



International Congress on Economics, Management and Business Studies

Hosted Online from New York, USA

Date: 23rd October, 2026

Website: <https://econferencia.com>

STROKE, SEASONALITY, NEW METHODS OF STROKE TREATMENTS, EXTENSION OF THE THERAPEUTIC WINDOW

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ABSTRACT

The article examines stroke, weather sensitivity, the significance of cold stress, new treatment methods, and the possibility of extending the therapeutic window.

Introduction:

Stroke remains one of the most acute and topical medical and biological problems, it ranks second in mortality after coronary heart disease among all nosologies. There is a very high degree of disability and a decrease in the standard of living of people who have had a stroke. Society suffers a huge economic loss. Studies have shown that over the past 30 years, the number of strokes has increased by 70%, and the number of deaths has doubled, in addition, stroke has become much younger " - the incidence rate of young people has increased [1, 2]. Due to the high degree of mortality and disability from stroke, the urgency of the problem, there remains an urgent need to develop new effective methods for diagnosing, preventing, treating and rehabilitating stroke patients.

The **Aim** of this article is to examine the role and significance of the cold factor in the seasonality of stroke incidence, propose new methods for treating stroke, extend the therapeutic window.

Materials and Methods:

Discussion of the role of seasonal factors—specifically cold—in the development of stroke and an assessment of their significance, a review of existing stroke



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treatment methods, an analysis of their advantages and disadvantages, proposals for new treatment approaches of stroke, ways to extend the therapeutic window.

Seasonality of stroke incidence

There is a clear seasonality and fluctuation in the incidence of stroke and mortality depending on the time of year [3].

In cold seasons, in winter, the incidence and mortality from stroke increases significantly. In the region of Kakheti, Georgia, and in particular in the Telavi Regional Hospital, there is also a clear increase in the incidence and mortality from stroke during the cold seasons. Scientists from different countries are studying the pathogenic factors that cause this phenomenon.

Among the seasonal pathogenic factors leading to an increase in morbidity and mortality, cold is especially distinguished. Lowering the temperature even by a few degrees leads to a significant increase in the risk of developing acute cerebrovascular diseases. In addition, a seasonal increase in the level of arterial pressure [4] and such complications of arterial hypertension as the same stroke, as well as myocardial infarction [5] in the cold seasons, are known. It is also well known that the seasonal increase in the incidence of respiratory diseases and mortality from them is associated with a decrease in temperature [6].

Summary:

If the wintertime rise in blood pressure and the incidence of stroke and myocardial infarction are linked to cold-induced vasoconstriction, then the use of vasodilators—specifically those targeting cerebral and cardiac vessels—along with increasing the dosage of antihypertensive medications during cold spells, might reduce the spike in stroke and myocardial infarction rates among patients at high risk for these conditions.



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To note the special significance of cold as a pathogenic factor in increasing the incidence of seasonal diseases and mortality from them, we propose to call it „**seasonal cold stress** ”.

Extension of the therapeutic window

Stroke treatment—basic therapy—until recently relied, and still relies primarily, on infusions, which may be supplemented with antioxidants, neuroprotectors, and antihypoxants. Additionally, anticoagulants and antiplatelet agents are used for ischemic stroke, and hemostatic agents for hemorrhagic stroke.

However, recently there has been a major breakthrough in the treatment of ischemic stroke with the introduction of thrombolysis and thrombectomy to restore perfusion. When these new methods are used during the '**therapeutic window**'—the critical period following a stroke (4.5 hours for thrombolysis [7] and 6 hours for thrombectomy [8] in cases of large artery occlusion)—complete restoration of brain function and the elimination of neurological deficits are possible. If the therapeutic window is incorrectly assessed and the above-mentioned treatments—thrombolysis or thrombectomy—are implemented too late, after the therapeutic window has passed, the treatment will be ineffective and serious complications, including cerebral hemorrhage, are possible.

Recent studies allow thrombolysis to be performed if MRI/CT perfusion data reveal a small infarct core and a large penumbra, within 9 hours after stroke when using alteplase [9] and within 24 hours when using tenecteplase [10]. The therapeutic window for thrombolysis has also been extended to 24 hours [11].

According to large clinical studies, for every minute of delay in thrombolysis, the brain loses 1.9 million neurons. Therefore, the "golden hour" rule has been adopted: the in-hospital time from the moment a patient is admitted to the hospital until the start of thrombolysis should not exceed 60 minutes[12].



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To reduce the time from stroke diagnosis to the start of thrombolysis, some countries—the United States, Germany, and Australia—have already used ambulances[13] equipped with CT scanners, mini-laboratories for rapid testing, thrombolysis equipment, and videoconferencing with a neurologist at the hospital. Thrombolysis begins in the ambulance.

Despite the fact that the latest technologies make it possible to extend the “therapeutic window” to 9–24 hours, thrombolysis continues to be administered in the first 3–4.5 hours, since in most cases primary stroke centers do not have the ability to provide MRI/CT perfusion. Thrombectomy is still primarily performed within the first six hours after stroke diagnosis. Therefore, if the primary and secondary stroke centers are located significantly apart, transporting the patient from the primary center to the secondary stroke center within the first six hours—the “therapeutic window”—is, in most cases, impossible.

At Telavi State University and the Telavi Regional Hospital, we have developed a **Device for cerebrospinal fluid (CSF) oxygenation** that also enables the administration of medications into the CSF and the lowering of cerebrospinal fluid temperature, thereby reducing hypoxia and cerebral edema.

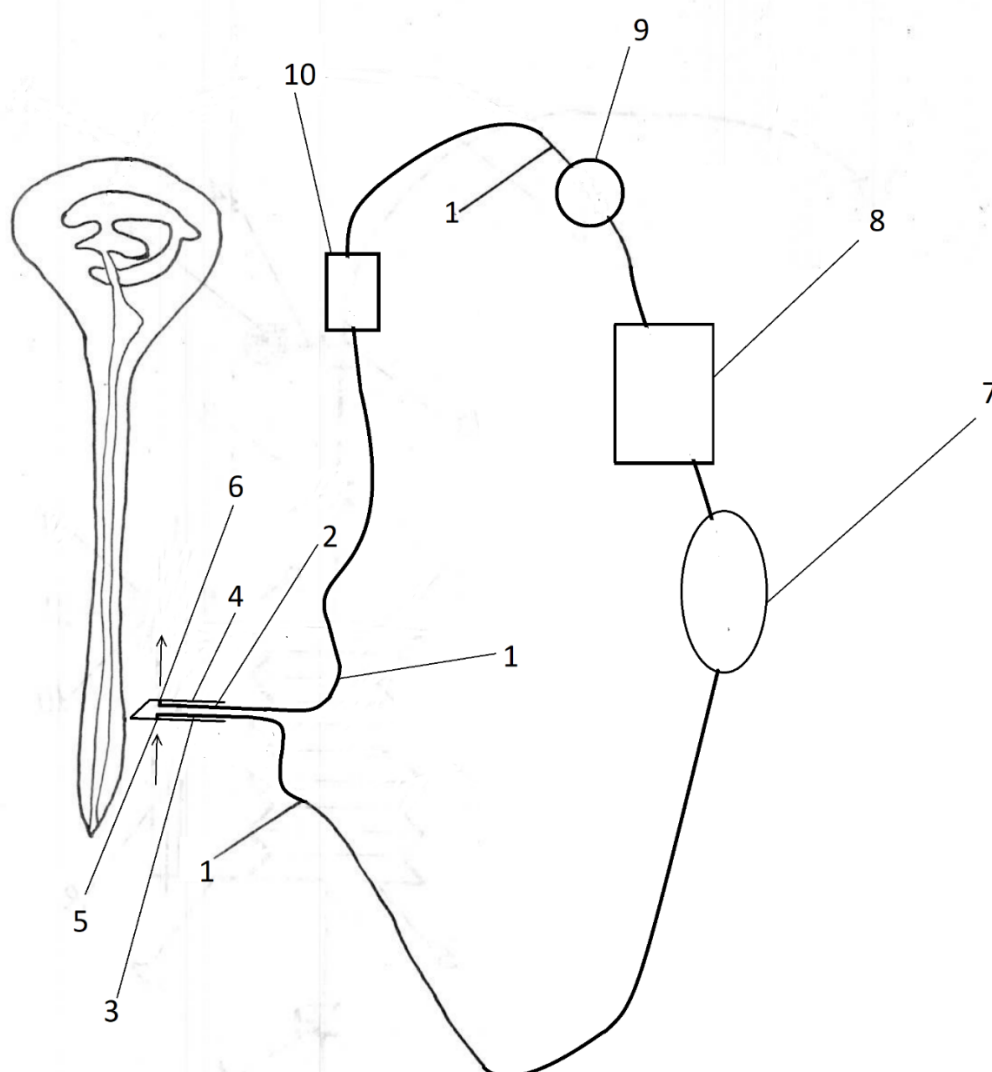


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Device for cerebrospinal fluid oxygenation

1 – flexible tube, 2, 3 – channels of the dual-channel catheter, 4 – dual-channel catheter, 5, 6 – openings of the dual-channel catheter, 7 – drug administration reservoir, 8 – pump, 9 – oxygenator, 10 – heat exchanger



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The device for cerebrospinal fluid oxygenation comprises a flexible tube 1, the ends of which are connected to channels 2 and 3 of a dual-channel catheter 4; one end of tube 1 is connected to one channel 2 of catheter 4, and the other end of the tube is connected to the other channel 3 of catheter 4. The channels open at the other end of the catheter (opposite the connection point of the flexible tubes) via openings 5 and 6 located on opposite sides of the catheter. The flexible tube 1 is connected to a reservoir 7 for drug administration (such as an Ommaya reservoir, though located outside the patient's body), a pump 8, an oxygenator 9, and a heat exchanger 10 (cooler).

Catheter 4 is inserted into the spinal subarachnoid space via lumbar puncture. Pump 8 is then activated; cerebrospinal fluid is withdrawn from the spinal canal through opening 5 of catheter 4 and reintroduced into the spinal canal through opening 6 of catheter 4 (the directions of fluid inflow and outflow are indicated by arrows). Oxygenator 9 makes it possible to increase the oxygen content of the cerebrospinal fluid and improve oxygen supply to neurons, while heat exchanger/cooler 10 allows for lowering the temperature of the cerebrospinal fluid and the brain, thereby reducing nerve cell activity and oxygen demand. Reservoir 7 enables the administration of neuroprotective agents into the cerebrospinal fluid.

Results:

This device can be used if, following a stroke, the time window for thrombolysis has already closed but there is still time to perform a thrombectomy

If there is still time to administer thrombolysis, this device is not used, as performing a lumbar puncture within the preceding 7 days is considered a relative contraindication to thrombolytic therapy; this precaution aims to prevent the



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development of a spinal hematoma and spinal cord compression, which could result in lower-limb paraparesis or paraplegia.

Specialized stroke ambulance teams can be equipped with this device. Upon the team's arrival, if the time window for thrombolysis has expired or is running out, the device's catheter is inserted into the lumbar space, and an intrathecal infusion begins. This process boosts oxygen supply to the brain, administers neuroprotective agents, and induces cerebral hypothermia to reduce the oxygen demand of brain cells. This approach helps salvage a significant portion of the ischemic penumbra and minimize brain cell death (an estimated 1.9 million neurons die every minute during an untreated stroke) prior to thrombectomy; furthermore, it extends the therapeutic window for performing the thrombectomy procedure.

If a specialized stroke team equipped with a CT scanner diagnoses a hemorrhagic stroke, this device can be put into use immediately.

Conclusions:

Given the pressing nature of the issue, research into stroke continues intensively across all fronts.

In our view, modern medicine does not pay sufficient attention to the study of seasonal pathogenic factors, which play a significant role in the fluctuations of morbidity and mortality rates associated with seasonal diseases. It is worth recalling the experience of ancient Chinese medicine, which paid close attention to these factors, identified a number of them, and studied their pathogenic effects. We believe that more intensive study of these factors and their pathogenetic impact would make it possible to at least mitigate the seasonal rise in morbidity and mortality from these diseases.



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The proposed device for cerebrospinal fluid oxygenation in acute ischemic stroke can be used when thrombolysis is not performed, as damage to the vessels of the internal vertebral plexuses—particularly the epidural venous plexuses—could lead to the development of a spinal hematoma and spinal cord compression.

Coating the catheter with bio-glue prior to its insertion into the lumbar space—before thrombolysis—might stabilize the walls of damaged vessels during insertion and prevent bleeding both during and after the procedure.

It should be noted that in some cases where thrombolysis was no longer an option—as the therapeutic window had already closed—administering a combination of cardiomagnyl, clopidogrel, and enoxiparin resulted in the complete resolution of the neurological deficit.

Research should likely be intensified into the development of an artificial oxygen carrier[14] suitable for intrathecal administration without causing complications, as well as into the creation of an artificial cerebrospinal fluid [15] that additionally functions as an oxygen carrier.

There is no doubt that intrathecal perfusion will play a significant role in the future management of serious brain disorders, particularly within the fields of neuro-intensive care and psychiatric intensive care.

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