

Hypertension and emergency medicine: an update

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ABSTRACT

The objective of this paper is to evaluate the correct management of hypertension in emergency departments. Studies were identified searching PubMed up to April 30, 2012, combining the terms 'HYPERTENSIVE EMERGENCY' and 'HYPERTENSIVE URGENCY'. The search strategy was limited to English and Italian language papers on adult and pediatric patients. Hypertensive crises are commonly found in emergency departments. A range of pharmacological options are available in this setting, but each physician should tailor theoretical principles to the individual patient according to his or her clinical parameters.

Introduction

It is estimated that 2% of patients with hypertension will, during their clinical course, have at least one so-called hypertensive crisis. This is commonly defined as an increase in blood pressure with diastolic values of at least 120-130 mmHg. It would be more correct to speak of hypertensive emergency since high blood pressure is associated with sudden or progressive organ damage, in the absence of which the term hypertensive urgency is preferred.¹

Hypertensive crises can theoretically occur in patients of any age, and can be associated with congenital conditions of renal artery hypoplasia, acute pediatric glomerulopathies, eclampsia, and renal artery atherosclerosis in elderly patients. However, from a general epidemiological viewpoint, incidence is almost the same as that of general hypertension, with peaks over

the last decades of life and in some ethnic groups, e.g. Afro-Americans, with incidence in males double that of females.¹

Hypertensive crises account for approximately 3% of presentations at hospital emergency units (EU). Of all those identified as non-surgical urgency or emergency, 21% is represented by hypertensive urgency and approximately 7% by hypertensive emergency.²

On the other hand, a 2005 report showed that of the 1400 subjects defined as hypertensive and sent to an EU, only 30% had stage 3 hypertension. Indeed, the diagnosis of hypertensive urgency and emergency were made in only 42% and 20% of these patients, respectively.³

In this report, the recommendations for clinical practice have been rationalized on the basis of the current relative guidelines in use (evidence-based medicine levels of evidence - LE and/or degree of recommendation - DR) (Table 1).⁴

Epidemiology

The so-called hypertensive crises can occur *de novo* or can be complications of an underlying essential or secondary arterial hypertension. Such crises can also be attributed to other ongoing diseases with a characteristic increase in blood pressure, such as, for example, renal parenchymal diseases, glomerulonephritis, renovascular pathologies and pheochromocytoma.

It goes without saying that the acute nature of the hypertensive crisis in these conditions suggests that some trigger factors are involved. Therefore, any increase in blood pressure to levels of urgency or emergency could be due to inadequate pharmacological

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therapy administered in cases of chronic hypertension, by interruption of or unsatisfactory adherence to the programmed therapeutic regime (in particular, during treatment with clonidine or β -blockers), by drug abuse or recreational drug taking, by monoamine oxidase inhibitors, or by the failure to correctly diagnose secondary hypertension (LE IIB).¹

In such situations, hypertension can be associated to cerebro-, cardio- and nephro-vascular events, to endocrine dysfunction and pregnancy. In particular, the significant increase in blood pressure that can sometimes develop in the immediate post-operative period, usually 2 h after surgery (often vascular, cardio-thoracic, neurological, or head and neck), is often defined as acute post-operative hypertension and can also require the prolongation of specific pharmacological therapy for up to the next 6 h.

In a retrospective case-controlled study on patients attending EUs throughout Italy, around 140 subjects diagnosed with hypertensive emergency were compared with subjects with arterial hypertension but who did not satisfy the diagnostic criteria of diastolic pressure over 120-130 mmHg. Identified risk factors, or at least predisposing factors, for the hypertensive crises included the inadequate monitoring of systolic blood pressure values during patient care at home, the pre-existence of altered diastolic values, and congestive heart failure ($P < 0.001$, $P < 0.07$ and $P < 0.06$, respectively) (LE IIB).⁵

After correcting for multiple co-variables, statistical analysis in a case control study on around 100 Afro-American and Hispanic patients who attended the EU showed that uncontrolled hypertension is frequently associated with the lack of adequate primary medical care or to the inadequate patient adherence to programmed therapy regimes (absence of primary medical care OR 3.5, 95% CI: 1.6-7.7; lack of compliance OR 1.9, 95% CI: 1.4-2.5). It is, therefore, true that starting specific therapy and the first controls of blood pressure for those subjects who are socially vulnerable or who have little access to free health care more often take place directly in the EU (LE IIA).⁶

Physiopathology

Although not fully understood, the pathogenetic mechanisms underlying hypertensive crises can be considered to be due to a rapid increase in the peripheral vascular resistance and to the action of vasoconstriction mediators, such as neuromediators of the sympathetic nervous system and the renin-angiotensin system). These are in some way unrelated to the self-regulatory mechanisms of tissue perfusion and from the physiological equilibrium between the vasoactive substances released by the vascular wall during stress.⁷

Diagnosis

In spite of the false myths that have developed concerning its diagnosis, chronic hypertension is usually completely asymptomatic. On the contrary, an observational study of around 450 patients attending EUs (LE IIA)² showed that correctly recognized hypertensive urgency may also involve cephalaea (22%), epistaxis (17%), psychomotor agitation (10%), and, as far as hypertensive emergency is concerned, chest pain (17%), dyspnea (22%) and focal neurological deficit (21%).²

There is no constant correlation between epistaxis and hypertensive crisis, and the bleeding from the nose is strictly linked to otorinolaryngoiatric factors rather than to a real underlying hypertensive condition. Sudden onset and highly intense cephalaea would, however, be more frequently related to a subsequent diagnosis of subarachnoid hemorrhage (LE IIB).⁵

As always, the patient's case history plays a fundamental role in the diagnostic procedure. Most patients have already been suffering from high blood pressure for many years before the onset of the crisis and this is why the speed in which the blood pressure rises can be even more significant than the values reached. This still means that the therapy carried out at home and the patient's adherence to it should both be carefully checked, as should the timing of the last dose administered and any possible drug abuse.

Table 1. Levels of evidence and degree of recommendation.

Level of evidence	Scientific substrate	Degree of recommendation
Ia	Systematic revision or meta-analyses of randomized clinical studies	A
Ib	At least one controlled, randomized clinical study	A
IIa	At least one controlled clinical study but not randomized	B
IIb	Other well-conducted controlled studies	B
III	Well-conducted non-controlled studies (case reports, correlation studies, descriptive and retrospective studies)	B
IV	Opinion of experts	C

Routine laboratory tests are an essential part of the diagnostic procedure in the case of hypertensive crisis (creatinine, azotemia and electrolytes), together with myocardial necrosis markers and standard urine tests, with the possible inclusion of toxicology screening.

Another observational study was conducted on 300 middle-aged patients with blood pressure in the emergency range and on significant subsequent mortality data (40% deaths from acute kidney damage, stroke and cardiac pathologies).⁵ The study showed that the total duration of hypertension and azotemia levels at onset of acute damage predicted survival (LE III, DR B).⁵

Finally, together with an electrocardiogram, second level imaging should only be used in cases of reported organ damage.

General principles of treatment

A recent Cochrane systematic revision of over 15 randomized trials confirmed that there is no definitive evidence of the role of specific treatments capable of improving outcome of patients with hypertensive urgency or emergency, although different efficacy in correcting blood pressure was described for the main classes of drugs, such as ACE inhibitors, diuretics, α -adrenergic antagonists, calcium antagonists and nitrates (LE Ia).⁸

In spite of this, the reported achievement of various secondary end points (*e.g.* improvement in renal function or regression of papillary edema, regression of pulmonary edema) only serves to confirm that the lack of evidence is not necessarily evidence of a lack of data. Big strides forward have been made in improving patient and treatment management in this setting, and some key considerations can be made: i) it is harmful to aim at lowering blood pressure too much or too quickly (LE III, DR B). Neurovascular control of the cerebral circulation of the chronically hypertensive and/or elderly patient is no longer sufficient to allow the organism to self-regulate. Also, these patients are, in any case, less tolerant to radical and sudden lowering of blood pressure. Treatment of high peaks of blood pressure must be gradual and carefully managed. This is also why the use of sublingual nifedipine has gradually been abandoned after a series of reports of cerebrovascular ischemia and stroke;⁹ ii) the use of diuretics in every situation is also harmful (LE III, DR B). The anti-hypertensive effect of the diuretics (especially loop diuretics) is to be reserved exclusively for situations of supposed hyporeninemia with excess hydrosaline volume, or, for example, when the clinical profile also includes hypertensive crises and congestive heart failure. In all other conditions, blood pressure can undergo a potentially dangerous *rebound*. Indeed, endovenous infusion of saline solution can rebalance intravascular volume in patients with hyperten-

sive crises thus achieving correct organ perfusion even after inappropriate treatment has been started;¹⁰ iii) first, do no harm. In the absence of reports of organ damage, it is preferable to start anti-hypertensive therapy via oral administration followed by a period of clinical observation more or less prolonged in order to gradually reduce blood pressure over no less than 24-48 h. Once blood pressure values have been corrected, therapy to be taken at home should be re-evaluated and adequate patient follow up should be programmed (LE II, DR B).

On the other hand, subjects with emergency hypertension require access to adequate intensive care units or be admitted to hospital for observation, with continuous monitoring and control of their blood pressure. In these cases, parenteral drug treatment should be administered with indications for a shift towards oral therapy as soon as vital parameters have stabilized, usually after 6-12 h.

In any case, it is a good rule to reduce blood pressure by no more than 10% during the first hour of treatment, and by only another 15% in the next 2-3 h. Blood pressure should always be lowered gradually and greater care should be taken in elderly patients (LE III-IV, DR B-C).

An important exception is the presentation of clinical and/or instrumental signs suggestive of aortic dissection. Here, the seriousness of the clinical condition requires that blood pressure be corrected within 5-10 min, targeting a systolic value of 120 mmHg or under and/or a mean arterial pressure of 80 mmHg or under.¹¹

Hypertensive crises and neurological damage

Among the clinical presentation of hypertensive encephalopathy, an unusual but significant complication of serious hypertension is the onset of often aspecific signs and symptoms, such as intense cephalgia and emesis, besides the progressive change in sensory function from delirium to coma in less than 48 h.

This cannot always be related to the hypertensive emergency and a differential diagnosis with ischemic or hemorrhagic cerebrovascular events should be made.¹²

Treatment of brain damage during an acute increase in blood pressure must also consider the basal physiopathological aspects of intracranial circulation. If on the one hand the cerebral blood flow is strictly linked to the above mentioned self-regulatory mechanisms, on the other hand it depends on the perfusion pressure in the same district, in its turn quantifiable in the difference between mean systemic blood pressure [calculated as diastolic pressure + (systolic pressure - diastolic pressure / 3)] and intracerebral venous pressure (equal to central venous pressure).

When the cerebral perfusion pressure falls below levels at which the system can self-regulate, the resultant

hypoperfusion causes ischemic damage. On the contrary, levels rising above this limit lead to an increase in cerebral blood volume and in intracranial pressure, resulting in vasogenic edema.¹²

During hypertensive encephalopathy, mean arterial pressure usually increases to over 150 mmHg, well over the limit at which efficient self-regulation is possible. From a therapeutic point of view, it is useful to aim for a 20-25% reduction during the first 8 h of treatment with a target of systolic pressure of no more than 160 mmHg and of diastolic pressure of no more than 90-100 mmHg. Any further correction should be managed over the subsequent 24 h. First choice treatment could include endovenous labetalol and nicardipine (LE III, DR B).^{13,14}

Even greater care must be taken during reported ischemic insult because of the risk of worsening the already reduced perfusion of the interested area. In order to reduce the extent of the edema and the risk of hemorrhage, and also to reduce peri-infarct extension, immediate anti-hypertensive treatment is only indicated if systolic pressure is 220 mmHg or over and diastolic pressure is 120 mmHg or over, also in this case without reducing blood pressure by more than 15-25% in the first 24-72 h.

An exception is to be made for those patients admitted to the Stroke Unit who are programmed to undergo fibrinolysis. In these cases, treatment should only be started when systolic values are 185 mmHg or over and diastolic values 110 mmHg and over, aiming to maintain blood pressure at 180/105 mmHg or under for at least the first 24 h after the procedure.^{13,14}

In the case of intracerebral hemorrhage, it should be remembered that there is often a spontaneous reduction in blood pressure during the first 72 h after the event, with obvious consequence in terms of adequately modifying the dosage of any drugs to be given. Treatment should be based on achieving an appropriate balance between risk and benefit.

It is generally accepted that therapy should be started once systolic values reach 180 mmHg or over or a mean 130 mmHg or over. Patients in whom it is reasonably believed better not to consider an increase in intracranial blood pressure (such as normal pupillary reaction, Glasgow Coma Score >8, absence of indirect signs from computerized tomography), the mean arterial pressure can be maintained at 110 mmHg or under (systolic \leq 160 mmHg) for up to the first 24 h after the onset of symptoms.

Otherwise, the values that signal the need for caution are 130 mmHg or under and 180 mmHg or under for the physiopathological reasons given above. These generally require blood pressure to be maintained at a mean of 90 mmHg or over and cerebral perfusion at 70 mmHg or over.

Finally, systolic blood pressure should be maintained at 160 mmHg or under in cases of subarachnoidal hem-

orrhage, at least until an endovascular or surgical approach, when indicated, for the underlying vascular pathology has been considered.¹⁵

In all cases of hypertensive crises and brain damage, correction of any concomitant algic symptoms, such as cephalaea, in itself results in a partial reduction in blood pressure and must be accompanied by a wide range of possible specific therapeutic strategies (Tables 2 and 3).¹⁶

Hypertensive crises and cardiovascular damage

According to the methods reported in Tables 2 and 3, the objectives of treatment of hypertensive crises during acute coronary syndrome, unstable angina and congestive heart failure are reducing blood pressure, maintaining adequate diuresis, and controlling the myocardial oxygen requirements.

Chest pain requires an immediate differential diagnosis between myocardial ischemia and aorta dissection, whether this be ascending (Stanford type A, proximal with risk of extension of the carotid district, stroke and syncope) or not (Stanford type B, distal).

Aortic dissection requires a combination of vasodilators and β -blockers to prevent, at least theoretically, reflex tachycardia which, if not controlled, could aggravate the extent of the damage. The pharmacological options include esmolol and metoprolol on the one hand and nicardipine, sodium nitroprussate or fenoldopam on the other (Tables 2 and 3).¹⁷

Acute post-operative hypertension

Characterized by an acute increase (systolic \geq 190 mmHg, diastolic \geq 100 mmHg) registered at least twice in the 2-3 h after surgery, acute post-operative hypertension is probably due to stimulation of the sympathetic nervous system and, more generally, of the adrenergic system. The resultant main adverse event is surgical-site bleeding.

Literature often reports contrasting data, except in the setting of heart surgery in which start of therapy is recommended for values below those of diagnostic criteria (\geq 140/90 mmHg, or a mean \geq 105 mmHg).

Acute post-operative hypertension is usually self-limiting in the short and medium term (up to 6 h) and is adequately managed by a parenteral approach using esmolol, nicardipine, labetalol, sodium nitroprussate and clevidipine. Clevidipine is a vaso-selective calcium antagonist with an extremely short duration of action (half life <1 min), tested in a cardiosurgical setting through an analysis of three randomized trials, and in the treatment of hypertensive emergency crises in a study of 130 patients admitted to emergency units (LE III).¹⁸

Hypertensive crises and drug abuse

Altogether, it is estimated that around 13% of hypertensive patients who attend emergency units take cocaine. Forty percent of cases present chest pain and signs suggestive of heart failure, besides α -adrenergic neuropsychiatric disturbances.

In spite of the likely coronary vasoconstriction, in itself systemic hypertension and tachycardia only require specific therapy in a small number of cases. More often, alongside phentolamine, nicardipine, diltiazem, verapamil, and nitroglycerin, common benzodiazepine can prove useful. Indeed, in cases of suspected sympathetic overstimulation, use of

Table 2. Main antihypertensive drugs.

Antihypertensive drug	Dosage	Indications	Side effects
Labetalol	20 mg EV, followed by repeated increased doses (20-80 mg) at 10-min intervals, or continuous EV infusion (0.5-2 mg/min); time to onset of effect 5-10 min, duration of effect 2-6 h	Acute post-operative hypertension, aortic dissection, hypertensive crises during acute coronary syndrome or unstable angina, hypertensive encephalopathy, ischemic stroke, stroke due to hemorrhage or subarachnoid hemorrhage, eclampsia and pre-eclampsia	Nausea, vomiting, cardiac arrest, bronchospasm
Esmolol	500 μ g/kg/min for the 1 st min, then 25-50 μ g/kg/min for 4 min (can be increased by 25 μ g/kg/min up to a max. 300 mg/kg/min)	Acute post-operative hypertension, aortic dissection, hypertensive crises during acute coronary syndrome or unstable angina, stroke due to hemorrhage or subarachnoid hemorrhage	Cardiac arrest
Metoprolol	2.5-5 mg EV every 2-5 min up to max. 15 mg in 10-15 min; EV infusion velocity 2-5 mg/h	Aortic dissection	Cardiac arrest, bronchospasm
Sodium nitroprusside	0.3-0.5 μ g/kg/min EV; increased by 0.5 μ g/kg/min up to target (max. dose 10 μ g/kg/min); immediate effect, duration of effect 1-2 min	Acute post-operative hypertension, aortic dissection	Hypotension, vomiting, cyanate toxicity
Enalaprilat*	1.25 mg EV in 5 min every 6 h, increasing by 1.25 mg at intervals of 12-24 h, up to max. 5 mg every 6 h; time to onset of action 15 min, duration of effect 4-6 h	Stroke due to hemorrhage or subarachnoid hemorrhage	Hypotension, acute renal damage, angioedema
Fenoldopam	Initial EV dose 0.1-0.3 μ g/kg/min; adjusted by approx. 0.05-0.1 μ g/kg/min every 15 min (max. dosage 1.6 μ g/kg/min); time to onset of effect 5-10 min, duration of effect 10-15 min	Aortic dissection	Hypotension, cephalaea
Nicardipine*	Initial EV infusion at 5 mg/h, increased by 2.5 mg/h every 5 min, up to max. 15 mg/h; time to onset of effect 5-10 min, duration of effect 2-4 h	Acute post-operative hypertension, aortic dissection, hypertensive encephalopathy, ischemic stroke, stroke due to hemorrhage or subarachnoid hemorrhage, eclampsia and pre-eclampsia, hypertensive crises during drug abuse	Reflex tachycardia, flushing
Clevidipina ^o	Initial dose 1-2 mg/h EV; can be doubled every 90 s until target is reached, then increased more slowly every 5-10 min until max. 16 mg/h	Acute post-operative hypertension	Reflex tachycardia
Idralazine ^o	During pregnancy, usual initial dose 5-10 mg, with further 5-10 mg every 20-30 min if necessary. Time to onset of effect 10 min; duration of effect 2-6 h	Eclampsia and pre-eclampsia	Reflex tachycardia
Fentolamine*	5-15 mg bolus EV, can be repeated within 5 min (max 15 mg). Time to onset of effect 1-2 min; duration of effect 3-5 min	Hypertensive crises during drug abuse, pheochromocytoma	Reflex tachycardia

EV, endovenous administration. *Not currently available in Italy for EV; ^oNot currently available in Italy.

β -blockers is contraindicated because of the possible resultant uncontrolled expression of α -mediated effects.¹⁹

Hypertensive crises and eclampsia

In pre-eclampsia syndrome in which hypertension is associated with edema and proteinuria, and above all in those clearly eclamptic cases with neurological signs, the arterial spasm can be accompanied by hemolytic microangiopathic events with anemia and thrombocytopenia.

Endothelial dysfunction and the changes in activation of the hemocoagulative cascade often complicate the clinical profile. Even though specific drugs in this setting have not yet received complete approval from the US Food and Drug Administration (FDA) and similar agencies worldwide, labetalol, nicardipine or hydralazine are commonly used once systolic values of 155-160 mmHg or over are recorded. The target diastolic value is 90-105 mmHg or under. The recommended approach to prevent convulsions includes also infusion of magnesium sulfate (1-3 g/h with serum tests) together with adequate volume expansion.²⁰

Table 3. Treatment schedules.

Acute post-operative hypertension	Clevidipine* Esmolol Labetalol Nicardipine° Sodium nitroprusside
Aortic dissection	Metoprolol Esmolol Labetalol <i>in association with</i> Nicardipine° Sodium nitroprusside Fenoldopam
Pheochromocytoma	Pre-operative stabilization: i) volume expansion to prevent risk of post-operative hypertension; ii) alpha-adrenergic blockade (phenoxybenzamine* 10 mg OA, 2-4 times a day, for 2 weeks; in alternative metirosine* and prazosin*); iii) α -adrenergic blockade (propranolol 20-40 mg OA, every 6 h, for 3 days). Treatment of pre- and intra-operative hypertensive crises: phentolamine° 2-5 mg EV every 1-2 h (or sodium nitroprusside + β -blocker).
Hypertensive crises during heart failure	Nitroglycerin EV: initial dose 5 μ g/min; increased by 5 μ g/min every 3-5 min, up to 20 μ g/min; in cases of inadequate response. Further increase of 10 μ g/min every 3-5 min; max. 100-200 μ g/min. Furosemide, loop diuretic (dose regulated according to renal function).
Hypertensive crises during acute coronary syndrome or unstable angina	Labetalol Esmolol Nitroglycerin
Hypertensive encephalopathy	Labetalol Nicardipine°
Ischemic stroke	Labetalol Nicardipine°
Stroke due to hemorrhage or subarachnoid hemorrhage	Labetalol Esmolol Nicardipine° Enalaprilat°
Eclampsia and pre-eclampsia	Labetalol Nicardipine° Hydralazine*
Hypertensive crises during drug abuse	Nicardipine° Nitroglycerin Fentolamine° Diltiazem (verapamil) Benzodiazepine

*Not currently available in Italy; °Not currently available in Italy for endovenous administration. OA, oral administration; EV, endovenous administration.

Hypertensive events in children and adolescents

In children and adolescents, arterial hypertension is defined as *severe* when systo-diastolic values are over 5 mmHg of the 99th percentile according to age, gender and height, or on the simple presence of signs of direct or indirect organ damage.

Real emergency, which is fortunately not common, is associated with convulsions, encephalopathy and heart failure. But the condition can also be hidden by abdominal pain, nausea, emesis, intense cephalgia, and an agitated or a lethargic state. In children and adolescents, such conditions are highly suggestive of genesis secondary to hypertensive disease (parenchymal or vascular renal disease, aortic coarctation, pheochromocytoma, neuroblastoma) or associated to specific comorbidities (neurofibromatosis, hyperthyroidism, adrenal diseases, use of steroids, mercury intoxication).

Above all in the presence of neurological symptoms, it is essential that blood pressure is corrected gradually so, once again, the target should be 25% in the first 8-12 h, followed by a further 25% in the next 8-12 h, and a final 50% within 24-48 h; careful and continuous monitoring of vital parameters is mandatory.

Endovenous sodium nitroprusside, although approved and widely used in the past, is not considered first-line therapy. Parenteral administration of nicardipine, above all in cases with renal and cerebral involvement, and of labetalol, the basis of treatment of aortic coarctation, is currently off-label. There is, however, much evidence of efficacy of parenteral administration of nicardipine in the available literature. The reported efficacy is similar to that of hydralazine, although greater caution should be exercised in its use.

Also in pediatric patients, the use of diuretics should be reserved for situations of excessive volume and should never be used alone. Finally, among the drugs for oral administration, useful in cases of urgency in patients with chronic hypertension, nifedipine is commonly reported (only with prolonged release), propranolol and hydralazine.²¹

Conclusions

The so-called hypertensive crises, frequently observed in emergency units throughout Italy, should more correctly be called hypertensive urgencies or emergencies according to the absence or presence of reported organ damage.

Rather than concentrate on the choice of a certain drug or of a particular dosage, emergency unit medical staff should adopt the correct principles of patient management: i) *first, do no harm*; an old adage

that has never been more pertinent given the worrying potential incidence of harmful side-effects experienced by patients admitted to emergency units; ii) remember always to treat the patient and not consider him or her simply as a number. In other words, never concentrate solely on the individual blood pressure values but rather consider the whole clinical profile in all its complexity; iii) arterial hypertension should never be seen as an isolated episode, but should be considered within the context of a cardiovascular pathology that must be managed in the long term through the co-operation between specialists and the family doctor; iv) drugs with a short duration of action and rapid response should only be used in selected and life-threatening cases; v) endovenous drug administration requires careful management in a suitable intensive therapy unit or in structures in which the patient can be appropriately monitored according to the intensity of treatment by adequately trained medical and nursing staff.²²

As we can see, scientific evidence cannot replace the experience to be gained from continuous clinical practice. Physicians and nursing staff must adapt the general theoretical principles to the individual patient and his or her clinical case history, rather than to pharmacokinetic and pharmacodynamic data of the molecule used.

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