative characteristics of the blood flow in the posterior cerebral artery (PCA) and basilar artery during transcranial doppler ultrasound [11].

It should be noted that the disappearance of the diastolic component in the VA during head rotation as a sign of VA compression was also described by J. Yeh et al. (2005) [21]. An additional criterion was reduction of PSV by more than 50% in the extracranial segments of the VA during head rotation. The assessment of the blood flow parameters in the PCA with transcranial doppler ultrasound is used in other countries as well. In particular, reduction of PSV in the PCA ≥ 50% (compared to the baseline value) during head rotation is a criterion for VACS in some ultrasound laboratories in the USA and Europe [5, 12, 22].

The criteria are presented in Table 2. In our practice, in patients with suspected VACS, we evaluate blood flow in the V2 segment during head rotation. The patient is recommended to perform angiography to verify the diagnosis, if PSV decreased by more than 50%, the diastolic component is absent, and typical clinical signs of VACS are observed, which is quite rare.

Table 2

Ultrasound criteria of extravascular compression of the vertebral arteries

Author	Method	Criteria
M.L.Dicheskul, 2012 [16]	Duplex study	Reduction of PSV ≥ 30% in the V4 segment of the VA during rotation
T.V. Zakhmatova, 2014 [13]	Duplex study	The V3 / V1 maximum blood flow rate or V3 / V1 time-averaged maximum blood flow rate of more than 1.0 – compensated blood flow; less than 0.7 – decompensated blood flow
M. Kamouchi, 2003 [20]	Duplex study	Qualitative changes in the blood flow in the VA (reduction of the PSV in the V2 segment and disappearance of the diastolic component during head rotation)
J. Yeh, 2005 [21]	Duplex study	Reduction of PSV ≥ 50% in extracranial VA segments and disappearance of the diastolic component during head rotation
Y. Iguchi, 2006 [11]	Transcranial Doppler	Qualitative changes in the blood flow in the PCA and basilar artery (gradual reduction of the blood flow and blood flow cessation with maximum head rotation)
M.D. Vilela, 2005 [12], M. Sturzenegger, 1994 [22], G.F. Jost, 2015 [5]	Transcranial Doppler	Reduction of PSV in PCA ≥ 50% with head rotation followed by reactive hyperemia when returning the head to the neutral position with an increase in PSV > 10% compared to the baseline value

From the data demonstrated above, the absence of common ultrasound criteria for VACS is clearly observed, which affects the difference in the rate of VACS diagnosis in individual diagnostic centers. However, ultrasound examination is preceded by the analysis of complaints and history, which misinterpretation can also lead to overdiagnosis of VACS.

EVALUATION OF PATIENT COMPLAINTS AND HISTORY IN SUSPECTED VACS

Interpretation of patient complaints and history is of great importance, since this is the starting point for formulation of a diagnostic hypothesis [23] and differential diagnosis [24]. The common complaints in VACS include dizziness, headache, nausea, vomiting, and visual disturbances during head rotation relieved by returning to a neutral position [10]. In the literature, these complaints are considered as a part of VBI, which includes only transient ischemic attack and ischemic stroke in the vertebrobasilar arteries. So, VBI is defined solely as stroke (acute cerebrovascular disease).

In Russian literature, VBI is understood as a variant of chronic cerebral ischemia [25], a diagnosis often masked by other diseases (tension-type headache, depressive disorders, etc.) [26]. Perhaps, the difference in the perception of VBI (as an acute condition in the world literature and as a chronic process in the Russian one) contributes to the overdiagnosis of VACS in Russia. At the same time, clinical manifestations of VA compression are non-specific and can occur in various pathologies. For example, BPPV is the most common cause of vertigo in adults [27], and migraine is a common cause of headaches [28]. Both conditions are often accompanied by nausea, vomiting, and visual disturbances. Considering that in a patient with BPPV, a vertigo attack can be triggered by turning the head [29], it is BPPV that should be suspected upon examination, and not rare VACS [7]. However, low awareness of doctors in medical and diagnostic centers about BPPV [30] and chronic migraine [31] can also lead to misinterpretation of patient complaints, followed by overdiagnosis of VACS.

Of course, the reasons for VACS overdiagnosis are not limited to the absence of uniform criteria and misinterpretation of patient complaints. Cognitive biases should also be taken into consideration. They include overconfidence bias, confirmation bias, and anchoring bias. Table 3 provides descriptions and examples of the listed psychological phenomena. More detailed information about them can be found in the study by E.D. O'Sullivan and S.J. Schofield [32].

Table 3

A short list of possible cognitive biases

Cognitive biases	Definition	Example
Overconfidence bias	Overestimation of the likelihood of a diagnosis based on the ease with which it comes to mind	Establishing a diagnosis based on a previous patient with similar clinical presentation
Confirmation bias	Selective preference for evidence supporting the diagnosis	Use of scientific articles that describe criteria for confirming the diagnosis, ignoring alternative publications
Anchoring bias	Striving to adhere to a specific diagnosis despite evidence of refutation	Doctor's refusal to change the diagnosis of VACS despite a peer-confirmed diagnosis of BPPV

CONCLUSION

Different approaches to the interpretation of diagnostic criteria for VACS are reflected in different frequency of diagnosis of VA compression. It is obvious that unified criteria for diagnosis of VACS and raising awareness of doctors about alternative diagnoses (BPPV, migraine, and other diseases) are required in order to reduce the likelihood of VACS overdiagnosis and improve the quality of medical care.

REFERENCES

1. Jadeja N., Nalleballe K. Pearls & Oy-sters: Bow hunter syndrome: A rare cause of posterior circulation stroke: Do not look the other way. *Neurology*. 2018; 91 (7): 329–331. DOI: 10.1212/WNL.0000000000006009.
2. Molokov D.D., Yagunov P.V., Molokov V.D. Rehabilitation therapy for vertebral artery compression syndrome. *Siberian Medical Journal.* 2010; 97 (6): 266–267 (in Russ.).
3. Alekseeva N.S. Vertigo mediated by cervical spine pathology. *Treating Doctor.* 2009; (7): 60–63 (in Russ.).
4. Zinovyeva G.A., Babanina L.P. Vertebral artery compression syndrome in vertebrogenic pathology of the cervical spine. *Bulletin of BolgSMU.* 2006; 1 (17): 9–13 (in Russ.).
5. Jost G.F., Dailey A.T. Bow hunter's syndrome revisited: 2 new cases and literature review of 124 cases. *Neurosurgical Focus*. 2015; 38 (4): 1–15. DOI: 10.3171/2015.1.FOCUS14791.
6. Simpkin C.T., Davis K.E., Davis B.S., Vosko A.M., Jorgensen M.E. Bow hunter's syndrome in a patient with vertebral artery atresia, an arcuate foramen, and unilateral deafness: a case report. *Radiol. Case Rep*. 2017; 12 (3): 597–601. DOI: 10.1016/J.RADCR.2017.04.001.
7. Rastogi V., Rawls A., Moore O. et al. Rare Etiology of Bow Hunter's Syndrome and Systematic Review of Literature. *J. Vasc. Interv. Neurol.* 2015; 8 (3): 7–16.
8. Go G., Hwang S.H., Park I.S., Park H. Rotational vertebral artery compression: Bow Hunter's Syndrome. *J. Korean Neurosurg Soc*. 2013; 54 (3): 243–245. DOI: 10.3340/JKNS.2013.54.3.243.
9. Mileva N.B., Vassilev D.I., Serbezova I., Rigatelli G., Gill R.J. Vertebral artery stenting in a patient with Bow Hunter's Syndrome. *JACC: Case Reports*. 2019; 1 (1): 73–74. DOI: 10.1016/J.JACCAS.2019.05.010.
10. Duan G., Xu J., Shi J., Cao Y. Advances in the Pathogenesis, Diagnosis and Treatment of Bow Hunter's Syndrome: A Comprehensive Review of the Literature. *Interv. Neurol.* 2016; 5(1-2): 29–38. DOI:10.1159/000444306.
11. Iguchi Y., Kimura K., Shibazaki K., Iwanaga T., Ueno Y., Inoue T. Transcranial doppler and carotid duplex ultrasonography findings in Bow hunter's syndrome. *J. Neuroimaging*. 2006; 16 (3): 278–280. DOI: 10.1111/J.1552-6569.2006.00040.X.
12. Vilela M.D., Goodkin R., Lundin D.A., Newell D.W. Rotational vertebrobasilar ischemia: hemodynamic assessment and surgical treatment. *Neurosurgery*. 2005; 56 (1): 36–45. DOI: 10.1227/01.NEU.0000146441.93026.CE.
13. Zakhmatova T.V., Shchedrenok V.V., Moguchaya O.V., Sebelev K.I., Krasnoshlyk P.V. Comparative analysis of vertebral artery hemodynamics in injuries and degenerative diseases of the cervical spine using Duplex color scanning. *Russian Biomedical Journal.* 2014; 15 (2): 352–365 (in Russ.).
14. Dicheskul M.L., Kulikov V.P. The effect of maximum head rotation on the blood flow parameters in the intracranial segment of the vertebral arteries. *Manual Therapy.* 2011; 41 (1): 27–32 (in Russ.).
15 Sitel A.B., Nefedov A.Yu. Treating vertebrobasilar insufficiency with manual therapy – prevention of cerebral stroke. *Manual Therapy.* 2008; 29 (1): 22–31 (in Russ.).
16. Dicheskul M.L., Kulikov V.P. Ultrasound-guided assessment of blood flow dynamics in the vertebral arteries during head rotations. *Fundamental Research*. 2012; (5-1): 22–25 (in Russ.).
17. Yeh W.Z., Blizzard L., Taylor B.V. What is the actual prevalence of migraine? *Brain Behav.* 2018; 8 (6): 1–6. DOI: 10.1002/BRB3.950.
18. Ciorba A., Cogliandolo C., Bianchini C. et al. Clinical features of benign paroxysmal positional vertigo of the posterior semicircular canal. *SAGE Open Med.* 2019; 7: 2050312118822922. DOI: 10.1177/2050312118822922.

19. Nicolas-Puel C., Faulconbridge R.L., Guitton M., Puel J.L., Mondain M., Uziel A. Characteristics of tinnitus and etiology of associated hearing loss: a study of 123 patients. *Int. Tinnitus J.* 2002; 8 (1): 37–44.
20. Kamouchi M., Kishikawa K., Matsuo R. et al. Ultrasonographic detection of extracranial vertebral artery compression in bow hunter's brain ischemia caused by neck rotation. *Cerebrovasc. Dis*. 2003; 16 (3): 303–305. DOI: 10.1159/000071134.
21. Yeh J.F., Lin Y.J., Po H.L. et al. A case of bow hunter's stroke caused by non-dominant vertebral artery. *Acta Neurol. Taiwan.* 2005; 14 (2): 69–73.
22. Sturzenegger M., Newell D.W., Douville C., Byrd S., Schoonover K. Dynamic transcranial Doppler assessment of positional vertebrobasilar ischemia. *Stroke*. 1994; 25 (9): 1776–1783. DOI: 10.1161/01.STR.25.9.1776.
23. Nierenberg R. The chief complaint driven medical history: implications for medical education. *Int. J. Med. Educ*. 2017; 8: 205–206. DOI: 10.5116/IJME.5907.74D8.
24. Heneghan C., Glasziou P., Thompson M. et al. Diagnostic strategies used in primary care. *BMJ*. 2009; 338: b946. DOI: 10.1136/BMJ.B946.
25. Barulin A.E., Kurushina O.V., Puchkov A.E. Vertebrobasilar insufficiency. *Bulletin of VolfSMU.* 2014; 3 (51): 3–8 (in Russ.).
26. Parfenov V.A. Modern aspects of diagnosis and treatment of chronic cerebral ischemia: the use of naftidrofuryl. *Medical Council.* 2015; 18: 11–17 (in Russ.) DOI: 10.21518/2079-701X-2015-18-11-17.
27. Hornibrook J. Benign Paroxysmal Positional Vertigo (BPPV): History, pathophysiology, office treatment and future directions. *Int. J. Otolaryngol.* 2011; 2011: 835671. DOI: 10.1155/2011/835671.
28. Ahmed F. Headache disorders: differentiating and managing the common subtypes. *British Journal of Pain.* 2012; 6 (3): 124–132. DOI: 10.1177/2049463712459691.
29. Jung H.K., Soo B.I., Je H.J., Dong S.S., Sang M.Y., Jong H.M. Bow Hunter Syndrome treated via an anterior approach and selective decompression only. *Biomed. J. Sci. Tech. Res*. 2018; 9 (5): 1–4. DOI: 10.26717/ BJSTR.2018.09.001870.
30. Lebedeva N.V., Zamergrad M.V., Parfenov V.A., Antonenko L.M. Diagnosis and treatment of aptients with benign paroxysmal positional vertigo in routine clinical practice. *Archives of Therapy*. 2017; 89 (1): 57–61 (in Russ.). DOI: 10.17116/terarkh201789157-61.
31. Amelin A.V., Bogdanova Yu.N., Koreshkina M.I., Protsenko E.V., Skoromets A.A., Tarasova S.V. Diagnosis of primary and symptomatic forms of daily chronic headache. *Journal of Neurology and Psychiatry named after S.S. Korsakov.* 2011; 111 (4): 82–84 (in Russ.).
32. O'Sullivan E.D., Schofield S.J. Cognitive bias in clinical medicine. *J. R. Coll. Physicians Edinb.* 2018; 48 (3): 225–232. DOI: 10.4997/JRCPE.2018.306.

Authors information

Shvedov Andrei N., Cand. Sci. (Med.), Assistant, Department of Intermediate Level Surgery, Siberian State Medical University, Tomsk, Russian Federation. ORCID 0000-0002-3967-725X.

Ivchenko Andrei O., Dr. Sci. (Med.), Associate Professor, Head of the Department of Intermediate Level Surgery, Siberian State Medical University, Tomsk, Russian Federation. ORCID 0000-0002-3697-1816.

Fedorova Elena P., Neurologist, Department of Neurology, Medical Association "Zdorovie", Tomsk, Russian Federation.

Ivchenko Oleg A., Dr. Sci. (Med.), Professor, Department of Intermediate Level Surgery, Siberian State Medical University, Tomsk, Russian Federation. ORCID 0000-0002-7686-3481.

(✉) **Shvedov Andrei N.,** e-mail: doc.shvedov@gmail.com.

Received 21.06.2020
Accepted 28.12.2020