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ЕЖЕМЕСЯЧНЫЙ НАУЧНЫЙ ЖУРНАЛ

Медицинские новости Грузии

საქართველოს სამედიცინო სიახლენი

GEORGIAN MEDICAL NEWS

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GMN: Georgian Medical News is peer-reviewed, published monthly journal committed to promoting the science and art of medicine and the betterment of public health, published by the GMN Editorial Board since 1994. GMN carries original scientific articles on medicine, biology and pharmacy, which are of experimental, theoretical and practical character; publishes original research, reviews, commentaries, editorials, essays, medical news, and correspondence in English and Russian.

GMN is indexed in MEDLINE, SCOPUS, PubMed and VINITI Russian Academy of Sciences. The full text content is available through EBSCO databases.

GMN: Медицинские новости Грузии - ежемесячный рецензируемый научный журнал, издаётся Редакционной коллегией с 1994 года на русском и английском языках в целях поддержки медицинской науки и улучшения здравоохранения. В журнале публикуются оригинальные научные статьи в области медицины, биологии и фармации, статьи обзорного характера, научные сообщения, новости медицины и здравоохранения. Журнал индексируется в MEDLINE, отражён в базе данных SCOPUS, PubMed и ВИНИТИ РАН. Полнотекстовые статьи журнала доступны через БД EBSCO.

GMN: Georgian Medical News – საქართველოს სამედიცინო სიახლენი – არის ყოველთვიური სამეცნიერო სამედიცინო რეცენზირებადი ჟურნალი, გამოიცემა 1994 წლიდან, წარმოადგენს სარედაქციო კოლეგიისა და აშშ-ის მეცნიერების, განათლების, ინდუსტრიის, ხელოვნებისა და ბუნებისმეტყველების საერთაშორისო აკადემიის ერთობლივ გამოცემას. GMN-ში რუსულ და ინგლისურ ენებზე ქვეყნდება ექსპერიმენტული, თეორიული და პრაქტიკული ხასიათის ორიგინალური სამეცნიერო სტატიები მედიცინის, ბიოლოგიისა და ფარმაციის სფეროში, მიმოხილვითი ხასიათის სტატიები.

ჟურნალი ინდექსირებულია MEDLINE-ის საერთაშორისო სისტემაში, ასახულია SCOPUS-ის, PubMed-ის და ВИНИТИ РАН-ის მონაცემთა ბაზებში. სტატიების სრული ტექსტი ხელმისაწვდომია EBSCO-ს მონაცემთა ბაზებიდან.

WEBSITE

www.geomednews.com

К СВЕДЕНИЮ АВТОРОВ!

При направлении статьи в редакцию необходимо соблюдать следующие правила:

1. Статья должна быть представлена в двух экземплярах, на русском или английском языках, напечатанная через **полтора интервала на одной стороне стандартного листа с шириной левого поля в три сантиметра**. Используемый компьютерный шрифт для текста на русском и английском языках - **Times New Roman** (**Кириллица**), для текста на грузинском языке следует использовать **AcadNusx.** Размер шрифта - **12**. К рукописи, напечатанной на компьютере, должен быть приложен CD со статьей.

2. Размер статьи должен быть не менее десяти и не более двадцати страниц машинописи, включая указатель литературы и резюме на английском, русском и грузинском языках.

3. В статье должны быть освещены актуальность данного материала, методы и результаты исследования и их обсуждение.

При представлении в печать научных экспериментальных работ авторы должны указывать вид и количество экспериментальных животных, применявшиеся методы обезболивания и усыпления (в ходе острых опытов).

4. К статье должны быть приложены краткое (на полстраницы) резюме на английском, русском и грузинском языках (включающее следующие разделы: цель исследования, материал и методы, результаты и заключение) и список ключевых слов (key words).

5. Таблицы необходимо представлять в печатной форме. Фотокопии не принимаются. **Все цифровые, итоговые и процентные данные в таблицах должны соответствовать таковым в тексте статьи**. Таблицы и графики должны быть озаглавлены.

6. Фотографии должны быть контрастными, фотокопии с рентгенограмм - в позитивном изображении. Рисунки, чертежи и диаграммы следует озаглавить, пронумеровать и вставить в соответствующее место текста **в tiff формате**.

В подписях к микрофотографиям следует указывать степень увеличения через окуляр или объектив и метод окраски или импрегнации срезов.

7. Фамилии отечественных авторов приводятся в оригинальной транскрипции.

8. При оформлении и направлении статей в журнал МНГ просим авторов соблюдать правила, изложенные в «Единых требованиях к рукописям, представляемым в биомедицинские журналы», принятых Международным комитетом редакторов медицинских журналов - http://www.spinesurgery.ru/files/publish.pdf и http://www.nlm.nih.gov/bsd/uniform_requirements.html
В конце каждой оригинальной статьи приводится библиографический список. В список литературы включаются все материалы, на которые имеются ссылки в тексте. Список составляется в алфавитном порядке и нумеруется. Литературный источник приводится на языке оригинала. В списке литературы сначала приводятся работы, написанные знаками грузинского алфавита, затем кириллицей и латиницей. Ссылки на цитируемые работы в тексте статьи даются в квадратных скобках в виде номера, соответствующего номеру данной работы в списке литературы. Большинство цитированных источников должны быть за последние 5-7 лет.

9. Для получения права на публикацию статья должна иметь от руководителя работы или учреждения визу и сопроводительное отношение, написанные или напечатанные на бланке и заверенные подписью и печатью.

10. В конце статьи должны быть подписи всех авторов, полностью приведены их фамилии, имена и отчества, указаны служебный и домашний номера телефонов и адреса или иные координаты. Количество авторов (соавторов) не должно превышать пяти человек.

11. Редакция оставляет за собой право сокращать и исправлять статьи. Корректура авторам не высылается, вся работа и сверка проводится по авторскому оригиналу.

12. Недопустимо направление в редакцию работ, представленных к печати в иных издательствах или опубликованных в других изданиях.

При нарушении указанных правил статьи не рассматриваются.

REQUIREMENTS

Please note, materials submitted to the Editorial Office Staff are supposed to meet the following requirements:

1. Articles must be provided with a double copy, in English or Russian languages and typed or compu-ter-printed on a single side of standard typing paper, with the left margin of **3** centimeters width, and **1.5** spacing between the lines, typeface - **Times New Roman** (**Cyrillic**), print size - **12** (referring to Georgian and Russian materials). With computer-printed texts please enclose a CD carrying the same file titled with Latin symbols.

2. Size of the article, including index and resume in English, Russian and Georgian languages must be at least 10 pages and not exceed the limit of 20 pages of typed or computer-printed text.

3. Submitted material must include a coverage of a topical subject, research methods, results, and review.

Authors of the scientific-research works must indicate the number of experimental biological species drawn in, list the employed methods of anesthetization and soporific means used during acute tests.

4. Articles must have a short (half page) abstract in English, Russian and Georgian (including the following sections: aim of study, material and methods, results and conclusions) and a list of key words.

5. Tables must be presented in an original typed or computer-printed form, instead of a photocopied version. **Numbers, totals, percentile data on the tables must coincide with those in the texts of the articles.** Tables and graphs must be headed.

6. Photographs are required to be contrasted and must be submitted with doubles. Please number each photograph with a pencil on its back, indicate author's name, title of the article (short version), and mark out its top and bottom parts. Drawings must be accurate, drafts and diagrams drawn in Indian ink (or black ink). Photocopies of the X-ray photographs must be presented in a positive image in **tiff format**.

Accurately numbered subtitles for each illustration must be listed on a separate sheet of paper. In the subtitles for the microphotographs please indicate the ocular and objective lens magnification power, method of coloring or impregnation of the microscopic sections (preparations).

7. Please indicate last names, first and middle initials of the native authors, present names and initials of the foreign authors in the transcription of the original language, enclose in parenthesis corresponding number under which the author is listed in the reference materials.

8. Please follow guidance offered to authors by The International Committee of Medical Journal Editors guidance in its Uniform Requirements for Manuscripts Submitted to Biomedical Journals publication available online at: http://www.nlm.nih.gov/bsd/uniform_requirements.html
http://www.icmje.org/urm_full.pdf
In GMN style for each work cited in the text, a bibliographic reference is given, and this is located at the end of the article under the title "References". All references cited in the text must be listed. The list of references should be arranged alphabetically and then numbered. References are numbered in the text [numbers in square brackets] and in the reference list and numbers are repeated throughout the text as needed. The bibliographic description is given in the language of publication (citations in Georgian script are followed by Cyrillic and Latin).

9. To obtain the rights of publication articles must be accompanied by a visa from the project instructor or the establishment, where the work has been performed, and a reference letter, both written or typed on a special signed form, certified by a stamp or a seal.

10. Articles must be signed by all of the authors at the end, and they must be provided with a list of full names, office and home phone numbers and addresses or other non-office locations where the authors could be reached. The number of the authors (co-authors) must not exceed the limit of 5 people.

11. Editorial Staff reserves the rights to cut down in size and correct the articles. Proof-sheets are not sent out to the authors. The entire editorial and collation work is performed according to the author's original text.

12. Sending in the works that have already been assigned to the press by other Editorial Staffs or have been printed by other publishers is not permissible.

Articles that Fail to Meet the Aforementioned Requirements are not Assigned to be Reviewed.

ავტორთა საყურადღებოდ!

რედაქციაში სტატიის წარმოდგენისას საჭიროა დავიცვათ შემდეგი წესები:

1. სტატია უნდა წარმოადგინოთ 2 ცალად, რუსულ ან ინგლისურ ენებზე,დაბეჭდილი სტანდარტული ფურცლის 1 გვერდზე, 3 სმ სიგანის მარცხენა ველისა და სტრიქონებს შორის 1,5 ინტერვალის დაცვით. გამოყენებული კომპიუტერული შრიფტი რუსულ და ინგლისურენოვან ტექსტებში - **Times New Roman (Кириллица)**, ხოლო ქართულენოვან ტექსტში საჭიროა გამოვიყენოთ **AcadNusx.** შრიფტის ზომა – 12. სტატიას თან უნდა ახლდეს CD სტატიით.

2. სტატიის მოცულობა არ უნდა შეადგენდეს 10 გვერდზე ნაკლებს და 20 გვერდზე მეტს ლიტერატურის სიის და რეზიუმეების (ინგლისურ, რუსულ და ქართულ ენებზე) ჩათვლით.

3. სტატიაში საჭიროა გაშუქდეს: საკითხის აქტუალობა; კვლევის მიზანი; საკვლევი მასალა და გამოყენებული მეთოდები; მიღებული შედეგები და მათი განსჯა. ექსპერიმენტული ხასიათის სტატიების წარმოდგენისას ავტორებმა უნდა მიუთითონ საექსპერიმენტო ცხოველების სახეობა და რაოდენობა; გაუტკივარებისა და დაძინების მეთოდები (მწვავე ცდების პირობებში).

4. სტატიას თან უნდა ახლდეს რეზიუმე ინგლისურ, რუსულ და ქართულ ენებზე არანაკლებ ნახევარი გვერდის მოცულობისა (სათაურის, ავტორების, დაწესებულების მითითებით და უნდა შეიცავდეს შემდეგ განყოფილებებს: მიზანი, მასალა და მეთოდები, შედეგები და დასკვნები; ტექსტუალური ნაწილი არ უნდა იყოს 15 სტრიქონზე ნაკლები) და საკვანძო სიტყვების ჩამონათვალი (key words).

5. ცხრილები საჭიროა წარმოადგინოთ ნაბეჭდი სახით. ყველა ციფრული, შემაჯამებელი და პროცენტული მონაცემები უნდა შეესაბამებოდეს ტექსტში მოყვანილს.

6. ფოტოსურათები უნდა იყოს კონტრასტული; სურათები, ნახაზები, დიაგრამები - დასათაურებული, დანომრილი და სათანადო ადგილას ჩასმული. რენტგენოგრამების ფოტოასლები წარმოადგინეთ პოზიტიური გამოსახულებით **tiff** ფორმატში. მიკროფოტოსურათების წარწერებში საჭიროა მიუთითოთ ოკულარის ან ობიექტივის საშუალებით გადიდების ხარისხი, ანათალების შეღებვის ან იმპრეგნაციის მეთოდი და აღნიშნოთ სურათის ზედა და ქვედა ნაწილები.

7. სამამულო ავტორების გვარები სტატიაში აღინიშნება ინიციალების თანდართვით, უცხოურისა – უცხოური ტრანსკრიპციით.

8. სტატიას თან უნდა ახლდეს ავტორის მიერ გამოყენებული სამამულო და უცხოური შრომების ბიბლიოგრაფიული სია (ბოლო 5-8 წლის სიღრმით). ანბანური წყობით წარმოდგენილ ბიბლიოგრაფიულ სიაში მიუთითეთ ჯერ სამამულო, შემდეგ უცხოელი ავტორები (გვარი, ინიციალები, სტატიის სათაური, ჟურნალის დასახელება, გამოცემის ადგილი, წელი, ჟურნალის №, პირველი და ბოლო გვერდები). მონოგრაფიის შემთხვევაში მიუთითეთ გამოცემის წელი, ადგილი და გვერდების საერთო რაოდენობა. ტექსტში კვადრატულ ფჩხილებში უნდა მიუთითოთ ავტორის შესაბამისი N ლიტერატურის სიის მიხედვით. მიზანშეწონილია, რომ ციტირებული წყაროების უმეტესი ნაწილი იყოს 5-6 წლის სიღრმის.

9. სტატიას თან უნდა ახლდეს: ა) დაწესებულების ან სამეცნიერო ხელმძღვანელის წარდგინება, დამოწმებული ხელმოწერითა და ბეჭდით; ბ) დარგის სპეციალისტის დამოწმებული რეცენზია, რომელშიც მითითებული იქნება საკითხის აქტუალობა, მასალის საკმაობა, მეთოდის სანდოობა, შედეგების სამეცნიერო-პრაქტიკული მნიშვნელობა.

10. სტატიის ბოლოს საჭიროა ყველა ავტორის ხელმოწერა, რომელთა რაოდენობა არ უნდა აღემატებოდეს 5-ს.

11. რედაქცია იტოვებს უფლებას შეასწოროს სტატია. ტექსტზე მუშაობა და შეჯერება ხდება საავტორო ორიგინალის მიხედვით.

12. დაუშვებელია რედაქციაში ისეთი სტატიის წარდგენა, რომელიც დასაბეჭდად წარდგენილი იყო სხვა რედაქციაში ან გამოქვეყნებული იყო სხვა გამოცემებში.

აღნიშნული წესების დარღვევის შემთხვევაში სტატიები არ განიხილება.

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BINOCULAR INTERACTION IN AMBLYOPIA

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Abstract.

The review presents new ideas about developmental mechanisms of amblyopia, which are currently discussed in literature. Objective evidence has accumulated that amblyopia affects both monocular and binocular functions in visual processing. Given the increasing evidence of fundamental and clinical research, it is most likely that binocular dysfunction is primary, and monocular reduction is visual acuity is secondary to this disease. Amblyopia is rather binocular than monocular pathology, and binocular interaction seems to play a critical role in the pathogenesis of strabismic, anisometropic and combined forms of amblyopia. According to this theory, it seems appropriate to begin the treatment with the restoration of binocular vision, which also leads to the restoration of the vision of the amblyopic eye. Poor visual functions in amblyopia is associated with the presence of suppression in binocular conditions of vision. Suppression transforms the structurally binocular system functionally monocular. Therefore, the first and most important step to restore binocular vision should be considered the elimination of suppression. The quantification of suppression is a critically important step for the improvement of new therapies for amblyopia based on the global perception of movement associated with the function of the dorsal extrastriate visual cortex. This new understanding of amblyopia now becomes the basis for carrying out a large volume of diverse studies that provide the development of more effective therapies that will primarily focus on eliminating suppression and restoring binocular vision.

Key words. Binocular interaction, amblyopia.

Introduction.

Amblyopia is a monocular or binocular visual reduction in visual acuity that results from prolonged visual deprivation in the early years of life. Amblyopia is a postnatal violation of a development of the visual cortex due to abnormal visual experience during the critical period in infancy or early childhood (the first 7-10 years) and leads to the chronic insufficiency of cortical processes, even after the elimination of the amblyogenic factor [1,2].

Amblyopia affects 3-5% of the population [3] and it is the second to refractive error as the most common cause of vision loss in infants and children. According to the etiological factor, it is divided to anisometropic, strabismic, deprivational due to cataracts or ptosis, and mixed amblyopia. The most common amblyogenic factors are strabismus, anisometropia, and deprivation. In the presence of several amblyogenic factors speak mixed mechanism of amblyopia. The most common is strabismic amblyopia with anisometropia [4-6].

The pathogenetic mechanisms of amblyopia have not been sufficiently studied, despite the data of numerous electrophysiological and psychophysical studies and the presence of ultramodern methods of neuroimaging, for example, functional magnetic resonance imaging (fMRI). Until now, it remains unclear whether there are different or a single pathogenetic mechanism for the development of the various types of amblyopia.

Because amblyopia often involves one eye, it is related to a monocular pathology, and accordingly, the treatment is also monocular. The primary methods of amblyopia treatment are the occlusion of the "healthy" eye and penalization. However, monocular therapy for amblyopia has recently been questioned due to the frequent recurrence of amblyopia after the occlusion was stopped and the presence of residual amblyopia [2].

The current hypothesis about the pathogenesis of amblyopia.

Amblyopia arises from a lack of consistency between the images of each eye; information from one eye becomes more privileged, while the picture coming from the other eye is actively suppressed by the visual cortex [7]. Suppression leads to decreased visual acuity in the amblyopic eye and, therefore, disrupts binocular vision. However, the question arises whether low vision is the cause or consequence of a violation of binocular vision? According to the hypothesis, the primary and leading factor in the development of amblyopia is the disruption of binocular vision, which leads to suppression with further development of visual loss [2,8]. The new hypothesis lays the foundation for an alternative approach to developing more effective treatment for amblyopia.

Alterations in the primary and secondary visual cortex of patients with amblyopia have been shown in numerous studies [9-15]. Recent evidence suggests that in amblyopic patients, there is a deficit of processing of visual information at high levels of the visual system, in the areas of the parietal-occipital and temporal cortex [16]. These areas of higher level of the visual system processing the binocular signal are part of the cortical neural network involved in the 3D vision of the object. The study of the visual cortex of the brain revealed a reduction in the number of binocularly controlled neurons in the primary visual cortex V1 and a decrease in the number of neurons controlled by the amblyopic eye [17,18]. It was found that a violation of fixation of one eye with strabismus [19,20] can be caused by weakening of binocular cortical connections [19]. Also, investigation of the binocular interaction in the V1 region found a reinforcement of binocular suppression [21,22]. An increase of suppression may be associated with a decrease the number of binocularly controlled neurons in the V1 region,

as earlier studies have shown that weakening of suppression by the bicuculline promoted the recovery of more than half of the cortical neurons [23]. Numerous of electrophysiological studies have shown a decrease of amplitude of visual cortical evoked potentials (VEP) and their normal or elongated peak latency when stimulating the amblyopic eye [15,24-26]. Thus, a decrease in the response of the visual cortex may be due to a disruption of binocular vision in patients with amblyopia.

Anisometropic and strabismic amblyopia are characterized by different patterns of visual deficits. Anisometropic amblyopia is associated with proportional deficits in optotype, vernier and grating acuity [27,28] but strabismic amblyopia is associated with a disproportional visual impairment in optotype visual acuity and vernier acuity, compared with grating acuity. The difference in the patterns of visual deficits between the strabismic and anisometropic amblyopia can talk about various pathophysiological processes (the etiological hypothesis) [29].

The second (age) hypothesis implies that the degree of various spatial deficiencies with strabismic and anisometropic amblyopia depends on the onset of the disease, that is, of the level of maturation of the visual system [29]. Anisometropic amblyopia develops later when the visual system is more developed, and the binocular system is more or less formed. Therefore, disturbances of visual functions are weaker in comparison with the strabismic amblyopia.

E. Birch with colleagues investigated the visual acuity in optotypes, vernier, and grating in patients with amblyopia, which developed at different stages of visual maturation: in infancy and preschool age, taking into account the exact onset of the disease and etiology [30]. In case of strabismic amblyopia, which arose both in infancy and preschool age, there was a disproportionately large deficit of visual acuity in optotype and vernier in comparison with grating acuity. When analyzing the data obtained in the infantile group, different patterns of visual deficits were found in patients with strabismic and anisometropic amblyopia. Children with infantile strabismic amblyopia showed a disproportionately high visual acuity deficiency in optotypes and vernier as compared with grating acuity, and children with infantile anisometropic amblyopia showed a proportional impairment of the same functions. The researchers concluded that binocularity is the leading factor of visual deficiency in these patients.

In macaques with experimentally induced anisometropia, the blurred vision of one eye leads to a selective loss of neurons responding to high spatial frequencies [31,32]. On the other hand, infantile strabismus disrupts the binocular connections of the cortical neurons and can lead to the development of a preferential fixation of one eye and some disturbances of monocular functions in the non-dominant eye, where visual acuity is specifically severe in optotypes and vernier [33]. These facts indicate that the presence or absence of binocular function but not of strabismus drives the pattern of visual deficits.

According to E. Birch and colleagues [2], regardless of the etiology of amblyopia, those patients who do not have stereoscopic vision give a disproportionate deficiency of visual acuity in optotypes compared to patients with stereoscopic vision. Visual acuity with amblyopia correlates with binocular status; in the absence of binocular vision in patients, as a rule, there is low visual acuity [34]. The violation of stereoscopic vision also depends on the type of amblyopia and is more often disturbed in the case of strabismic than with anisometropic amblyopia [34]. These research results led to a new approach to the treatment of amblyopia to eliminate suppression in the visual cortex of the brain. Suppression is the strongest inhibiting factor concerning recovery of binocular vision, and its elimination is an indispensable first step in any binocular therapy [35,36].

Suppression in amblyopia.

Suppression plays a significant role in the disturbance of monocular and binocular visual functions in patients with amblyopia [37]. Under suppression, we mean the inhibitory effect of the paired eye on the amblyopic eye when looking with both eyes. Suppression inhibits information from the amblyopic eye to prevent diplopia or confusion. When images of one object fall onto non-corresponding areas of the retina of two eyes in the primary visual cortex, it is projected accordingly into disparate areas and causes diplopia. When different images hit the corresponding retinal points, they are projected into the same area of the cerebral cortex, as a result, which causes confusion. In the 1930-80's suppression was an actual topic raised in many works. The functional scotoma, its size, and location for various types of strabismus were carefully examined.

It is believed that the scotoma involves that part of the visual field of the declined eye that corresponds to the foveal area of the fixing eye. Sometimes the scotoma widens and includes the foveal region of the deflected eye.

Recently, there has been a sharp revival of interest in suppression research, which radically changed the notion of suppression, and offered new and much less dichoptic ways of measuring it [39]. Previously, suppression was determined only qualitatively, it was possible to detect its presence or absence with tests of Worth or the Bogolini, etc.

Visual processing in amblyopia.

To date, methods for quantifying suppression are based on the global visual processing. Anomalous of visual processing can be associated with a violation of the perception of individual elements of the visual scene (local processing), and with a defect in the integration of multiple elements and image parameters in space and time (global processing) [40]. The deficit of the local processing is often associated with a violation in the primary visual cortex V1, where the cells with relatively small receptive fields are located. On the other hand, disturbances in the global processing of visual information are believed to be associated with the involvement of extra-cortical areas. Neurons in these zones have large receptive fields, can integrate signals coming from the lower levels of the visual system, and play a significant role in the segregation of the signal and noise [41]. In the local processing, spatial and temporal processing is singled out.

Psychophysical studies have shown that amblyopia affects many aspects of spatial vision: contrast sensitivity, hyperacuity, crowding-effect [42]. That is, the parvocellular pathway is interested. Unlike local spatial processes, local temporal processing is less altered in amblyopia. Global processing of visual information is considered within the framework of the parallel information processing hypothesis [43-45].

According to this hypothesis, the dorsal extrastriate visual regions sensitive to movement of the V5 / MT zone (middle temporal) are specialized for the perception of the localization and displacement of objects, and, therefore, provides the basis for visual-motor coordination. This pathway of processing of the visual information is called the dorsal stream (the pathway "where" or "vision for action"). It extends from the occipital lobe to the parietal lobe and represents a continuation of the magnocellular pathway. The second pathway, known as the ventral stream (the "what" or "vision for recognition" pathway), includes the ventral areas of the occipital and temporal lobes [46,47]. The connections and structures of this pathway specialize in processing a form that supports object recognition and represents the continuation of the parvocellular pathway.

Recently, convincing evidence has appeared that in addition to anomalous local spatial processing, there is a deficit of global perception of movement, that is, of temporal processing, which does not depend on the local processing of visual information. The evidence for a deficit for the global perception of a form of the object is less convincing than for the movement [48-50].

It is particularly interesting taking into account the well-known hypothesis suggested that local temporal processes are less affected by amblyopia than local spatial processes. It is surprising that the patterns of deficiency of local processing do not correspond to the patterns of a deficit of global processing in monolateral amblyopia. The deficit of global information processing is present only in tasks requiring the isolation of a signal from noise. It turns out that the dorsal extra-striate visual cortex is susceptible to the influence of abnormal developments to a more significant than the ventral region [51].

Recent work has shown that suppression in amblyopia is most pronounced in the dorsal than in the ventral flow [52], so the authors proposed a method for determining suppression in patients with anisometropic and strabismic amblyopia based on the global movement of kinematogram dots.

Conclusion.

1. By now, objective evidence has been accumulated that amblyopia is a binocular pathology. Binocular interaction plays a critical role in the pathogenesis of strabismic, anisometropic and combined amblyopia.

2. Binocular dysfunction can be primary, and a monocular decrease of visual acuity - secondary. Therefore, it is recommended to start the treatment with the restoration of binocular vision, which also can lead to the recovery of the visual acuity of the amblyopic eye.

3. The elimination of suppression should be considered as a first and most necessary step to restore binocular vision. Suppression renders a structurally binocular system functionally monocular.

4. Quantitative evaluation of suppression is the primary step in developing new methods for the treatment of amblyopia, based on the perception of global movement associated with the function of the extra-striate dorsal visual cortex.

5. This new understanding of amblyopia provides the basis for carrying out a large volume of further research and developing more effective therapies that will primarily focus on eliminating suppression and restoring binocular vision.

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