Introduction

In contrast to clinical pathology, forensic pathology investigates small scale studies that are often unique to a specific case and are bound by legal restrictions. A forensic pathologist then has to deal with several areas of expertise, also at autopsies. There however is a need for additional tools in forensic autopsies. In this thesis we aimed to improve diagnostics related to I) skin wound age determination, II) taphonomy and III) cardiovascular pathology.

I: Skin wound age determination

For correct diagnosis of an injury, amnestic information of the estimated time of injury is combined with macroscopic and microscopic analysis of the wound. The collection and processing of samples usually involves photographing the wounds and taking either the entire wound or representative samples for microscopic analysis and immunohistochemical studies (1, 2). Immunohistochemical studies have been reported to be useful in detecting and differentiating between the different inflammatory cells and/or inflammatory markers in wound analysis (3, 4, 5). After the induction of wound injury, an immune response is induced in the process of wound healing, beginning immediately and lasting up to several weeks. This has been reviewed in Chapter 2. The aim of this chapter was to introduce the reader to the fact that although much work has been done on injury dating the gold standard as such is yet to be found.

In forensic pathology a crucial question always remains in the dating of wound injuries: Did a wound occur before death or was it a post mortem injury. Therefore determining the vitality of skin wounds is of vital importance. What determines a vital wound? Several histological characteristics such as hemorrhage have been suggested; although these have also been shown to occur in non-vital wounds (6, 7, 8, 10). We wondered whether analysis of markers related to the process of coagulation, which theoretically is induced in hemorrhage of vital wounds, could discriminate between vital and non-vital wounds. This we have studied in Chapter 3, including the development of a probability scoring system for wound injury dating.

It is known that the immune response differs between different types of wound injury. This is especially true for burn injuries, as they induce a severe immunological response and if not treated can lead to systematic infection causing death. The acute phase proteins complement and C-reactive protein are known to be increased in plasma up to months after a burn injury (10, 11, 12, 13). Although it is known that these acute phase proteins play a role in wound healing, their role locally in burn wounds has not yet been studied. In Chapter 4 we have investigated these acute phase proteins locally at a tissue level of a burn injury site.

II: Taphonomical and tissue degradation studies

Taphonomy is a relatively new and unknown science. It was first introduced in palaeontology and has since been embedded in the world of geo-archaeology and anthropology. Well known taphonomic features such as bone or cementum degradation, the development of livores, rigor mortis and calor mortis are all part of the decomposition process (9) of decaying organisms and are central to the study of taphonomy. Taphonomy can answer questions that are of interest to forensic pathologist such as ones
dealing with ongoing body decomposition. Large studies investigating human decomposition were performed in Knoxville at the anthropological research facility in the United States. However whether the obtained results can be extrapolated to conditions observed in The Netherlands is questionable. Therefore a study using a pig model, to address different typhonomic questions relating to body decomposition was undertaken and is presented in Chapter 5 of this thesis.

III: Cardiovascular pathology

Acute myocardial infarction (AMI) is a leading cause of death in the western world (14, 15, 16) and needs to be ruled out in the first instance at an autopsy as the cause of death, also in forensic autopsies. AMI can be identified, as studies have shown, using routine histochemistry using the nitro blue tetrazolium staining method of heart slides. In this way AMI was able to be detected macroscopically at 3 hours or more after its onset (17, 18).

To detect cardiac infarctions earlier, studies looking at ultrastructural changes of the mitochondria in the heart have been described in both canine and rat hearts. Mitochondrial deposits which are small osmiophilic amorphous densities (19, 20) that can be detected using electron microscopy as early as one hour post AMI (21) have been described. Other mitochondrial changes have also been reported to occur with AMI such as swelling of the mitochondria and the formation of disorganized cristae. However due to the fact that these changes are also observed during the autolysis process of cells (21), the ability to use electron microscopy to detect changes associated with early AMI in both canines and rat hearts has been questioned. Since autolytic changes also occur in human hearts, it is unclear whether mitochondrial deposits detected as early as half hour post mortem could be associated with AMI. In Chapter 6 we investigated whether or not ultrastructural analysis of mitochondrial deposits can be used as a method for the detection of early AMI.

It is common knowledge that alterations to coronary artery structure can induce AMI by changing regional blood flow and provoking ischaemia (22). In an earlier study, the results obtained suggested that the induction of AMI can be aided by the presence of pre-existing aberrations in the intramyocardial microvascular, related to the induction of local inflammation in these arteries. Further ultrastructure studies were carried out to investigate whether or not intramyocardial capillaries also play a role in AMI induction. These studies are described in Chapter 7 where the basement membrane thickness of intramyocardial capillaries was measured.

Another cause of ischemia and infarction that is widely seen in patients with other diseases is pulmonary embolism. As in the case of AMI, pulmonary embolism is a significant cause of morbidity and mortality (23, 24). An experimental model has shown that pulmonary embolism causes a very selective decrease in blood flow only to the right ventricular subendocardium (25) that might result in the reported increase in the level of CD68 positive macrophages in the right ventricles of patients with pulmonary embolism (26, 27). Although the study concluded that the influx of macrophages was a result of an ischemic episode to the heart that occurred subsequently to the pulmonary embolism, these ischemic changes were never proven. We wondered whether this could be explained by the induction of myocarditis in these patients. In Chapter 8 of this thesis, the relationship between pulmonary embolism and (endo) myocarditis was thus investigated.
References


