

Our study is relevant in that it demonstrates that there are significant changes in regional oxygen saturation (NIRS) a few hours before the clinical diagnosis. It also demonstrates differences in oxygenation associated in time with gastric perforation, an infrequent acute pathology, and other and more frequent insidious gastrointestinal diseases, such as necrotising enterocolitis (NEC). A study on NEC found that decreases in regional cerebral oxygen saturation predated the clinical diagnosis by several days.¹⁻³ Nevertheless, in this case of gastric perforation, regional oxygenation changes happened quickly and showed an unexpected increase. We still do not know the reason for this phenomenon. One possible explanation is the difference in the pathophysiology of these 2 intestinal diseases.

To optimise the information yield of NIRS, it is necessary for nurses to report any significant changes reflected in the monitor's display. Thus, continuous training of the nursing and medical staff is of the essence.

In conclusion, NIRS monitoring of abdominal regional oxygen saturation may provide a new approach to the early diagnosis and treatment of gastric and intestinal perforation in the neonatal population.

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Ester Torres-Martínez^{a,b}, Pilar Sáenz-González^{a,b},
Lucía Rodríguez-Caraballo^c, Carsten Driller^c,
Máximo Vento^{a,b}, Álvaro Solaz-García^{b,*}

^a División de Neonatología, Hospital Universitario y Politécnico La Fe, Valencia, Spain

^b Unidad de Investigación Neonatal, Instituto de Investigación Sanitaria La Fe, Valencia, Spain

^c Servicio de Cirugía Pediátrica, Hospital Universitario y Politécnico La Fe, Valencia, Spain

* Corresponding author.

E-mail address: alvarosogar@gmail.com (Á. Solaz-García).

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Use of intravenous prostaglandins in neonatal vasospasm[☆]



Utilidad de las prostaglandinas endovenosas en el espasmo vascular neonatal

Dear Editor:

Arterial puncture and venepuncture are common procedures in neonatal units, but they are not free of risk.^{1,2} The most frequent adverse events are infection, vasospasm and vascular thrombosis, in addition to extravasation. We

present 2 cases of extremity ischaemia secondary to vascular catheterization in extremely preterm infants and the favourable outcomes achieved with intravenous infusion of prostaglandins.

The first case corresponded to a female infant born at 24⁵ weeks of gestation with a birthweight of 640g. At 32 days post birth, following venepuncture for insertion of a vascular access line in the right forearm, the patient developed pallor and coldness in that forearm in absence of a palpable pulse at the site. The initial management consisted in the application of warm gauze in the contralateral arm and topical administration of nitroglycerine 2% ointment. A Doppler ultrasound scan of the affected limb evinced absence of distal blood flow. Since previous measures had proven ineffective, we decided to initiate treatment with continuous infusion of prostaglandin E2 at a dose of 0.014 µg/kg/min and subcutaneous enoxaparin at a dose of 1.5 mg/kg/12 h. In a few hours, there was evidence of improved perfusion with recovery of normal skin

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colour, temperature and the humeral, brachial and radial pulses and normalization of the Doppler waveform of the palmar arch. The dose of prostaglandin was tapered off to its complete discontinuation at 48 h of treatment. Enoxaparin was discontinued after 11 days. The patient did not experience complications of either treatment.

The second case corresponded to a male infant born at 26⁺³ weeks of gestation with a birthweight of 554 g. The mother had a history of deep vein thrombosis with negative results of thrombophilia testing. At 60 days post birth, during an ultrasound-guided catheterization attempt, the right lower extremity appeared pale for a few seconds, with subsequent reperfusion, but followed by posterior swelling of the extremity and an asymmetric femoral pulse relative to the contralateral leg. The extremity was maintained in the extended position and local heat applied to the contralateral extremity, without improvement. A Doppler ultrasound scan evinced blood flow in the common femoral artery, but not in the popliteal and superficial femoral arteries. Continuous infusion of prostaglandin E2 was initiated at a dose of 0.005 mcg/kg/min combined with subcutaneous administration of low-molecular-weight heparin at a dose of 1 mg/kg/12 h. The patient exhibited a favourable clinical and sonographic response that allowed discontinuation of prostaglandin infusion after 3 days of treatment and of heparin after 7 days, and did not experience any adverse events.

Vascular catheterization is performed daily in neonatal care units. Neonates are particularly vulnerable to some of the risks associated with these procedures, such as vasospasm and/or thrombosis, due to the small calibre of their vessels and the immaturity of the coagulation system with a tendency toward hypercoagulation,^{1,3} risks that are particularly increased in infants with lower gestational ages or lower weights. Vasospasm usually develops within hours, or even minutes, of vascular puncture, while thrombosis tends to develop after a few days or weeks.³ Both can cause ischaemia of varying severity in the involved extremity, with manifestations ranging from pallor to gangrene associated with the absence of pulses distal to the lesion. The diagnosis can be confirmed by angiography, which is the gold standard but an invasive procedure, or bedside Doppler ultrasound, a procedure that is not invasive but requires previous training.^{1,3} The initial approach should be to maintain the affected extremity in a horizontal position and warm the contralateral limb to produce a reflex vasodilation effect.¹⁻³ If a catheter has been inserted, it should be removed.^{1,3} Previous studies have described the use of nitroglycerine 0.4% administered topically every 8 h until the resolution of vasoconstriction.² The adverse events associated with this treatment are headache, dizziness, hypotension and methaemoglobinemia. If there is no improvement, and after careful consideration of potential benefits and risks, especially in preterm infants, treatment can continue with intravenous infusion of prostaglandins, which have proven effective for treatment of vasospasm in adult patients.^{4,5} We

extrapolated the dose used in adults (80 µg/day) since there are no data on the use of prostaglandins for treatment of vasospasm in newborn infants. We estimated that neonates would require approximately 1/10 of the dose used in adults (10 µg/day). In the first patient, we used a higher initial dose of 20 µg/day, which we deemed safe based on evidence on the use of prostaglandins to maintain the patency of the ductus arteriosus in patients with cyanotic heart disease, due to concern that a lower dose may not be effective, which was not the case in the second patient. We believe that our experience may be relevant, given the positive outcomes and lack of complications associated with the treatment. In the case of risk of thrombosis secondary to vasospasm and endothelial damage, prophylaxis with low-molecular-weight heparin is recommended for as long as normal blood flow is not restored.⁶ Surgical repair is the treatment of last resort.⁶

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Cristina Juberías Alzueta ^{a,*},
Cristina Durán Fernández-Feijoo ^b, Jorge Vidal Rey ^b,
Noelia Puime Figueroa ^b, Ana Concheiro-Guisán ^b

^a Hospital Universitario Lucus Augusti, Lugo, Spain

^b Hospital Universitario Álvaro Cunqueiro, Vigo, Spain

* Corresponding author.

E-mail address: Cristina.juberias@gmail.com
(C. Juberías Alzueta).

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