



# Free Radial Forearm Flap Failure due to Un-Autonomization in a 105-Year Old Patient

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## Abstract

Age itself should not be considered a contraindication to serve as the technique of choice to treat complex reconstructive challengers in older patients. Octogenarian is defined being older than 80 years or more. General surgery practice shows a mortality rate of 5 percent for octogenarians. This is more for those older than 90 years. Older patients are less capable of handling longer water electrolyte imbalances. Renal blood flow drops 50 percent from young adulthood to the age of 80 years.

## Introduction

In search for related medical literature, there are several case series of elderly patients who had free flap surgery [1-3]. In those series, age range varies between 70 and 95 years. Upper age that withstands with the difficulties of microsurgical tissue transfer is important for showing the capabilities of human body. This report briefly aims to show an experience of free flap transfer in a 105 year-old female patient.

## Case Presentation

The patient was diagnosed as squamous cell ca of the lower lip 5 years earlier and had 5 operations which included wedge lip excision and repairs with local tissues. The end result was wound dehiscence with exposing bare mandibular symphysis with granulation tissue. Xerosis of the tongue, poor nutritional status and lack of competence oral functions were noted. As local flap options had already been used, a free radial forearm flap repair was planned taking the risk of ASA IV (older age, diminished organ functions, poor nutrition etc).

Under general anesthesia, a radial forearm flap harvested from the right upper extremity was transferred to the mandibular defect by anastomosing radial artery to right facial artery with concomitant vein to facial vein. She was administered heparin IV treatment for three weeks postoperatively. The flap was good in place until the 10<sup>th</sup> post-op day. A sudden enlarging cyanosis covered the whole flap with bleeding every side of it (Figure 1). A hand Doppler examination showed the radial artery was patent beneath the flap. A further examination with pulse -oxymeter was made at post-op 18<sup>th</sup> day. It proved that there was 93% of oxygen saturation on the screen (Figure 2). At that time, a skin biopsy was taken from the flap for histopathologic examination. Hematoxylin eosin staining with x40 and x100 magnification showed an ischemic necrosis without any inflammation of dermis and sub-dermal fat layer. Clinically the prominent abnormality with the biochemical data were low albumin, low fibrinogen and low total protein (Figure 3). Human albumin infusion was given at the second postoperative week in order to correct low albumin level. Peripheral parenteral solutions were also administered during the whole postoperative period. D-dimer level was also elevated, as a result of anticoagulant therapy presence (D-dimer; 418 mg/dl). This was a reflection of fragmentation of fibrin network consisting D-dimers by plasmin in the blood. A fibrin layer forms within the first 2 days and this would be one of the most important steps for gaining a good revascularisation process.

## Discussion

A general consensus that flap survival is possible, when thrombosis or pedicle ligation occurred after postoperative day 12 is present. This minimal critical period of time may even be as low as 6 days for arterial and 9 days for pedicle and 4 days for venous compromise [5]. According to a recent review, most of thromboses occur within the first two postoperative days. Some of them are related

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Figure 1: (A) Preoperative view of the lower lip defect and 1 (B) Cyanotic flap bleeding from sides.



Figure 2: Pulse oximeter probe on the flap surface and the screen showing 93% oxygen saturation.

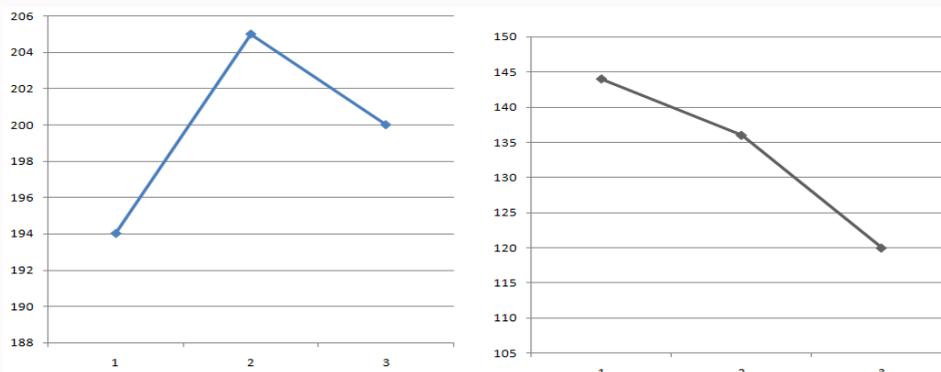


Figure 3: The graphics show the course of the levels of total protein, albumin, fibrinogen and platelet counts during the follow-up period. A) albumin and total protein as g/dl B) fibrinogen as mg/dl and platelet counts (1<sup>st</sup>, 2<sup>nd</sup>, and 3<sup>rd</sup> blood samplings were done at 1<sup>st</sup>, 10<sup>th</sup> and 18<sup>th</sup> postop days).

to heparin induced thrombocytopenia and thrombosis (HITT) and it is associated with drop in platelet count seen 4-10 days following heparin treatment [6]. We exclude this option, as the platelet count had not dropped below any critical level during the follow-up period. Establishment of functional revascularization is essential for the successful healing of cutaneous wounds. Angiogenesis, the formation of new blood vessels from an existing vascular bed, is a normal physiological process. It is an integral factor in determining the success or failure in Plastic and Reconstructive Surgery [7]. Critical time period required for independent neovascularisation to allow flap survival is not known. Most authors conclude that salvage surgical intervention for flaps presenting with delayed vascular compromise after fourth postoperative day are unsuccessful.

Free flap autonomisation can be defined as a certain time after which neovascularisation or angiogenesis from recipient bed of free flap transfer has become sufficient enough to allow its survival without revision [8]. In ischemic flaps after resuming reperfusion, it was

shown that inflammatory cells infiltrate the reperfusion tissue which can be detected within flap by biopsies taken at 7<sup>th</sup> day experimentally [9]. It can be said that a free tissue transfer to a non-vascularised environment carry a risk of flap failure due to major pedicle loss, even in the long term. Histologic neovascularisation evidence has been demonstrated in animal studies [10]. In surviving flaps, the reduced blood flow gradually increases in a favorable recipient site. Early neovascularisation starts at 4 days in the pig and rabbit models [11]. Even the presence of ischemic cells within flap, there must be inflammatory cells scattered around the necrotic areas on histopathologic examination. We have found very low inflammatory cell in the skin biopsy. We speculate that there was a reduced skin perfusion during the early post-surgery days and this discrete skin perfusion should be clinically present in the dermal layer of the flap, while there was active bleeding beneath the flap. A hypothesis would be that atrophic skin of a very elderly person did not permitted a clear skin perfusion via skin perforators of radial forearm flap. In other words, the perfusion was limited only deep fascial layer of the

fasciocutaneous flap. A second hit by low fibrinogen level may have not propagated a physiologic angiogenesis from the poor recipient bed. All these events sequence ended up with total flap failure.

## Conclusion

Free flap failure in the very old patient was resulted from the following factors;

- Poor recent bed and vascularisation
- Low fibrinogen level
- Reduced skin perfusion
- Reduced inflammatory response

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