CHAPTER 169

Nursing Strategies to Prevent Coagulation of the Extracorporeal Circuit

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OBJECTIVES

This chapter will:

1. Emphasize that clotting in the continuous renal replacement therapy circuit is complex and affected by many factors in the critically ill patient.
2. Explain that anticoagulant agents can prevent or delay clotting but are not the only preventive strategy necessary.
3. Indicate that clotting occurs in either the membrane or the venous air trap chamber and propose how this clot may form.
4. Discuss the need to prevent clotting by ensuring the extracorporeal circuit is not mechanically obstructed and blood flow is maintained.
5. Describe the influence of the access catheter and site, membrane and venous chamber, predilution fluid administration, blood flow setting, and nursing competence on circuit clotting.

The mechanism for blood coagulation in the renal replacement therapy (RRT) extracorporeal circuit (EC) supporting a critically ill patient is complex. Acute renal failure, inflammation with critical illness, and other factors influence the normal roles of key mediators such as thrombin, tissue factor, platelets, and endothelium. A precise explanation of how clotting occurs, or why it does not occur, when blood is being pumped outside the body via plastic tubes is not easily achieved.[1–3] This also may explain why a wide variation in response to anticoagulation strategies to prevent clotting in the EC can be observed, with time before clotting ranging from only hours to several days with and without anticoagulation, and despite improvements in RRT technology and clinician expertise over the past 20 years.[2,4]

However, clotting in the EC is inevitable, because this nonbiologic environment places blood under stress, with coagulation occurring at places of high resistance, stasis, and positive or negative pressures with shearing forces. In this respect, the blood is behaving normally. During flow through the EC, cells and plasma separate, cellular aggregation occurs, proteins deposit or build up, and an inflammatory response occurs. This may explain why thrombin generation is increased during continuous hemofiltration by activation of the tissue factor pathway,[5] with this being only one of the many responses causing coagulation. Anticoagulants can be successful in delaying clot formation and make an RRT procedure functional for long periods of time without clotting. However, components of the EC and other influences on the EC are important as adjuncts to the use of anticoagulants and are very important when anticoagulants are not used.

CLOTTING: WHERE AND HOW?

The two components of the EC where clotting occurs are the hemofilter (membrane) and the venous bubble trap chamber.[1,6] Coagulation is not common along the circuit tubing or in the blood pump tubing segment. Additional chambers in the EC for fluid addition, or where smaller tubing enters the main blood path at a T junction, also may be sites of clot formation. However, clot development in these components is not a cause of circuit failure unless the clot formed at these junctions breaks off and embolizes into the main circuit path, obstructing flow into the membrane. This type of EC embolus clotting can obstruct blood flow at entry of blood to the membrane, or at exit of the venous chamber, causing an abrupt cessation to treatment by total blood flow obstruction, with an inability to return the EC blood to the patient. Fig. 169.1 shows a clot obstructing blood flow into a membrane. The picture depicts a membrane autopsy. Immediately after clotting, the membrane is cut in half to reveal the site of the clot obstructing the membrane at blood entry rather than by clotting along the fibers of the membrane, which is a slower progressive process. Fig. 169.2 shows a venous chamber with a large clot formed in the chamber, which can break off from the top section of the chamber and move down to the outlet, thereby causing an acute obstruction.

It is assumed that circuit life data reflect the time before membrane fiber clotting; however, the membrane entry or the venous chamber exit may clot before, and independent of, the membrane fibers. Not only do these events constitute a failure of the circuit but also, in the case of the venous chamber, unless the venous tubing segment can be changed quickly, and this may not be possible in many circuits, blood flow cannot continue, and the circuit volume of blood may not be returned (discarded) to the patient despite the membrane(s) itself being unaffected.[7,8]

Therefore strategies to prevent clot formation in the EC are aimed at these two components: the membrane and the venous bubble trap. For a more complete understanding of this problem when using continuous RRT (CRRT), this discussion must include all of the EC components and their contribution to circuit clotting at these two sites.

ANTICOAGULANTS

Anticoagulant drugs prevent or delay formation of clot in the EC, and many methods are used with variability in the quality of evidence supporting their success and safety.[9,10] However, the effectiveness of any anticoagulant, or the dose used, may be limited by the component and mechanical design of the EC. This suggests that in addition to an effective anticoagulant drug, or more importantly when no anticoagulation is used in patients at risk of bleeding,

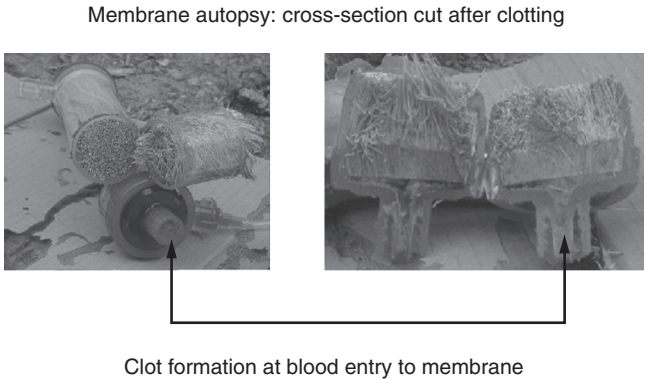

FIGURE 169.1 A membrane autopsy revealing a clot at the entrance to the membrane where resistance to flow occurs. A common site for clot formation independent of the membrane fibers.

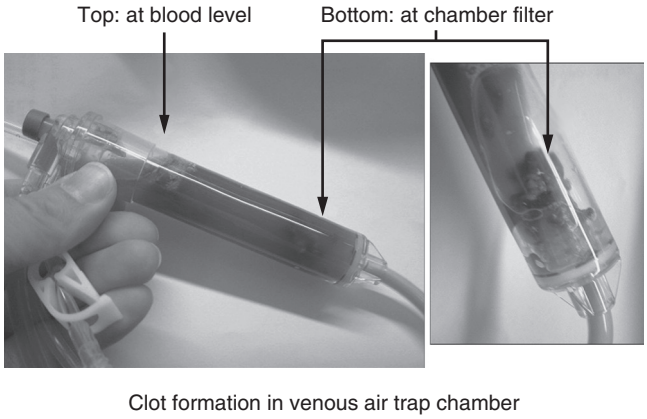

FIGURE 169.2 The venous air trap chamber showing clot formation commonly occurring at the top of the chamber and at the bottom around the outlet filter.

other strategies to prevent clot formation in the EC are useful to implement at all times, in every patient treatment.

When clotting does occur in the EC, differentiating between component failure (mechanical obstruction) and anticoagulant failure (dose too low, or poor response) is very important so that the correct remedy is used.[9–12] For example, prescribing an increase in heparin dose, or changing to a different method may not be appropriate when the cause is mechanical obstruction in the EC.

ACCESS CATHETER AND SITE

The double-lumen venovenous access catheter typically used for CRRT is not a common place for clot formation. However, the access catheter is implicated in the formation of clot. The essential task of the catheter is to allow blood to be drawn from the patient vein into the EC and returned to the vein from the EC. The catheter must be patent to allow both functions and, in addition, must not offer too much resistance to this flow. As blood pump speed increases, a greater demand is placed on the catheter (increasing negative arterial pressure) such that if the lumen size is inadequate, or the lumens are obstructed, the pump sucking blood from the catheter will fail and not deliver its prescribed output. This then can cause slowing of blood flow to the membrane and clotting, because ultrafiltration continues irrespective of blood-flow indicator speed.[13] Although not often an immediately identifiable event, this covert access catheter failure leading to unrecognized reduced blood flow may be a causative factor in membrane clotting as a terminal event. Furthermore, if obstruction to the venous or return limb of the double-lumen catheter occurs (high venous pressure), this creates stasis and slowing of blood flow in the venous bubble trap chamber, also promoting clotting in this chamber. These two pressures are fundamental for understanding the function of the access catheter and may be identified with their association and contribution toward circuit clotting when viewed during or after a treatment. These pressures commonly are displayed numerically as live data during a treatment but also may be viewed on graph throughout or after a treatment and can be downloaded information from the Prismaflex machine.[14] Fig. 169.3 provides a typical screen view as a schematic, and in this case, considerable negative pressure is apparent ("arterial") and with blood flow failure, pump stoppage failure to restart as the terminal event. A failure to restart treatment and/or clotting occurring because of these stoppages contributes to termination of treatment; this often is recorded as "circuit loss, clotted or treatment stopped" or "access problem" in the nursing notes. An awareness of this situation and being able to view a machine display and identify this is a useful clinical initiative.

The site (e.g., femoral, subclavian, internal jugular) of the venous access catheter may be related to more or less potential for obstruction. Evidence[15,16] suggests that femoral placement with the catheter tip close to the right atrial appendage identified from chest x-ray is best for circuit function. Nurses' anecdotal experience can generate many opinions on this topic as they observe a strong relationship between patient positioning and access function. For example, when a patient is supine and not sitting up, the femoral vein site may function well; however, if this patient is angled greater than 45 degrees, the catheter may fail. This also may apply to the subclavian or internal jugular site, with no obstruction when the patient is lying flat because of higher venous pressure, but sitting upright creates flow dysfunction. Side lying also can create obstruction, particularly at the subclavian site, with shoulder flexion and kinking of the catheter. Nursing care and physical therapy must be managed with caution and with attention to changes in arterial and venous circuit pressures indicating catheter obstruction. If physical therapy and other patient movements are scheduled during a treatment, it may be better to pause the machine fluid exchange process, slow the blood pump to 50% of set treatment speed (this may be inherently automatic for this intervention), then move the patient. After the move is complete, the fluid exchange reactivated, and with this the blood pump slowly returned to treatment speed. The arterial and venous pressures should be monitored, and if excessive negative access pressure occurs, subtle maneuvers can be made to the patient position to preserve stability before restarting the fluid exchange (the treatment). This strategy is highly recommended when moving a patient connected to CRRT out of bed to a suitable recliner chair in the intensive care unit (ICU).

BLOOD PUMP–FLOW SPEED

Blood flow controlled by the blood pump setting may have some influence on the development of EC clot formation.[12] In theory, a faster blood flow rate means less clot formation, and this may be associated with less filter clotting at faster rates.[17] A blood flow rate of 200 mL/min is applied commonly and is adequate to maintain flow across the length of the circuit, with minimal stasis, but not too fast to increase resistance and create turbulence and cell damage

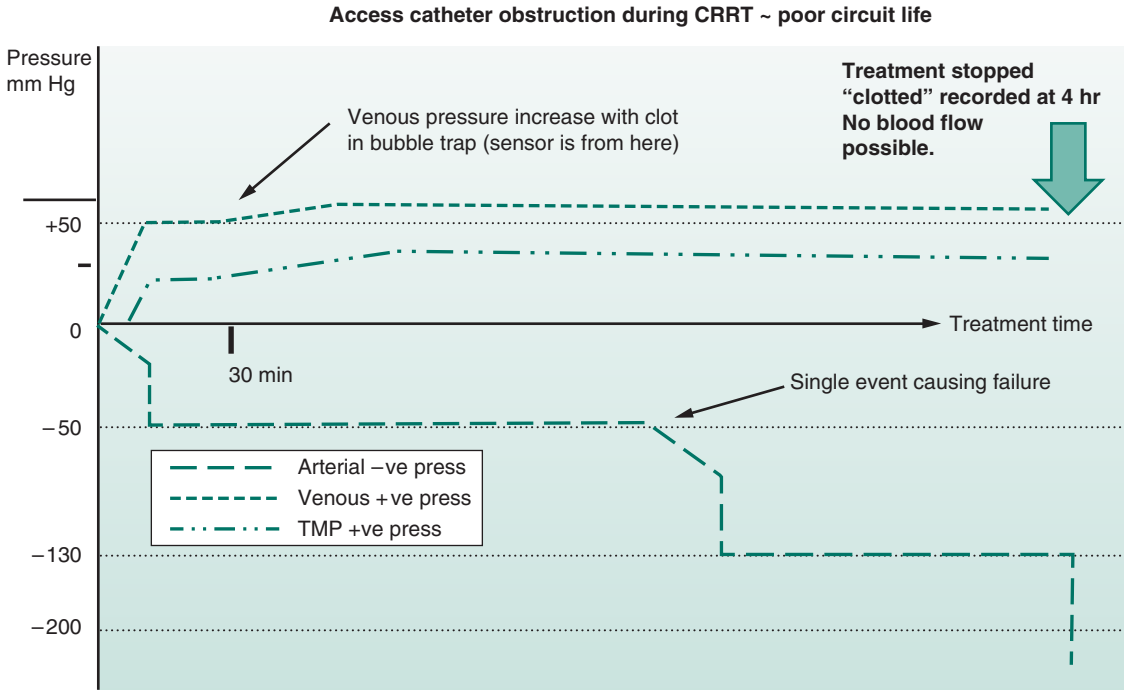

FIGURE 169.3 Schematic for typical machine display reflecting circuit pressures graphed since treatment start. Arterial is a negative and venous a positive reading. Increasing negative "arterial" pressure indicates access catheter failure and, if prolonged, is associated with blood flow stoppage(s) and clotting. Treatment ceased because of this. *CRRT,* Continuous renal replacement therapy; *TMP,* trans membrane pressure.

with shearing forces at resistance points, such as where blood is entering the membrane or exiting the venous chamber. When convective clearance is the sole mechanism for solute removal and replacement fluids are administered after the hemofilter (postdilution), blood flow must be adequate for the ultrafiltration rate setting to minimize concentration of blood in the hemofilter. For example, with an ultrafiltration rate of 2 L/hr or 33 mL/min, a blood flow rate of 200 mL/min is suitable because the plasma water removal is less than 15% of the blood flow.

With the use of diffusive clearance techniques, hemoconcentration such as this is less important. Despite the slow dialysate flow used in continuous therapy, blood flow up to 250 mL/min often is used to maintain flow and prevent blood stasis. However, this is without any evidence for circuit life or solute removal, and variability between 150 and 250 mL/min is apparent from surveying clinicians.[18] A relationship may exist between clearance mode or technique and clotting in the EC. Although there is currently no prospective study published, some clinicians suggest that convection clearance (continuous venovenous hemofiltration [CVVH]) may have a higher potential for clotting in comparison with diffusive clearance (continuous venovenous hemodiafiltration [CVVHDF]), and data from one study indicate a significant difference in circuit life favoring CVVHDF.[19] This was a randomized crossover design study treating 45 patients with CVVH (50 circuits—mean life 8 hr 39 min) and CVVHDF (43 circuits—mean life 18 hr 6 min), $p < .001$.

MEMBRANES: TYPE, SIZE, HEPARIN COATING

Membranes used for CRRT vary in fiber composition and surface area. It is also common to add heparin into a circuit during or after priming to heparin-coat the membrane.[2,7,8,20] No high-quality evidence suggests this has any effect on all membranes; however, plastic and membrane surfaces do uptake heparin, particularly after treating to neutralize negative charge,[21,22] and thromboresistance with reduced clotting has been reported in bovine experiments[23] with a surface-coated membrane. Therefore, unless contraindicated, heparin can be added to a circuit after priming and before connection to the patient and circulated within the EC at a slow blood pump speed to promote distribution and the coating effect. Depending on dose used and the specific patient requirements, this heparin may be flushed out before it is connected to the patient, or a bolus dose before the use of heparin during treatment can be considered—for example, 5000 international units of heparin into a circuit priming bag of 100 mL saline. Then the blood pump machine is set at 50 mL/min for 10 to 20 minutes with both limbs of the circuit into the priming bag for continuous recirculation.

Different fiber types also may influence clotting.[24–26] Although synthetic membranes are considered biocompatible, one patient may have premature clotting when exposed to an acrylonitrile membrane as opposed to a polysulfone membrane, or vice versa. There also may be some differences in racial and genetic disposition to clotting for different membrane compositions.[27] If one type of membrane clots frequently despite anticoagulation (always reassess access catheter function in this situation) and when no anticoagulation is used, trying a different membrane composition can be useful. Finally, larger surface area membranes may take longer to clot, possibly because of lower resistance to blood flow[28] or simply the surface area. This can be useful in the context of adults with frequent occurrence of membrane clotting. Increasing the membrane size from 1.0 m^2 to 1.4 m^2 or a 40% increase may create an increase in time before clotting and failure, but the dead space is larger and the purchase cost may be higher.

FLUID ADMINISTRATION—PREDILUTION

There is evidence that use of predilution reduces membrane clotting in comparison with postdilution in convection clearance CVVH.[29] The hemodilution of blood before passage through the membrane appears to reduce clot formation by hematocrit change. The amount of predilution volume required to achieve this effect is not clear. Citrate anticoagulation can be performed by adding citrate to the replacement fluids. This must be given as predilution and has the combined effect of anticoagulation and predilution.[30] Pre- and postdilution also may be used and with citrate added fluids can prevent clotting in the venous bubble trap. Predilution fluids must enter the CRRT circuit after the blood pump so that they increase the blood flow. If the fluids are administered before the blood pump, they become a percentage of the blood flow and reduce blood delivery to the membrane.

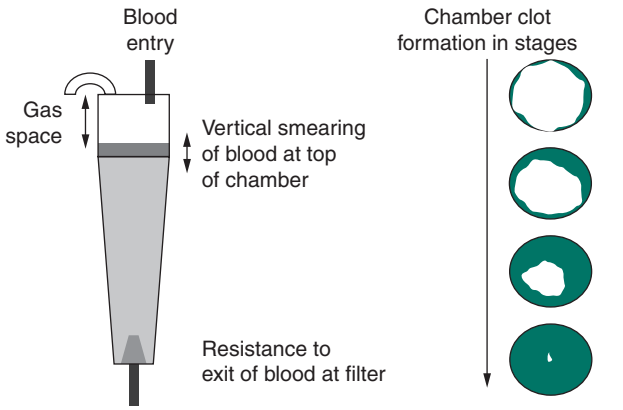

FIGURE 169.4 A schematic of the venous chamber showing the mechanism by which a clot forms. Smearing of blood at the top of the chamber with a fluctuating blood level is consistent with the blood pump rotations and access catheter resistance. This causes cell adhesion and clot formation with eventual obstruction.

VENOUS CHAMBER

An important safety feature of any EC is an air trap chamber placed in the EC before the blood returns to the patient. An ultrasound wave is passed through the chamber to detect air. A mesh filter also is provided in this chamber to prevent a clot from the EC from entering the patient's circulation. There should be no further line connections after the venous bubble trap bypassing these safety features. However, this EC component is often a site for clotting.[1,2,7,12,31,32]

A chamber must be designed to allow for the air detection mount and the exit mesh filter and also may include a fluid administration port. As blood enters the chamber, its flow characteristics change as a pool is evident, with the chamber level rising and falling consistent with the pulsatile flow of the blood pump, and the varying resistance at the return limb (venous) of the access catheter. It is necessary to permit a pocket of gas (air or CO_2) above the blood level to act as a conduit for pressure readings when connected to a gas-line pressure sensor and transducer. This is an easy way to determine pressure at this point of the EC. It is effective for a pressure display in millimeters of mercury (mm Hg) but differs from in-line blood-filled transducers commonly used for pre-blood pump or pre-filter pressure. The blood level in this chamber fluctuates up and down with an amplitude and frequency directly proportional to the main blood pump speed and venous return pressure. This causes a constant blood smearing and cell deposition on the inside of the chamber and eventually creates a ring around the chamber that gradually builds to form a clot (Fig. 169.4). The process appears to be hastened by the coagulability of the blood, amount of gas allowed in the chamber, and the chamber profile shape. To prevent this clot formation, readjustment of the blood level in this chamber to below the clot formation may reduce clot formation when frequent chamber clotting occurs. Therefore starting treatment with a full chamber is desirable.

Attempts have been made to prevent this clotting by adding heparin into the chamber before and during use,[31] adding fluids into the chamber (postdilution), using a design with blood entry as horizontal flow[32] or with incoming blood entering under the high blood level in the chamber. This last approach can create a cell-plasma separation with a small layer of plasma separating to the top of the chamber providing a blanket protecting the cells from exposure to the gas and reducing cell smearing with adhesion. None of these clot prevention methods is supported by data to suggest they are associated with less chamber clotting.

However, despite a lack of supporting evidence, it is common practice and may be useful to (1) keep the venous chamber full of blood, thereby minimizing the gas pocket, (2) adjust the level down when a ring of clot begins to form, resetting the advancement of this clotting, (3) add postdilution fluids directly into this chamber when used, and (4) add heparin into the chamber during the priming procedure of heparin coating.

Manufacturers are attempting to modify and/or eliminate the venous chamber from the CRRT EC, but because the safety feature must be retained, this is difficult. Gambro did this with the Prisma machine (Gambro, Lyon, France), but has reintroduced a de-aeration chamber with the latest Prismaflex machine that allows for postdilutional fluids administration using horizontal flow entry.

TRAINING, EDUCATION, AND THE MULTIDISCIPLINARY TEAM

Safe and skilled use of CRRT machines requires nursing education and training activities. In addition, without a suitable education process providing theoretical and practical information, patient safety and successful, effective therapy will be compromised.[33] It is more likely that premature clotting of the EC will occur if nurses managing a treatment do not understand the strategies discussed in this chapter and have poor troubleshooting skills for maintaining blood flow. For example, in the event of blood pump stoppage for an alarm event such as arterial or negative access pressure, if the pump is stopped for a prolonged period, blood stasis and clot development are likely. In situations in which nursing skills are poor in an ICU and nephrology nurses are required to attend the ICU for troubleshooting, the time delay involved can cause a simple alarm event to be irreversible because of clotting without blood pump operation. This may mandate a response call or protocol similar to the medical emergency team and require set criteria for when a nurse with low or minimal CRRT experience should call for help.[34] Current machines have troubleshooting prompts on screen displays and unlatched alarms that reset the alarm and restart the treatment if the error corrects itself

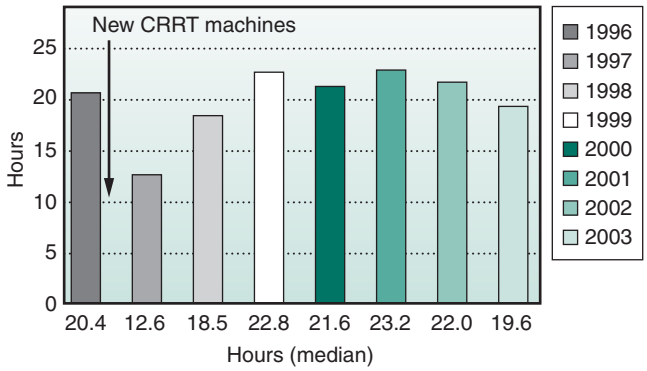

FIGURE 169.5 Filter life data indicated as the median. A reduction in median filter life can reflect loss of skills and poor troubleshooting ability among medical personnel, for example, after new continuous renal replacement therapy (CRRT) machines are introduced to an intensive care unit.

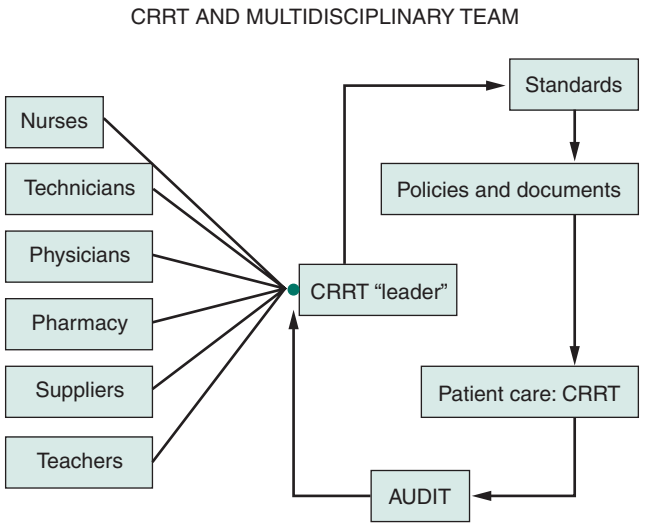

FIGURE 169.6 The team and audit concept for successful use of continuous renal replacement therapy (CRRT) as an important strategy to prevent excessive clotting.

or the ability to correct fluid balance errors automatically (AQUARIUS, Nikkiso Europe, Langenhagen, Germany). However, other alarms, such as air in the line, are latched and will not reset, and the blood pump will stop until a reset function is selected and the air is removed. Although this event may be rectified easily by a skilled nurse, untrained nurses may take a prolonged time or fail to fix the alarm, thus prolonging a period of no blood flow.

There are many strategies to train nurses for these events, using simulation setup of the machine and EC, interactive video activities of these alarms, and simple tutorial activities on a drawing whiteboard.[7,35] Bedside records indicating circuit life are useful and, when audited, can reflect variations in nursing expertise.[36] A reduction in median circuit life may be associated with a reduction in nursing expertise. Fig. 169.5 indicates circuit life audits over many years in a large ICU setting and reflects the reduction in circuit life after the introduction of new CRRT machines, with a recovery of expertise following.

A multidisciplinary team is also important to success. Without a leader or similar person(s) to champion the cause, poor outcomes of CRRT use, including frequent circuit clotting, may occur.[36,37] This team must include senior medical, nursing, pharmacy, technical, or allied health personnel, a nursing teacher, and industry representatives or suppliers. A final and further strategy to prevent clotting in the EC is to develop and nurture such a team with suitable leaders and specialist clinicians (Fig. 169.6). The team must meet regularly and undertake an audit of the circuit or filter life on a regular basis.[38] This information must be recorded at the bedside or at least in a similar way for every treatment. A calculation for median hours of function can be useful feedback to the group for the review of policies, protocols, nurse training, and patient care needs.

CONCLUSION

Clotting in the circuit during CRRT can be delayed or prevented by the administration of anticoagulants and by prevention of blood stasis and resistance in the circuit. Clot formation is a complex hematologic process in the critically ill patient, but clotting in the circuit membrane and venous chamber can be prevented by attention to blood flow mechanics, particularly when no anticoagulation is necessary. Nursing training and close monitoring of circuit life provide important feedback to a medical team focused on CRRT and preventive clotting strategies.

Key Points

1. Blood clotting in the circuit is more likely at places of high resistance or shear stress (particularly negative pressure) and where the blood cells and plasma separate.
2. Anticoagulants are useful, but clotting also may be due to mechanical obstruction.
3. Accessing catheter function, correct blood flow at approximately 200 mL per minute, pre- and postdilution fluid administration when possible, heparin coating of the circuit, and nursing training with an audit process monitoring circuit life are useful adjuncts to anticoagulation agents in preventing and managing circuit clotting.

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