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WEB SIMULATORS IN MEDICAL EDUCATION

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Abstract

Web simulators are electronic textbooks that run in any web browser and need nothing installed. They join explanation with a mathematical model: the student changes parameters with sliders, follows time courses and readings, and walks through a guided scenario that leads from health to a disorder and to its compensation. We present a set of simulators built on models of physiological systems: blood circulation with eight scenarios, from normal circulation through ventricular failure, valve defects and haemorrhage to intra-abdominal pressure and anti-shock trousers; respiration; the excitation threshold of a nerve membrane; iron storage; whole-body iron metabolism and its regulation by hepcidin; and an introductory model of a heated room. The circulation model deliberately has no regulation; the student switches it on step by step and sees what the body needs it for. Every number in a scenario text is measured on the model and verified by walking through the page.

Keywords

web simulators, medical education, pathophysiology, blood circulation, serious games

1 Introduction: the simulator as an electronic textbook

Pathophysiology explains why a disease looks the way it does. A textbook says so in words and pictures, but the relations involved are dynamic: when the left ventricle fails, blood accumulates upstream of it, pressure in the pulmonary veins rises and the ventricle dilates – and by how much depends on how stiff the ventricle is, how much blood there is in the body and what the reflexes do about it. A static picture captures one moment of one case. A simulator lets the student try out the same relations: change the force of contraction, let the patient bleed, switch on a reflex – and see what happens.

Our teaching simulators have rested on this idea – schola ludus, school as play – since the 1990s [1.]. In our paper at MEDSOFT 2024 we described how they became "explanatory models" that make it possible to understand the causes of clinical signs in the context of physiological regulation [2.], and earlier generations of the simulators still run on the *physiome.cz* and egolem.online websites. This paper shows a new set of simulators, built anew with the technology described in the first paper of these proceedings [3.]. The subject here is not the technology but the content: what the student does with the simulators and what they can learn from them.

Because each simulator can be downloaded into the browser as a single file, which then works in the browser on its own, without a permanent connection to a server, it can be used in several ways. Simulators can become part of a seminar or a lecture in which students carry out virtual experiments with the model under the guidance of a teacher. They can also be included as extensions to printed textbooks – a QR code in the textbook opens the interactive simulation application in the browser. And they can be part of a larger multimedia teaching narrative.

2 How to work with a simulator

All simulators of the set have the same controls (Fig. 1). At the top, the experiment and the language are chosen. Below the description of the experiment are buttons that start, pause or restart the simulation, and the simulation time. Sliders change the inputs of the model – for example heart rate, peripheral resistance or left ventricular contractility; the value can also be typed into the box beside the slider, and once a slider has moved, it offers a way back to the default value. While a slider is being dragged the simulation waits, and when it is released it continues with the new value.

Results are shown in three kinds of display. Time courses are drawn with a history window, as on a bedside monitor: the chart shows the last few seconds and scrolls with time. Charts of one value against another show, for example, the left ventricular pressure–volume loop with the ESPVR and EDPVR lines, which mark the limits within which the loop can move. And readout tiles show numbers as a monitor shows them – arm pressure, cardiac output, ventricular volume.

Most of the student's work is guided by a scenario. Each step has a text that says what to set and what to notice, and a button that sets the values of the step. Some steps start again, like a new patient: the valve defects can thus be compared with one another, each starting from a healthy heart. Other steps continue where the previous one ended, because the story goes on – a patient who is bleeding does not start again when a reflex is switched on, but is still the same patient, only half a litre of blood poorer.

Every simulator is in Czech and in English, in one application, for the Czech and the English-language study programmes. Both versions have the same numbers and the same scenarios; only the words and the decimal sign differ. The simulators work on a computer, a tablet and a mobile phone.

3 Blood circulation

The circulation model is based on the pulsatile model of the heart and vascular bed published by van Meurs [4.]. It contains both atria and both ventricles with time-varying elastance, the heart valves, the pulmonary circulation, and the systemic arteries and veins; the pressure measured at the arm is computed separately from aortic pressure. The baseline is normal circulation: heart rate 72/min, arm pressure 125/78 mmHg, aortic pressure 110/81 mmHg, stroke volume 71 ml and cardiac output 5.1 l/min. There are eight scenarios on this model.

The model deliberately has no regulation – no baroreflex, no sympathetic system, no renal regulation of blood volume. This is a didactic intention, not a shortcoming: the student sees what a disorder does by itself, then switches on the compensations himself, one after another, and sees what each of them can achieve. Where the scenario needs a response of the organism, it tells the student which slider to move and why.

3.1 Normal circulation

The first scenario introduces the model and how to read it. The student notices that arm pressure (125/78 mmHg) differs from aortic pressure (110/81 mmHg) – the pulse is amplified on its way to the periphery – and then tries tachycardia, increased peripheral resistance and positive intrathoracic pressure. On the pressure–volume loop he sees how each change is reflected in the filling and emptying of the ventricle.

3.2 Ventricular failure, systolic and diastolic failure

Three scenarios deal with heart failure. In the first, the student halves left ventricular contractility: arm pressure falls only slightly, to 117/73 mmHg, but the ventricle dilates – its volume stays between 72 and 139 ml instead of 47 and 119 ml – and

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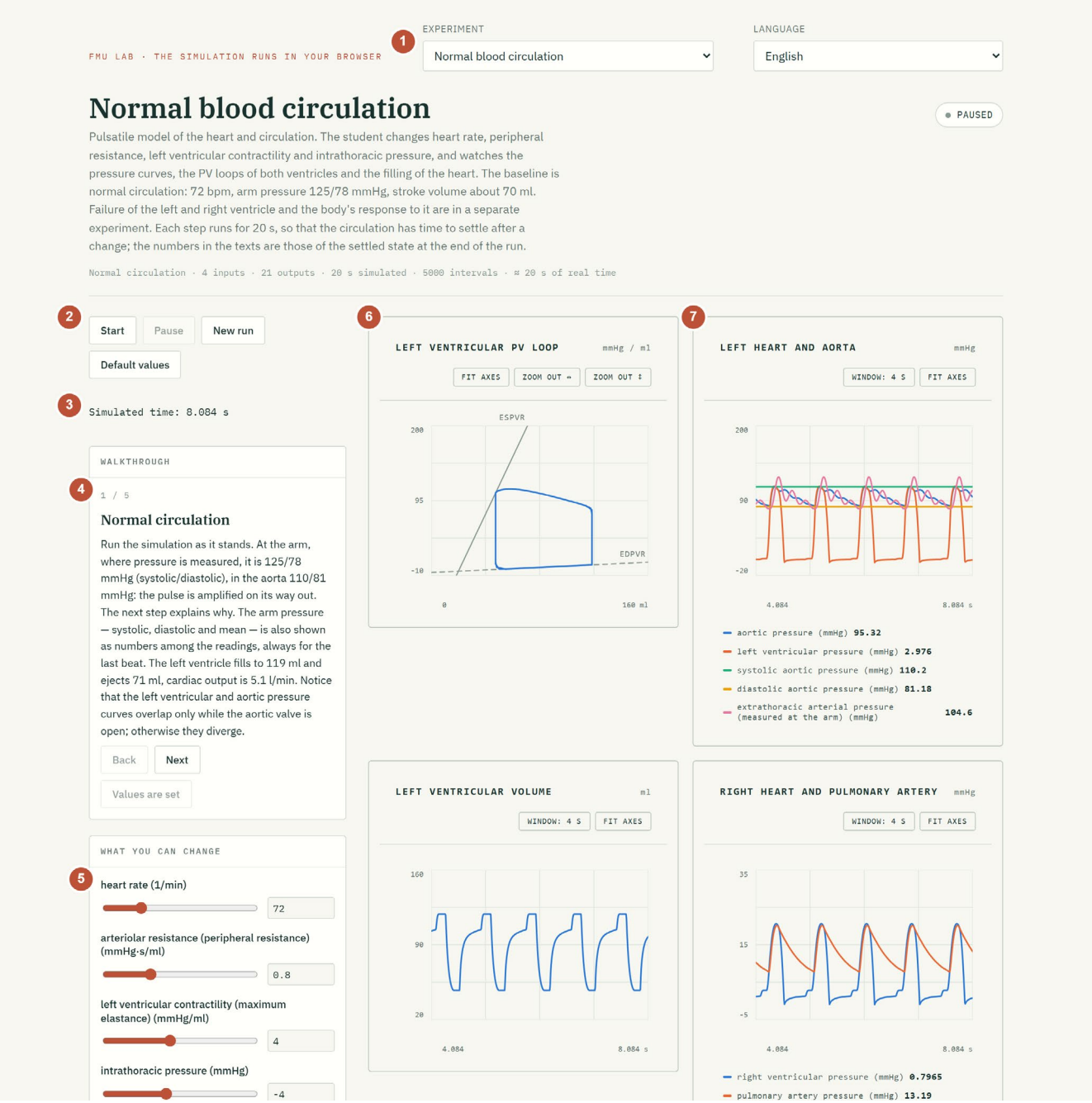


Figure 1 – the normal circulation simulator. 1 – choice of experiment (beside it, choice of language); 2 – run controls: start, pause, new run and return to the default values; 3 – simulation time; 4 – a step of the guided scenario with the text for the student and the Back, Next and Set values buttons; 5 – sliders with a number box; 6 – the left ventricular pressure–volume loop with the ESPVR and EDPVR lines; 7 – pressure time courses with a history window and readouts.

the blood that the weaker ventricle does not eject accumulates upstream of it, in the pulmonary veins. The student then simulates the first response of the organism, constriction of the pulmonary veins: blood that was merely lying in them moves to where it exerts pressure, the ventricle fills more and, by the Frank–Starling law, ejects more. Cardiac output rises back to 5.0 l/min, at the cost of higher pulmonary pressure. The same scenario then shows diastolic failure of a stiff ventricle and right ventricular failure.

The systolic failure scenario goes further, from acute infarction to compensated chronic failure. After the infarction, the ejection fraction falls to 34 % and cardiac output to 4.2 l/min; over the following weeks the ventricle dilates (remodelling), and then the student switches on the compensations: the sympathetic system speeds up the heart and constricts the arterioles – pressure rises, but output does not –, constricts the veins, and the kidneys increase blood volume. The result is a cardiac output of 5.3 l/min at an ejection fraction of 22 % and nearly threefold filling pressures: a normal output paid for with congestion. The diastolic failure scenario shows the opposite case, a ventricle that contracts well but fills poorly – heart failure with preserved ejection fraction.

3.3 Valve defects

Each of the four defects of the left heart draws a different figure on the pressure–volume loop (Fig. 2). In mitral stenosis the ventricle does not manage to fill through the narrowed orifice, the loop shrinks, stroke volume falls from 71 to 36 ml, and blood accumulates in the pulmonary circulation – pulmonary artery pressure rises from 20/8 to 28/21 mmHg. In mitral regurgitation the ventricle fills up to 277 ml and "ejects" 250 ml, but only 32

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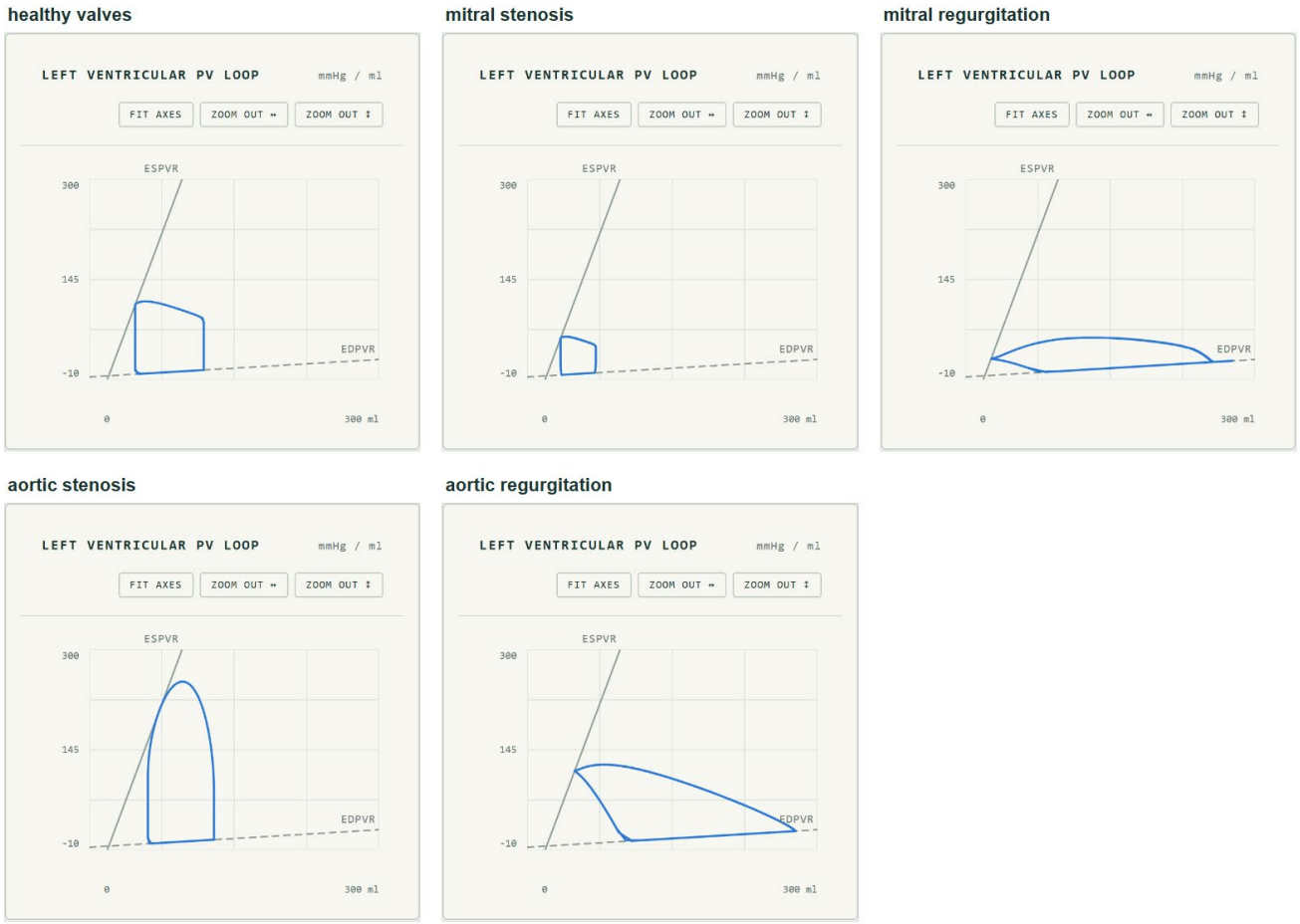


Figure 2 –The left ventricular pressure–volume loop with healthy valves and in four valve defects, each starting from a healthy heart, on the same axes. Mitral stenosis: the ventricle does not manage to fill and the loop shrinks. Mitral regurgitation: the ventricle fills up to 277 ml, but isovolumic contraction is missing because the mitral valve does not close. Aortic stenosis: ventricular pressure climbs to 250 mmHg. Aortic regurgitation: the ventricle fills from both sides and isovolumic relaxation is missing. The ESPVR and EDPVR lines stay in place, because neither the contractility nor the stiffness of the ventricle changes.

ml of it go into the aorta; isovolumic contraction disappears from the loop because the mitral valve does not close. In aortic stenosis, ventricular pressure climbs to 250 mmHg while aortic pressure is only 104 mmHg – and arm pressure is almost normal, 115/76 mmHg: the cuff does not betray the stenosis. Aortic regurgitation, by contrast, gives an arm pressure of 170/10 mmHg, a systolic pressure 50 mmHg higher than in the aorta, which is the amplification of the pulse in the periphery typical of this defect. Three defects – both mitral ones and aortic regurgitation – the student also tries with tachycardia and sees, for example, why a fast heart rate is dangerous for a patient with mitral stenosis.

3.4 Haemorrhage and what regulation is for

The haemorrhage scenario is the clearest demonstration of what regulation is for (Fig. 3). The patient loses half a litre of blood in 15 s, a tenth of his volume. Without reflexes the circulation settles at a pressure of 92/57 mmHg and a cardiac output of 3.8 l/min. Then the student switches on the reflexes one by one. A faster heart rate and arteriolar constriction raise pressure to 102/72 mmHg, but cardiac output stays at 4.0 l/min – and the scenario asks why: what would make the ventricle fill more? The answer comes with the next step: the sympathetic system constricts the veins too, 300 ml of blood that were merely lying in them return to the circulation, and with a stronger contraction the pressure is 128/90 mmHg and the output 4.9 l/min. The loss of a tenth of the blood is almost fully compensated – and arm pressure no longer betrays it. The scenario then continues with a further haemorrhage, for which the reflexes are not enough, with an infusion after which the reflexes raise pressure too much, and finally with a return to the baseline.

At the end of the scenario the student reads that a more advanced model will have its own regulation – and that it will do exactly what he did by hand: watch the pressure and change heart rate, arterioles, veins and contraction according to it, in both directions.

3.5 Prolonged AV conduction, intra-abdominal pressure and anti-shock trousers

Two further scenarios widen the view. The first lets the student prolong atrioventricular conduction, separately for the left and the right heart, and watch how the atrial contribution to ventricular filling is lost. The second shows what pressure from outside does to the circulation. At an intra-abdominal pressure of 20 mmHg cardiac output falls by a quarter, and yet central venous pressure rises to 10 mmHg. The scenario explains why: half of the intra-abdominal pressure passes through the diaphragm into the chest and presses on the veins from outside, so central venous pressure here shows intrathoracic pressure, not the filling of the heart. In the second part the patient loses a litre of blood and arm pressure falls to 52/32 mmHg; anti-shock trousers inflated to 20 mmHg push 246 ml of blood from the veins of the legs and abdomen to the heart, and pressure rises to 81/50 mmHg although there is no more blood in the body. The same device in a person who is not short of blood raises pressure to 175/110 mmHg.

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Figure 3 –the haemorrhage scenario from start to finish (165 s of simulation, eleven steps): arm pressure (systolic, diastolic and mean), cardiac output and total blood volume. Blood volume falls with two haemorrhages and returns with an infusion; pressure and cardiac output show what happens to the circulation without reflexes and what switching on the reflexes one by one does – a faster heart rate, constriction of the arterioles, constriction of the veins and a stronger contraction.

4 Respiration

The simple respiration model shows how the alveolar pressures of oxygen and carbon dioxide depend on ventilation, metabolism, cardiac output and pulmonary shunt (Fig. 4). The student lowers ventilation and sees how carbon dioxide can no longer leave fast enough and accumulates in the alveoli; he raises it and finds that the changes are not symmetrical – oxygen rises less than carbon dioxide falls.

The model of the alveolar-capillary unit with tissue goes a step further, to saturation and pH. Both models compute the oxygen and carbon dioxide content of the blood, saturation and pH according to Siggaard-Andersen's oxygen status algorithm [5.]. With hypoventilation to 2500 ml/min, PAO2 falls to 67.6 mmHg and PACO2 jumps to 64.0 mmHg, pH to 7.26 – respiratory acidosis. Saturation, however, falls only to 90.2 %, because the haemoglobin dissociation curve is still flat in this range. With hyperventilation, saturation rises only from 98.0 to 98.8 %: the added oxygen has nothing to bind to. Raising metabolism at the same ventilation gives almost the same picture as hypoventilation – what matters is ventilation relative to metabolism, not ventilation itself. The last step of the scenario honestly shows where the model does not reach: a pulmonary shunt does not show in the alveolar values, because it is admixed only beyond the capillary.



Figure 4 – the "cockpit" of the simple respiration model – a designed page in which the scenario step, charts of alveolar partial pressures, ventilation and the respiratory quotient, sliders and readouts are arranged as the teacher intended. The resting state is shown: ventilation 4860 ml/min, alveolar PO2 106.6 mmHg and PCO2 37.81 mmHg.

5 Further examples

Excitation threshold. The Hodgkin–Huxley model of the nerve membrane [6.] has a single slider: the amplitude of a short stimulus. At 18 mV the membrane only bends to –69.7 mV and returns. One millivolt more, at 19 mV, fires an action potential to +31.1 mV – 101 mV elsewhere (Fig. 5). On the gate chart the student sees why: the sodium gates open, sodium rushes in, and then the inactivation gate closes by itself and potassium pulls the membrane back, even below its resting value. A stronger stimulus no longer makes the response larger – that is the all-or-none law.

The model of ferritin and the labile iron pool shows how a cell keeps free iron at a low level. As long as the ferritin cages have room, they take up added iron and free iron hardly increases. Once they are full, free iron jumps forty-seven-fold – and that is the beginning of its toxic effect. More cages then render the same iron harmless again: toxicity is decided not by how much iron the cell contains, but by whether it has somewhere to go. The model is based on the work of Masison and Mendes [7., 8.].

Iron metabolism. The model of systemic iron metabolism in the mouse after Enculescu et al. [9.] follows iron in seven compartments – serum, liver, spleen, duodenum, bone marrow, red blood cells and other organs – and its regulation by the hormone hepcidin, which blocks ferroportin, the only way by which iron leaves a cell. The simulator is drawn as a scheme in which live values are shown directly at the compartments and at every ferroportin "door" (Fig. 6). The student can set hepcidin by hand, knock out the hepcidin gene, give lipopolysaccharide or intravenous iron, set a transfusion or a haemorrhage, and hold

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Figure 5 – the Hodgkin–Huxley model of the nerve membrane: the response to a short stimulus with an amplitude of 18 mV (left) and 19 mV (right). At 18 mV the membrane only bends and returns to the resting potential; a stimulus just one millivolt stronger elicits an action potential. In the right chart the run is longer and also captures the second, regularly repeated stimulus.

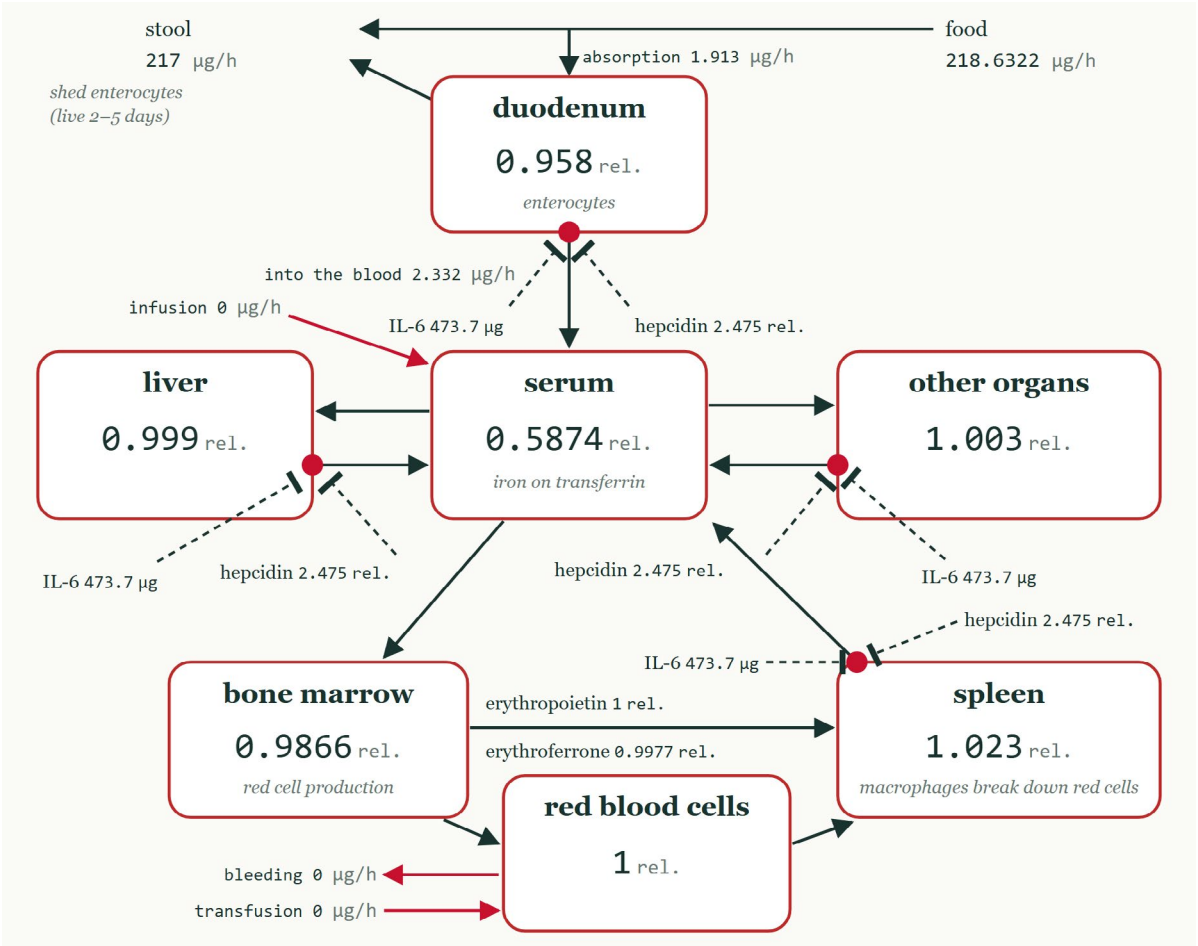


Figure 6 – the scheme of iron metabolism – a designed page of the simulator, drawn after a sketch by the model's author. Seven compartments (serum, liver, spleen, duodenum, bone marrow, red blood cells and other organs) with the live amount of iron in relative units (1 = normal steady state), the iron flows between them, the red ferroportin dots and the dashed blocking lines of hepcidin and interleukin 6; in red, what the student adds or removes (infusion, haemorrhage, transfusion). The state shown is about three hours after 1 µg of lipopolysaccharide: interleukin 6 is close to its peak, hepcidin has risen 2.5-fold and serum iron has fallen to 0.59 of normal. The values are computed by the model (Enculescu et al. 2017, converted to SI units); interleukin 6 is given in model units, not as a physiological concentration.

the serum iron level fixed. Lipopolysaccharide causes inflammation: interleukin 6 peaks at two and a half hours, hepcidin more than doubles and serum iron falls to about 60 % within three hours – the beginning of the anaemia of inflammation. If the student knocks out the hepcidin gene, iron starts to accumulate in the liver – after five days there is three times as much as normal – while the macrophages of the spleen lose iron: the picture of hereditary haemochromatosis. So that excess iron does not drain into erythropoiesis, we added to the model erythropoietin, according to which the bone marrow takes iron only as red cell production needs it, and erythroferrone, with which the bone marrow suppresses hepcidin during haemorrhage and releases iron from the stores for itself. Both regulations can be switched off and compared with the original model. And when the student holds both serum iron and hepcidin fixed, every organ receives a constant inflow of iron, and the student sees how the cells regulate its amount themselves: more iron in the cell means more ferroportin and a larger outflow.

Room heating. An introductory model outside physiology serves as a first acquaintance with dynamics: the room warms up towards a steady temperature with a time constant of 15 minutes. The student finds that insulation decides where the temperature ends up, whereas thermal mass decides only how long it takes to get there. It is the same reasoning that he later needs for compartments in the body.

Qualitative models. The set also contains several models that compute states rather than numbers – for example washing a car or brewing coffee, generated from diagrams of the Object-Process Methodology (OPM) [10.]. Instead of charts they have lamps that light up according to the state the system is in, and buttons that trigger an event. They are examples of the OPM method, which will be the subject of a separate paper, and in this paper we only mention them.

6 How a scenario comes into being

The scenario is written by the teacher. The numbers in it, however, are not estimates: they are readings from the running model, rounded as the page shows them. And because both the scenario and the model may change, every scenario is verified by walking through the page – an automatic tool goes through all the steps as a student would and compares each number in the text with what the page shows at that step.

Such a check finds errors that a student would experience as incomprehensibility. It repeatedly found steps that should have started again, like a new patient, but continued from the end of the previous step – and therefore showed different numbers from those the text promised: in the valve defects, in room heating and in the model from an OPM diagram they were revealed by the walk through the page, in the alveolar-capillary unit by the tool that compares the numbers in all scenarios at once. In the alveolar-capillary unit model, for example, the text gave a PACO2 of 27.3 mmHg in hyperventilation, whereas the page showed 28.7 mmHg, because hyperventilation started from hypoventilation instead of from rest. A student cannot explain such a difference; the tool finds it in a few minutes.

The same holds for the model itself. The circulation model showed zero cardiac output in the first second of a run, because the output monitor first needed one whole beat to measure it. On the chart this looked like cardiac arrest at the start of every run. Today the monitor shows the default value until the first beat, and the model no longer has a small flaw that would confuse the student.

7 Links to the running simulators

All the simulators are freely available and open directly in the browser. Each link leads to a particular experiment and language; replacing lang=en with lang=cs in a link opens the Czech version. **An overview of all simulators with links is at** *https://bodylight.physiome.cz/SvelteBodylight.js/en.html***.**

- Normal blood circulation: *https://bodylight.physiome.cz/SvelteBodylight.js/medsoft2026/fmu-lab.html?experiment=normal-circulation/student-basic&lang=en*
- Left and right ventricular failure: *https://bodylight.physiome.cz/SvelteBodylight.js/medsoft2026/fmu-lab.html?experiment=normal-circulation/selhani-komor&lang=en*
- Systolic heart failure: *https://bodylight.physiome.cz/SvelteBodylight.js/medsoft2026/fmu-lab.html?experiment=normal-circulation/systolicke-selhani&lang=en*
- Diastolic heart failure: *https://bodylight.physiome.cz/SvelteBodylight.js/medsoft2026/fmu-lab.html?experiment=normal-circulation/diastolicke-selhani&lang=en*
- Left-heart valve defects: *https://bodylight.physiome.cz/SvelteBodylight.js/medsoft2026/fmu-lab.html?experiment=normal-circulation/chlopenni-vady&lang=en*
- Haemorrhage and what regulation is for: *https://bodylight.physiome.cz/SvelteBodylight.js/medsoft2026/fmu-lab.html?experiment=normal-circulation/krvaceni&lang=en*
- Prolonged AV conduction: *https://bodylight.physiome.cz/SvelteBodylight.js/medsoft2026/fmu-lab.html?experiment=normal-circulation/av-blokady&lang=en*
- Intra-abdominal pressure and the anti-shock suit: *https://bodylight.physiome.cz/SvelteBodylight.js/medsoft2026/fmu-lab.html?experiment=normal-circulation/nitrobrisni-tlak-mast&lang=en*
- Simple respiration model: *https://bodylight.physiome.cz/SvelteBodylight.js/medsoft2026/fmu-lab.html?experiment=simple-respiration/student-basic&lang=en*
- Alveolar-capillary unit: *https://bodylight.physiome.cz/SvelteBodylight.js/medsoft2026/fmu-lab.html?experiment=alveolar-capillary-tissue/student-basic&lang=en*
- Excitation threshold: *https://bodylight.physiome.cz/SvelteBodylight.js/medsoft2026/fmu-lab.html?experiment=hodgkin-huxley/prah&lang=en*
- Iron store stabilisation: *https://bodylight.physiome.cz/SvelteBodylight.js/medsoft2026/fmu-lab.html?experiment=ferritin/ustaleni&lang=en*
- Iron metabolism: *https://bodylight.physiome.cz/SvelteBodylight.js/medsoft2026/fmu-lab.html?experiment=iron-metabolism/hepcidin&lang=en*
- Room heating: *https://bodylight.physiome.cz/SvelteBodylight.js/medsoft2026/fmu-lab.html?experiment=room-heating/vytapeni&lang=en*

8 Conclusion

Web simulators do not replace explanation or the textbook; they give the student a chance to try out what the explanation claims. On the circulation model without regulation, the student sees what a disorder does by itself, and when he switches the regulation on himself, he understands what the body needs it for. A guided scenario leads him step by step, and the numbers he reads in the text he also sees on the screen. On the iron metabolism model the student can try out the regulatory effect of hepcidin and erythroferrone and the response to malabsorption, inflammation or blood loss.

We have been using teaching web simulators in pathophysiology classes for several years, and the results show that web simulation games, as a complement to seminars and lectures, contribute to a better understanding of complex causal relationships than static text or a frontal lecture [2.]. After all, more than two thousand years ago a Chinese saying attributed to Confucius put it this way: "I hear and I forget, I see and I remember, I do and I understand." Our teaching experience with web simulators confirms this old Chinese wisdom. Our new technology, Svelte Bodylight, makes building web simulators easier.

It is also worth saying where the limits of these simulators lie. The models describe individual physiological systems, not the whole body: the circulation has no regulation, respiration has no circulation, and neither has kidneys. Linking the systems into an integrated model, such as the Golem model [11.], is the next step. The technology on which the simulators rest is described in the first paper of these proceedings [3.].

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WEBOVÉ SIMULÁTORY PŘI VÝUCE MEDICÍNY

Abstrakt

Webové simulátory jsou elektronické učebnice, které běží v libovolném internetovém prohlížeči a ke kterým student nepotřebuje nic instalovat. Spojují výklad s matematickým modelem: student mění parametry pomocí posuvníků, sleduje průběhy a odečty a prochází řízený scénář, který ho vede od zdravého stavu k poruše a k její kompenzaci. Ukazujeme sadu simulátorů nad modely fyziologických systémů: krevní oběh s osmi scénáři od normálního oběhu přes selhání komor, chlopenní vady a krvácení až po nitrobřišní tlak a protišokové kalhoty, dýchání, práh vzruchu nervové membrány, zásobu železa, metabolismus železa v celém těle s jeho regulací hepcidinem a úvodní model vytápění místnosti. Model krevního oběhu záměrně nemá regulace; student je zapíná sám a vidí, k čemu tělo regulace potřebuje. Každé číslo v textu scénáře je změřené na modelu a ověřené průchodem stránkou.

Klíčová slova

webové simulátory, lékařská výuka, patofyziologie, krevní oběh, simulační hry

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